XX CASE RECORDS AND COMMENTARIES

IN

OBSTETRICS AND GYNAECOLOGY

SUBMITTED BY

DR. JOSEPH W ANGIRAI MUSANA

AS

P \RT FULLFILMENT FOR THE

DEGREE OF M ASTER OF MEDICINE IN

OBSTETRICS AND GYNAECOLOGY

OF THE UNIVERSITY OF NAIROBI

JULY 2005

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GYNAECOLOGY LONG COMMENTARY

Dedication

This book is dedicated to my precious daughter Barbara and my lovely wife Margaret for their love, patience, tireless support and encouragement during my studies. May God bless them.

ACKNOWLEDEMENTS

It is by the grace and love of God that I have been able to complete this book.

I wish to express my sincere gratitude to the Ministry of Health for sponsoring my postgraduate studies.

I am greatly indebted and sincerely thank all the consultants, lecturers and senior registrars of the department of obstetrics and gynaecology for having dedicated their time to ensure that I acquired all the necessary knowledge and skills during my training at the University of Nairobi.

I would like to sincerely thank my supervisors Prof. S.B.O Ojwang and Dr. Weston Khisa for their guidance in writing my long commentaries and short cases. I am grateful to Prof. Kigondu and Dr. Kiarie for assisting me with my gynaecology and obstetrics dissertations. I would also want to thank Dr. Wanyoike Gichuhi for proof-reading my gynaecology long commentary and offering invaluable advice during my studies. I would also like to sincerely thank Dr. Rafique Parker of the Aga Khan Hospital Nairobi, for assisting me in my gynaecology long commentary.

I am most grateful to the nurses who assisted me in data collection for the obstetric long commentary especially Mr. Ambasa and Sr. Kibisu. I thank Francis and Mr. Muniu of KEMRI for assisting me in data entry and analysis.

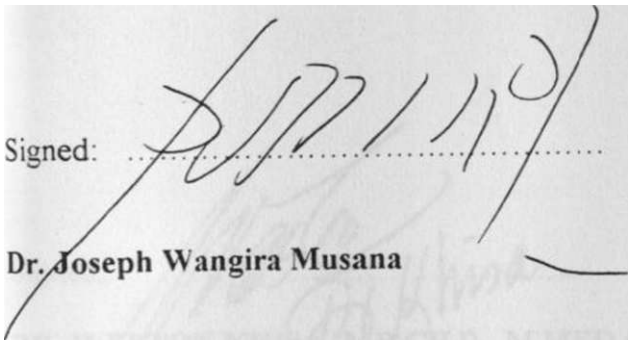
1 am greatly indebted to my parents Mr. Gabriel Musana and Bertina Wangira for their continuous encouragement during the difficult times. I cannot forget my sisters, Aggie, Maryann, Maggie and my brothers Peter, Shete and Edward for their prayers and support.

Last but not least, I would not have been able to complete my studies were it not for the love, inspiration and dedication that I received from my lovely wife Margaret and my little angel Barbara. May the Lord shower them with blessings.

DECLARATION

I declare that the short cases in this book were managed by me under the supervision and guidance of senior members of staff at the department of Obstetrics and Gynaecology of the University of Nairobi.

I further declare that the two long commentaries in this book are my original work and have not been presented for a degree in any other university.



JULY 2005.

CERTIFICATION OF SUPERVISION

This is to certify that Dr. Joseph Wangira Musana researched upon the long commentaries presented in this book under our guidance and supervision and that this book is submitted with our approval.

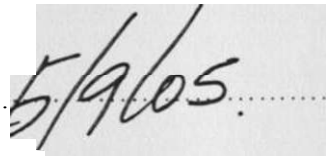
S i g n e d j S i i ^ [5] ^ . . . Date:. . . . .L . l S . l l ? ^

PROF. S.B.O. OJW ANG M.D., \I.MED (OBS/GYN)., Dip. GYN.ONCOL.

PROFESSOR AND CONSULTANT

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

UNIVERSITY OF NAIROBI.

Signed:.  y f l f j . . . Date:. 

DR. WESTON KHISA, MB!CH.B., M.MED (OBS/GYNT-

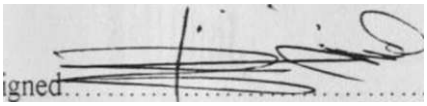
CONSULTANT AND LECTURER

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

UNIVERSITY OF NAIROBI.

CERTIFICATION

This is to certify that obstetric cases number 1, 5 and 7 and gynaecology cases number 10, 12, 13 and 14 were managed by Dr. Joseph. Musana under my supervision and guidance at the Kenyatta National Hospital.



*C-fa/c^*

PROF. S.B.O. OJTWANG., M.D., M.MED. (OBS/GYN)., DIP.GYN. ONCOL.
PROFESSOR .AND CONSULTANT
DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY
UNIVERSITY OF NAIROBI.

CERTIFICATION

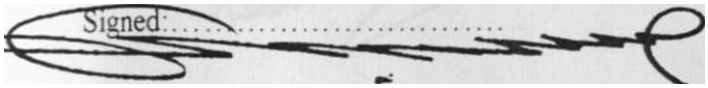
This is to certify that the obstetrics cases number 2, 3, 6 and 7 and gynaecology cases number 3, 4, 5 and 6 were managed by Dr. Joseph.W. Musana under my supervision at Kenvatta National Hospital

D t >

PROF. J. KARANJA., MB.CH.B., MMED (OBS/GYN).
PROFESSOR .AND CONSULTANT
DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY
UNIVERSITY OF NAIROBI.

CERTIFICATION

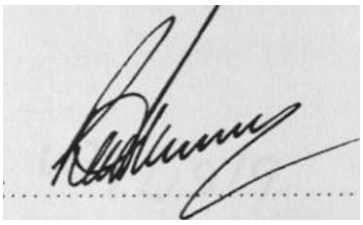
This is to certify that obstetrics cases number 4, 8, 11 and 12 and gynaecolgy cases number 7 and 8 were managed by Dr. Joseph W. Musana under my supervision and guidance at Kenyatta National Hospital.



DR. J.B.O. OYIEKE. MB.CH.B., M.MED (OBS/GYN).
CONSULTANT, SENIOR LECTURER AND CHAIRMAN,
DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,
UNIVERSITY OF NAIROBI.

CERTIFICATION

This is to certify that obstetrics cases number 9, 10 and 15 and gynecology case number 9 were managed by Dr. Joseph W. Musana under my supervision and guidance at Kenyatta National Hospital.

Signed  Date

DR. KOIGI KAMAU. MB.CH.B., M.MED(OBS/GYN)
CONSULTANT AND SENIOR LECTURER
DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY
UNIVERSITY OF NAIROBI

CERTIFICATION

This is to certify that the gynaecology cases number 1, 2, 11 and 15 and obstetrics cases number 13 and 14 were managed by Dr. Joseph W. Musana under my supervision and guidance at Kenyatta National Hospital.

DR. WANJALA. S.H.M MB.CH.B. MMED (OBS/GYN)
CONSULTANT AND SENIOR LECTURER
DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY
UNIVERSTY OF NAIROBI.

INTRODUCTION

Kenyatta National Hospital, the largest hospital in Kenya, is situated in Nairobi. It is about 3Kms from the city centre along Ngong Road. It is built on approximately 304 acres of land and was started in 1901 as the Native Civil Hospital before later becoming King George's Hospital. In 1964, it was renamed Kenyatta National Hospital (KNH). It now serves as a referral centre as well as serving the population within and around the city. It provides curative, preventive and rehabilitative services in all medical disciplines. It is a training centre for undergraduate and postgraduate students from the College of Health Sciences of the University of Nairobi. It is also a training centre for nurses, clinical officers and other paramedics from the Kenya Medical Training College.

The hospital is housed in a 10-storev building complex with extensions that serve as outpatient clinics, theatres, casualty, intensive care unit and laboratories.

The hospital is currently administered as a state corporation by a Parastatals Board established in 1986 by an Act of Parliament.

OBSTETRIC AND GYNAECOLOGY UNIT

The unit provides both out-patient and in-patient services. The out-patient sen ices are provided at casualty department, antenatal clinic, post-natal clinic, gynaecology clinics and the family welfare clinic (FWC). The in-patient services are provided in labour ward, acute gynaecology ward, cold gynaecology ward and antenatal/postnatal wards.

In terms of personnel the unit is divided into three Firms, each headed by a senior consultant obstetrician/gynaecologist, with a team of senior registrars, registrars, interns, nurses and paramedical staff. The senior medical staff are drawn from both the University of Nairobi and KNH.

Laboratory services are provided by the hospital laboratories. In addition to the hospital laboratory services, the Department of Obstetrics and Gynaecology of the University of Nairobi offers the follow ing laboratory services for the hospital: semen analysis, hormonal radio-immunoassav. cytology, chromosome analysis, bilirubin spectro-photometrv, surfactant test and glucose tolerance

test. Radiological examination such as ultrasound are provided in radiology department of KNH and also at the department of Radiology University of Nairobi.

Casualty department

This offers services 24 hours a day. Most patients are treated and discharged or referred to the gynaecology or obstetrics clinics. Patients requiring admission are admitted either to labour ward or acute gynaecology ward.

Antenatal care (ANC)

ANC patients are booked on Monday mornings by senior registrars and the three firms work in rotation, booking about 50 clients every week. The patients report to the clinic at 7.30 am and are interviewed by the nurse who record personal data medical and obstetric history. The patients' height, weight and blood pressure are measured and urinalysis is also carried out and these recorded in the antenatal card.

A senior registrar from the booking firm reviews the patients and selects the high risk patients for follow-up. The risk factors considered include the following:

- Prirrugravidas. especially the adolescent and those above 35 years.
- Previous operations or complicated deliveries e.g. vacuum extraction, caesarean section post partum haemorrhage, ruptured uterus and among others.
- Grandmultiparous woman.
- Bad obstetric history including habitual abortion, still births or neonatal deaths.
- Medical diseases complicating pregnancy such as cardiac disease, hypertension, renal diseases, diabetes mellitus. anaemia thyroid disease and so on.
- Rhesus negative clients.
- Previous gynaecological problems e.g. repaired genital fistulae. myomectomy, tubal surgery for infertility or ovulation induction.
- Other indications include; multiple gestation, breech presentation and pre-eclampsia.

All booked patients have their names entered into the register. The patient then proceeds to the examination room where a registrar does a thorough general and systemic examination noting the gestational age. uterine size, foetal lie and presentation, foetal activity and heart tone and the condition of the mother. Those patients requiring admission are admitted to the relevant wards. For

the rest of the patients, appointments for follow-up are given and antenatal profile done i.e. haemogram, VDRL (screen for syphilis), blood group and Rhesus factor determination and HIV serology by ELISA.

Subsequent antenatal follow-up

The follow- up is usually monthly up to 28 weeks; gestation and fortnightly, from 28 weeks to 36 weeks and weekly till deliver,'. However for patients with obstetric or medical complications, the frequency of follow-up is individualized. At each visit health education about pregnancy, breast care, puerpenum and baby care is given. The patients are then examined with particular attention to blood pressure, proteinuria, weight gain and edema. Abdominal examination is done to determine fundal height, the fetal lie, presentation, engagement and fetal heart rate. The findings are recorded on the antenatal card which is kept in the hospital. For first pregnancies or previous pregnancies older than 3 years, tetanus toxoid doses are given 4 weeks apart, otherwise only booster T.T. is given during the second trimester.

At 36 weeks of gestation, clinical pelvic assessment is done on all primigravidas and radiological pelvimetry on patients with borderline pelvis or one previous scar with cephalic presentation.. Patients for elective caesarean delivery are admitted at 38 weeks of gestation.

Hospital admissions

These fall into three categories namely: -

- Booked patients from our antenatal clinic.
- Referrals from other hospitals or health centres.
- Those without prior antenatal care.

The last two categories constitute the majority of admissions.

Booked patients report straight to labour ward when in labour or whenever they have a problem when clinics are closed e.g. during weekends and at night. Unbooked patients are first seen in casualty before being sent to labour ward admission area. In labour ward all patients are seen and those requiring immediate delivery are retained in labour ward until delivery and patient not due for delivery are sent to the antenatal clinic (ANC). Patients who need close monitoring are admitted to the acute room in labour ward and managed accordingly. Patients in labour ward are seen by the

House Officers and Senior House Officers. Difficult cases are managed in consultation with the specialist obstetrician and gynaecologist and/ or consultant obstetrician and gynaecologist.

COMMON OBSTETRIC PROCEDURES

The following procedures are performed frequently within the obstetric unit. The description of the procedures given in this book refers to the standard or preferred method(s) as performed and taught within the department.

Vaginal examination

This involves the speculum and digital examination. The description below refers to digital examination. Vaginal examination is an aseptic procedure and it is done on admission during the initial assessment of labour. The examiner washes his/her hands and wears sterile surgical gloves. Explanation is made to the patient on the nature of the procedure. After consent has been obtained, the patient is placed in dorsal position with knees drawn.

The vulva is inspected and any abnormalities noted. The vulva is then cleaned using five swabs soaked with antiseptic solutions as follows: a swab is picked by the right and transferred to the left hand, using the left hand, the left labia majora is swabbed once anteroposterior then the swab is discarded. .Another swab is picked again and the same procedure is repeated on the right side. The procedure is repeated on the right and left side. The left hand now separates the labia using the index and thumb fingers and the introitus is gently swabbed anteroposterior^.

The right index and middle fingers are gently introduced into the vagina The fingers are positioned in the anatomical direction the vagina and the status of the mucous membranes are noted. Next the position, consistency, effacement and dilatation of the cervix are noted. The status of the membranes, presenting part, presence or absence of caput and moulding are noted. The colour, smell and quantity of liquor and presence or absence of cord are also noted.

Speculum examination

In obstetric speculum examination, the bivalve Cusco's speculum is frequently used. Indications include; antepartum haemorrhage, premature rupture of membranes, vaginal discharge and removal of a McDonald stitch.

The procedure and reasons for it are explained to the patient and verbal consent obtained. The patient is placed in dorsal or lithotomy position on the examination couch. The surgeon scrubs and

wears sterile gloves. The vulva is swabbed as described above. The labia are than separated with the index and thumb of one hand to expose the vaginal introitus. The Cusco's speculum is then gently introduced into the vagina with the width of its blades in transverse. The blades are then opened and the lateral walls are exposed and observed for any abnormality. The cervix is observed for the dilatation, bleeding, and rupture of membranes and does not shoe any liquor, the patient is asked to cough of fiindal pressure is applied. The speculum is withdrawn in the same way it was introduced. During the procedure the patient is kept of each step as this makes the examination easy.

MANAGEMENT OF LABOUR

The main objective of labour management in our unit is to achieve delivery within 12 hours of admission for every patient admitted in active phase of labour.

First stage of labour

Patients in active or latent phase of labour are admitted in the first stage. Progress of labour is recorded graphically on a partogram where uterine contractions, fetal heart rate, maternal pulse rate and blood pressure are recorded every half hour. Vaginal examination to assess the cervical dilation in centimetres, presence and degree of moulding and colour of draining liquor is done and recorded even- 4 hours. Artificial rupture of membranes is performed for all patients in active phase of labour at cervical dilatation of 6cm or more. However this is not done in patients with unknown HIV serostatus and those who are HIV seropositive unless there is an obstetric indication or have attained cervical dilatation of 7cm or more. It has been shown that artificial rupture of the membranes in HIV positive mothers is associated with increased risks of mother to child transmission. To hasten cervical dilatation intramuscular injection of hyosine bromide 40mg is given. In patients at cervical dilatation of 4 to 6 cm an intramuscular injection of pethidine is given for analgesia.

The partogram has two parallel lines: the "alert line" and "the action line". The action line is 4 hours to the right of the alert line. At admission, for patients in active phase of labour, cervical dilatation is marked on the alert line and the time noted. Cervical dilatation of at least 1cm per hour is expected. Any deviation of cervical dilatation curve towards the action line is an indication of some abnormality in the progress of labour and corrective measures are instituted accordingly. Corrective measures may involve augmentation of labour if contractions are poor or caesarean

section delivery if there is cephalopelvic disproportion (CPD). Augmentation of labour with oxytocin is done in those patients without a previous uterine scar, maternal or fetal distress and those who are not grand multipara Induction of labour routinely starts in the morning and invariably done by artificial rupture of membranes followed by the oxytocin drip.

Management of second stage

When delivery is imminent a vaginal and abdominal examination are done. The patient will also have the urge to bear down. She is then transferred to the delivery room and placed on the delivery couch.

Normal deliveries are usually conducted by a midwife, student midwife or a medical student under instruction. High risk cases like multiple pregnancy, premature deliveries and breech presentations are delivered by the registrar on duty. Strict asepsis is observed during the deliveries; sterile gowns and towels are used. The vulva and perineum are cleaned with antiseptic solutions (commonly salvon) and then the patient is encouraged to bear down with each contraction and to take.deep breaths between contractions. Fetal heart rate is monitored every 5 minutes.

As the head distends the perineum, the left hand of the midwife maintains flexion of the fetal head and if episiotomy is indicated. 5-10mls of lignocaine are infiltrated on one side of the vulva and a mediolateral episiotomy is performed using a blunt tipped Mavo's scissors. The perineum is supported by the right hand with sterile pad.

Once the delivery of the head has occurred, the mouth and nose are wiped with gauze to prevent aspiration of blood or amniotic fluid. A finger is swept around the fetal neck for the cord. If the cord is too tight around the neck is divided between clamps and if it is loose it is stripped over the head The anterior shoulder is delivered then followed by the posterior shoulder and the trunk. If the umbilical cord was not clamped, it is done so and the baby shown to the mother for sex identification before handing over to another midwife who carries out oropharyngeal suction as need be In high risk case, a paediatrician is usually in attendance. At delivery of anterior shoulder 0 5mg of ergometnne is given intramuscularly to the mother except where contraindicated. like in Hypertensive diseases.

Repair of episiotomy

This is carried out in three layers using chromic catgut suture No 2/0. The apex of the incision is identified and from here repair of the vaginal mucosa is carried out in continuous suture while the muscle layer is approximated with interrupted sutures. The skin is apposed using interrupted or continuous chromic catgut No 2/0 burying the knots and starting from the lateral edge. After repair the patient is advised on perineal hygiene and saline sitz baths.

The fourth stage

After delivery and repair of episiotomy blood pressure, pulse rate, uterine contraction and lochial loss are observed and recorded. The patient is encouraged to empty the bladder. The patient is then observed half hourly for two hours and then transferred to the postnatal ward for subsequent observations. Patients with normal delivery are discharged home after 24 hours.

OPERATIVE DELIVERY

Vacuum extraction

Vacuum extractor is used to accomplish delivery in prolonged second stage due to poor maternal effort or where bearing down is contraindicated as in cardiac disease or where expedite delivery is desired as in fetal distress occurring in the second stage of labour.

The procedure and its indication are explained to the patient and a verbal consent is obtained. The patient is placed in lithotomy position. The vulva and perineum are cleaned with antiseptic solutions and draped. .Aseptic catheterisation of the bladder is done and repeat vaginal examination performed to rule out anv contraindication to vacuum delivery such as cephalo-pelvic disproportion and malpresentation. A mediolateral incision (episiotomy) is made under local anaesthesia. The largest suitable vacuum cap is passed against the fetal scalp taking care not to include maternal soft issues by running a finger round the cap.

Suction pressure is then built up slowly at a rate of about 0.1 kg/cm^2 per minute up to a maximum of 0.8 kg/cm2. This allows for formation of an artificial chignon within the ventouse cap that holds firmly and allows adequate traction.

Traction is then applied with each contraction, in a downward direction until the head descends and then upwards to allow delivery by extension. On delivery of the fetal head the pressure is released. The rest of the delivery is completed as described above.

CAESAREAN SECTION

The commonest caesarean section is the lower uterine segment caesarean section. Classical section is rarely done.

Pre-operative care

Caesarean section operations are either emergency or elective. For elective caesarean section, baseline investigations like haemogram and urea and electrolytes are done, blood is taken for grouping and cross-matching and two units of blood are reserved and an informed consent for general anaesthesia and operation is taken. The patient is starved for at least six hours before the operation. The abdominal wall is shaved clean before theatre. Premedication with atropine 0.6mg is given intramuscularly half hour before theatre. For emergency caesarean section, blood is taken for grouping and crossmatch. The abdominal wall preparation is similar to that of elective operation. The patient is premedicated with atropine 0.6mg intramuscularly before being wheeled to theatre. For cardiac patients. 0.4mg of hyoscine is used instead.

Operation

In theatre the patient is placed in dorsal position with the legs separated, the vulva and perineum are cleaned with antiseptic solution such as savlon. Aseptic catheterisation is done and the catheter is left in situ after draining all the urine. A repeat vaginal examination is done.

The anterior abdominal wall is cleaned with antiseptic solution and painted with iodine. The patient is then draped with sterile towels. Anaesthesia is induced with intravenous sodium thiopental. Succinylcholine 50 -80mg is also given for temporary muscle relaxation to enable endotracheal intubation. Anaesthisia is then maintained with nitrous oxide, oxygen and halothane.

The abdomen is opened in layers either through a lower midline incision or through a Pfannestiel incision depending on the surgeon's and/ or patient's preference. After opening the skin, the rectus sheath is opened with curved Mayo's scissors. One side of the divided rectus sheath is elevated with two artery forceps and the rectus muscle separated from their attachment to it. using a surgical blade

and then drawn to one side to expose the peritoneum from their attachment to it. The latter is held in between two long artery- forceps and opened. The incision is extended up and down to the incision limits taking care not to injure the bladder.

Wet abdominal packs are placed on either side of the uterus to prevent blood and liquor from running into the general peritoneal cavity. A Doyen's retractor is applied to reflect the bladder away as well as expose the utero-vesical fold of peritoneum

The utero-vesical peritoneum is lifted up with a pair of dissecting forceps and incised. The incision is extended in an elliptical fashion downwards. The peritoneum is stripped off the lower uterine segment with mounted swab. The Doyen's retractor is shifted to include the lower part of the peritoneal fold in retraction of the bladder away from the lower uterine segment.

A small incision of about 2cm is made in the lower segment about 2 cm below the uterine' attachment of the utero-vesical peritoneal fold. Once the membranes are reached or uterine cavity- opened the incision is extended laterally on either side using curved scissors directed by two fingers of the left hand. The incision is enlarged enough to allow delivery of the head and trunk. The Doyen retractor is then removed and the right hand is introduced into the uterine cavity under baby's head which is delivered gently out through the uterine incision. Delivery is aided by gentle trans-abdominal fundal pressure. After delivery of the head, the mouth and nostrils are wiped with soft gauze. The shoulders are then delivered using gentle traction and still with some fundal pressure. The trunk follows readily. The umbilical cord is divided between clamps and the baby is handed over to a midwife or paediatrician. The placenta is delivered by either controlled cord traction or manually. The inside of the uterus is wiped with a swab on a holder. Bleeding margins of the incision are held by Green Armitage clamps. In transverse lie or breech presentation, the baby is delivered by breech extraction.

The uterine incision is then repaired in 2 layers with chronic catgut stitch No 2 on a traumatic needle. The utero-vesical peritoneum is then closed with a continuous chromic catgut stitch No 1/0. The abdomen is mopped and the abdominal packs are removed. The pelvic viscera is then inspected for any abnormalities. Instruments and swabs are counted and if they tally with the initial count, the abdomen is closed in layers or en mass using No. 1 vicryl. Peritoneum is closed with continuous No 1/0 Chronic catgut stitch, rectus sheath is similarly closed with No 1 vicryl stitch and skin with

interrupted silk or nylon. The wound is cleaned and then dressed. The catheter is checked for the urine draining and if clear the catheter is removed and the uterus is massaged and clots evacuated from the vagina.

General anaesthesia is reversed with 1.2mg of atropine and 2.5mg of neostigmine intravenously Extubation is done and oropharyngeal suctioning done

Post-caesarean care

The vital signs: blood pressure, pulse rate, respiration and body temperature are observed continuously until the patient is fully awake then 4 hourly. Intravenous fluids are given until she can take orally. Intramuscular pethidine 50-100 is given every 4 hours for the first 48 hours to relieve the pain. She is also given antibiotics crystalline penicillin 2Mu 6 hourly and gentamicin 80mg 8 hourly intravenously. Metronidazole is added to those at risk of sepsis. On the first post-operative day the patient is ambulated and oral sips started if bowel sounds present. When she starts taking orally medications are converted to oral medicines, on the third post-operative day haemoglobin is checked. The stitches are removed after seven days of operation. The patient is discharged home with a case summary and having been explained to about the nature and findings of operation and wound care. She is booked to be seen inn the post-natal clinic after six weeks

Care of the newborn

All the newborn babies who are normal join their mothers after delivery unless the mother is moribund. The babies with problems or where complications are anticipated together with babies delivered by operative vaginal delivery or by caesarean section are all reviewed by a paediatric registrar. Those having problems or who may develop problems are transferred to New Bom Unit (N B U). The premature babies are managed in NBU until their weights are about 2000gms when they are discharged. All babies are immunised with BCG (Bacille Calmette Guerin) and oral polio vaccine before discharge. Normal mothers who have babies in NBU are lodged in the mother's hostel.

Post-natal follow-up

The clinic is held on every Friday from 9.00am Only those patients who had complications or operative delivery are seen. Patients with normal deliveries are followed up in their nearest health facility.

The blood pressure and weights are taken. Urinalysis is performed. History is taken of the puerperium, lactation and immunization of the baby. The patient is then examined and any problems managed. Family planning advice is given and the patient referred to the family planning clinic for the various methods available.

THE GYNAECOLOGY UNIT

The Gynaecology unit consists of the outpatients wing at clinic 18 and two gynaecological wards 1 B and ID on the first floor of the tower block.

Ward ID is the acute gynaecological ward while IB caters for non-emergency cases. The unit is managed by the three firms in the department.

Gynaecological outpatient services

There are three outpatient clinics per week. Specific firms run the clinics on different days; Firm I on Tuesdays, Firm II on Thursdays and Firm III on Wednesdays. The clinics are run by consultants, senior registrars and registrars. Teaching of the medical students takes place in the clinics. There is also a colposcopy and oncology clinic on Friday morning. The infertility clinic is conducted on Mondays from 2.00pm

The majority of the patients attending the gynaecology clinic are referred from casualty and emergency gynaecology ward after emergency management. Post-operative patients also attend this clinic. Some other patients are referred from the district and provincial hospitals.

Infertility patients constitute about two thirds of the gynaecology consultation followed by uterine fibroids, abnormal uterine bleeding and oncology patients. In the clinic a thorough history and physical examination is conducted and most of the diagnostic investigations are done. The investigations requested for depend on the diagnosis after history and physical examination. Some of the investigations include: pelvic ultrasound, hvsterosalpingoram. seminalysis, renal function, liver function, haemogram and hormonal assays among others.

Acute gynaecological admissions -ward ID

This is the emergency- gynaecological ward where patients are admitted through casualty . It caters for all gynaecological emergencies seen and admitted at the Kenyatta National Hospital. An average of 15 patients are admitted daily and more than two thirds of these cases are abortion. Patients are mainly admitted through the casualty department.

All patients for admission are clerked by the houseman and reviewed by the senior house officer (registrar) who undertakes the management in consultation with senior members of the department. Patients in the ward are reviewed daily by the registrar, senior registrar and consultant.

Apart from abortions, pelvic inflammatory disease and ectopic pregnancies are the next most common cases admitted into this ward. Uncomplicated cases of incomplete abortion have uterine evacuation done in the procedure room in ID using Karman's canula and syringe. They ai'e discharged immediately after being counselled about contraception. Patients who have undergone emergency laparotomy for pelvic abscesses, ectopic pregnancy or pelvic masses have a minimum stay of four days post-operativelv.

Patients with suspected carcinoma of the cervix are usually not admitted unless they have complications such as bleeding or severe anemia requiring transfusion. Examination under anaesthesia staging and biopsy is usually done as an outpatient procedure. Patients are then referred to ward IB if surgery is required or straight for radiotherapy.

Cold gynaecology admissions-ward IB

Ward IB is the non-emergency gynaecology ward to which patients are admitted from the clinic or transferred from acute gynaecology ward for further management. The ward has a bed capacity of 33 beds. The beds are shared equally among the three firms. The patients commonly admitted in this ward are those for elective gynaecology operations or for chemotherapy due to gynaecological malignancies. Uterine fibroids, vesico-vaginal fistulae (VVF), tubal infertility and gynaecological malignancies are among the commonest conditions necessitating patients to be admitted to this ward.

GYNAECOLOGICAL OPERATIONS

A theatre is reserved in main theatre for emergency gynaecological operations daily. Laparotomy for ectopic pregnancies (rupture and non-rupture), Pelvic abscesses, ovarian cyst and other tubo-ovarian masses are done here. Smaller procedures like diagnostic dilatation and curettage of the uterus, removal of misplaced intra-utenne contraceptive devices, marsupilization of bartholin's abscess and suction curettage are also performed here.

Flective operations are done on firm basis. Firm II doing theirs on Mondays and Firms I and III doing theirs on Thursdays. The operations are done between 8.00 am to 5.00 p.m Most of the operations are performed under general anaesthesia as outlined below:-

- Intravenous sodium thiopentone and succinylcholine are used for induction of anaesthesia.
- Nitrous oxide, oxygen and halothane provide maintenance anaesthesia.
- Curare is given intermittently for muscle relaxation.
- Atropine and neostigmine are used for reversal.

Some operations are done under light anaesthesia or under local anaesthesia

Pre-**operative preparations**

Patients for emergency laparotomy are prepared for theatre on admission. Ruptured ectopic pregnancies are the most common indications for emergency laparotomy. In this case blood is urgently cross-matched and an intravenous drip of N/saline started. The abdomen is shaved in the ward. Pre medication is provided by atropine 0.6 mg intramuscularly before theatre. For cold (non-emergency-) operations, baseline investigations such as the full haemogram. urea and electrolyte levels are done prior to the date of admission for surgery. The nature and purposes of the operation is explained to the patient and an informed written consent for the operation is obtained Enema is usuallv given at 6.00pm the day before surgery and at 6.00am on the day of surgery. Pre-medication is provided bv atropine 0.6 mg and pethidine 50-100mg intramuscularly half hour before theatre.

Post-operative management

After the operation general anaethesia is reversed and the patient wheeled to the recovery room where continuous observations of blood pressure, pulse rate, respiratory rate and temperature are monitored until she is fully awake and stable. She is then transferred to the ward where observations are done 4 hourly.

Patients who have had laparotomy for hysterectomy, ectopic pregnancy, ovarian cyst etc are usually kept in the ward for 4 days. For the first 24 hours the patients are maintained on intravenous fluids Oral fluids are given when bowel sounds are established. Blood transfusion is given when indicated. Pethidine 50-1 OOmg 6 hourly for 124 to 48 hours is routinely given for analgesia Prophylactic antibiotics are given routinely. A check hemoglobin level is determined on the third post-operative day.

Before discharge, the patient is informed about the findings at operation and a discharge summary is issued. Patients are reviewed in the gynaecology clinic after six weeks or earlier when there is an indication.

Total abdominal hysterectomy

General anaesthesia induction and maintenance are done as described above. A vulvo-vaginal toilet is done with antiseptic solution such as hibitane or savlon. Aseptic catheterisation is done next and the catheter left in situ to maintain continues bladder drainage during the operation. Pelvic examination under anaesthesia is done and findings noted. The vagina is then painted with methylene blue dye. The abdomen is thoroughly cleaned with savlon and painted with iodine and then draped with sterile towels.

The abdomen is opened in layers either through a Pfannestiel incision or through a lower midline incision. The intestines are packed away from the intestines with wet gauze packs and a self-retaining retractor applied. The round ligaments are identified and beginning on either side using straight long artery forceps the round ligament is double clamped and divided between the two forceps. He lateral stump is transfixed with No 0 or No 1 vicryl. This procedure opens the anterior leaf of the broad ligament, which is pushed forwards through this opening with a surgeon's finger and incised with scissors. The same is done for the opposite side.

next step depends on whether the tube and the ovary are to be saved or removed. If they are not to be saved, the tube and the ovarian ligament are double clamped en masse and cut using a scalpel. ~~The distal clamp holds the ovarian vessels as they approach the anastomosis with the uterine -s. essels. This stump is ligated using a transfixed vicryl No. 1 or No. 0. The same is done for the <r?pposite side. If the tube and ovary are to be removed with the uterus, the infundibulo- pelvic •portion of the broad ligament is double clamped with long curved artery forceps with the tips reaching the open window in the broad ligament. The ligament together with ovarian vessels are •divided between clamps and ligated using vicryl No 1 or No 0. The same is done for the opposite side.

The reflection of the bladder peritoneum onto the uterus is then freed by extending the incision in the anterior leaf of the broad ligament towards the midline. The bladder is thus separated from the lower uterine segment, the cervix and the vagina by careful sharp and blunt dissection of the fascial fibres beneath the bladder wall. Usually the bladder can be displaced into the lower pelvis quite easily, but if it is adherent, it is surgically released and not bluntly forced.

Next the posterior leaf of the broad ligament on either side is cut parallel with the side of the uterus to better demonstrate and skeletonise the uterine vessels between the leaves of the broad ligament for clamping. The uterine vessels are double clamped and cut using a scalpel and freed from the uterus by extending the incision around the tip of the distal clamp. This enables adequate ligation. Care should be taken to avoid freeing the tissue beyond the tip of the clamp, as this could permit bleeding from the collateral vessels that are not included in the clamp. Before clamping and cutting the uterine vessels, it is always advisable to palpate the lower portion of the pelvic ureters as they course beneath the uterine artery lateral to the internal OS and pass medially through the base of the broad ligament to the trigone of the bladder. The uterine vessels are ligated with vicryl No 1. The same is done for the opposite side.

The uterus is retracted forward and upward to demonstrate and stretch the uterosacral ligaments posteriorlv. A transverse incision is made through the uterine reflection of the cul-de-sac peritoneum between the attachments of the two uterosacral ligaments. The peritoneum is then incised with the scalpel and reflected, mobilizing it past the cervix to the posterior vaginal formic. Care is taken not to dissect extensively laterally where the haemorrhoidal vessels are inserted into

the rectum Each utero-sacral ligament is double clamped, cut and ligated with a No 1 vicryl suture. Here particular care is exercised to avoid the pelvic portion of the ureter as it courses along the base of the broad ligament. Next the cardinal ligaments on either side of the uterus are clamped, cut and ligated.

More commonly the uterus is removed by the opening in the anterior vaginal fornix, initially with the scalpel and then the vagina is circumcised by a sharp knife or scissors. As the anterior, posterior and lateral margins of the vagina are opened, straight artery forceps are used to secure vaginal margins. These margins are then closed using a series of figure-of-eight sutures. Particular care is taken when tying the lateral angles to ensure the descending vaginal branches of the uterine vessels are securely ligated.

Suspension of the vaginal vault is done by tying the peritonization suture to the lateral and mid sutures of the vault. Peritonization is accomplished by means of a continuous No. 1. chronic suture, that first pierces the vaginal walls near the midline and passes through the posterior leaf of the broad ligament, the free margin of the uterosacral ligament and the anterior bladder peritoneum. The suture is tied at the centre. The same is done for the opposite side with the suture being tied at the midline and lateral angles. If the ovaries have been preserved an alternative suspension may be used in which the tip of the broad ligament is stitched separately with a purse string of No 2/0 chronic catgut. The free margin of the pedicle is left high against the pelvic wall and is not anchored to the vaginal vault. This is advised in order to avoid subsequent dvspareunia and avoid stitching of the ovarian vessels with possible thrombosis, ischemia and cystic changes of the ovary. After this abdominal viscera are well inspected. If haemostasis has been achieved, and instruments and swabs count are normal, the abdomen is closed in anatomical layers.

The post-operative management is as described above.

COUNSELLING CLINICS

There are three such in the hospital, which offer counselling to obstetrics and gynaecology patients. These are the patient support centre. GOPC, teenage clinic and the Nairobi Hospice.

The patient support centre

This is situated in the old hospital buildings where patients regularly attend from all the departments of the hospital. Sometimes the counsellors are called to the wards to counsel those patients who cannot go there. The counsellors consist of psychiatrists, sociologists, psychologists and trained nurses. Mostly, they deal with HIV counselling, puerperal psychosis patients and those patients who are poor and neglected by relatives. They counsel, treat and even assist patients find their way home.

The high risk clinic (HRC)

This clinic is situated on the ground floor next to the maternity wards. It deals with young single mothers who have had an abortion, those who have delivered babies and even those who do not wish to rear their children. The counsellors are also trained nurses, sociologists and consultant obstetrician/gynaecologists.

They counsel their clients, treat them for any illness they may have with assistance from the obstetric and gynaecology wards, and also provide them with family planning and STD management services. The patients come from other institutions or from the obstetrics and gynaecology wards.

The Nairobi hospice

Workers here also offer counselling care in addition to management of terminal disease. They also offer narcotic analgesia and encourage home based care for such patients instead of hospital care. Most of their patients have cancer of the cervix.

THE HOSPITAL CHAPEL

This provides spiritual nourishment to those who are in need. It is situated on level 2 of the tower block.

THE MOTHER'S HOSTEL

This accommodates mothers with babies in nursery. When they get sick, they are treated from the wards where they were initially admitted.

OBSTETRIC CASE No. 1

FETAL DISTRESS: EMERGENCY CAESAREAN SECTION- LIVE BAY

NAME:	S.K	DOA:	16.03.2004
AGE:	30 YEARS	DOD:	20.03.2004
IP. No:	9908643	LMP:	18.06.2003
PARITY:	2+0	EDD:	25.03.2004
		GBD	39 WEEKS

PRESENTING COMPLAINT

S.K was admitted with labor pains for the last 6 hours. There was no drainage of liquor and no vaginal bleeding.

HISTORY OF PRESENT PREGNANCY

She attended antenatal clinic at Kenyatta National hospital from 24 weeks gestation. The antenatal profile was as follows: hemoglobin 12.8g/dl, VDRL negative, HIV negative and blood group A positive. The antenatal period was uneventful.

OBSTETRICS AND GYNECOLOGY HISTORY

She was a para 2+0 gravida 3. Her LMP was 18.06.2003 with an EDD of 25 03.2004 giving her a gestation by dates of 39 weeks. Her first delivery was in 1995, SVD in hospital to a live female infant 3000gm who wass alive and well. Her second delivery was in 1999 hospital SVD a live male infant 3100gm who is alive and well. She had used oral contraceptives. Her menstrual cycle was a regular 26 day cycle lasting 3-4 days with no dysmenorrhoea or menorrhagia.

FAMILY AND SOCIAL HISTORY

She was a housewife, the husband was a security officer. She had no chronic illness or family history of chronic illness. She neither smoked nor drank alcohol.

PAST MEDICAL HISTORY

This was not contributory.

GENERAL EXAMINATION

She was in good general condition not pale, not jaundiced and not cyanosed. Her temperature was 36.7 °C, blood pressure 110/75mmHg, pulse rate of 95 beats per minute and a respiratory rate of 22 per minute.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and non tender. The fundal height was term, the lie longitudinal and the presentation was cephalic. The fetal heart rate was rapid at 160 beats per minute but with no irregularities

PELVIC EXAMINATION

She had normal external genitalia, the cervix was 5 cm dilated and fully effaced, membranes were intact with mild bulge, descent was 2/5 and the pelvis felt adequate. There was no caput or moulding.

The other systems were essentially normal.

Artificial rupture of membranes was done and thick meconium stained liquor was obtained.

IMMPRESSION

An impression of fetal distress in early labour was made.

MANAGEMENT

She was put in left lateral position and started on oxygen by mask and a drip of 500ml 10% dextrose. She was informed of the diagnosis and the need for caesarean delivery She accepted and signed an informed consent and blood was taken for grouping and crossmatch. She was then taken to theatre for emergency caesarean section.

INTRAOPERATIVE FINDINGS

Caesarean section was done as outlined in the earlier section of this book but through a pfannestiel incision. A live female infant was extracted with tight cord round the neck two times. Apgar score was 5/1 7/5 and 8/10. Thick meconium stained liquor was obtained. The placenta was grossly normal. The infant was resuscitated by the paediatrician and taken to the newborn unit. The mother had an uneventful operation and reversed from GA and taken to the antenatal wards.

POSTOPERATIVE

The infant was admitted into the newborn unit for one day and was given oxygen and antibiotics and did well subsequently and was discharged to join the mother. On the fourth post-operative day both the mother and baby were stable and were discharged home to be seen again in the postnatal clinic in three weeks.

POSTNATAL VISIT

She was seen in the postnatal clinic and was well and the baby was also doing well. The wound had healed well and there was no abdominal tenderness. The baby was breastfeeding well. She opted to use oral contraception and was discharged from the postnatal clinic to book an appointment in the family planning clinic.

DISCUSSION

S.K had thick meconium in early labor and persistent fetal tachycardia which together implied fetal reaction to stress hence necessitating delivery by emergency caesarean section.The outcome was a live baby with good Apgar score.

Fetal distress may be defined as a complex of signs indicating a critical response in the fetus to stress. It implies metabolic derangements notably hypoxia and acidosis that affect the functions of vital organs to the point of temporary or permanent injury or death (1).

The causes of fetal distress are varied. Maternal causes include decreased uterine blood flow such as in maternal acute hypotension, shock or cardiac failure. Cases of maternal hvpoxia from any cause, hypercapnia, hypertonic uterine contractions or tetanic contractions can lead to acute fetal compromise.Conditions such as abruptio placentae, placenta praevia, lack of sufficient placental reserve to tolerate labor(postmaturity,premature placental ageing), ruptured vasapraevia and cord accidents (knots, prolapse, entanglements) could lead to fetal distress. Other maternal causes of chronic fetal compromise include diabetes mellitus, chronic hypertension, pre-eclampsia/eclampsia which lead to pelvic and placental vascular disease. Fetal causes of distress include multiple gestations, postmaturity, congenital anomalies, congenital infections and erythroblastosis fetalis.

In our patient a tight cord two times round the neck was found and this was inferred to be the cause of the fetal distress.

The true incidence of fetal distress is unknown due to controversies in diagnosis (2,3). The Nairobi Birth Survey (1983) found a prevalence of 5.2 % at KNH (4). In western countries the reported incidence ranges from 4.5 % to 9.3 % (5).

The diagnosis of fetal distress based upon fetal heart rate patterns is imprecise and controversial (6). Experts in interpretation of these patterns so often disagree with each other as to whether a certain pattern is normal, suspicious or pathological (6). Abnormal cardiotocographic (CTG) suggestive of fetal distress include:

1. Abnormalities of fetal heart rate

   a. Bradycardia: A 10 minute baseline FHR of less thanl 10 bpm

      i) Mild bradycardia: 100-119 bpm

      ii) Moderate bradycardia: 80-100 bpm

      iii) Severe bradycardia: less than 80 bpm in 3 mins (6)

   Bradycardia of 100-119 bpm in the absence of other changes is usually not considered to represent fetal compromise. It can be due to head compression from occiput posterior or transverse positions particularly during second stage labor Other causes of fetal bradycardia include maternal hypothermia, use of beta blocker therapy, congenital cardiac conduction defects and placental insufficiency as in abruptio placenta.

   b. Tachycardia: FHR of more than 160 bpm

      i) Mild: 160-180 bpm

      ii) Severe: More than 180 bpm (6)

Tachycardia may be associated with maternal fever, maternal hyperthyroidism, amnionitis, use of drugs, fetal hypovolemia, fetal heart failure or fetal hypoxia. Fetal tacchvarrthmias may cause fetal tachycardia. Tachycardia may be an early sign of fetal distress.

2. Abnormalities of rhythmn

   a. Late Decelerations: A late decelation is a smooth gradual symmetrical decrease in fetal heart rate beginning at or after the peak of the contraction and returning to the baseline after the contraction has ended. Any process that causes reduced uteroplacental gas exchange could lead to late decelerations.

   b. Reccurrent Variable Decelerations: This is defined as a visually apparent abrupt decrease in FHR. The onset of deceleration commonly varies with successive contractions . The duration is less than 2 minutes. The American College of Obstetricians and Gynecologists has defined significant variable decelarations as those decreasing to less than 70 bpm and lasting more than 60 seconds (7). Variable decelarations are due to different degress of cord compression during labor.

c. Others: Other fetal heart rate patterns associated with fetal distress include, saltatory and sinusoidal baseline heart rates, prolonged decelarations and fetal cardiac arrythmias.

Ayre-de-Campos and colleagues (1999) using inter-observer interpretations of FHR patterns found out that experts agreed only on 62 % of normal patterns, 42 % of suspicious patterns and only 25 % of pathological patterns (8)

Dellinger and colleagues (2000), using a combination of zero variability plus late or moderate-severe decelerations or baseline heart rates less than 110 bpm for 5 minutes to define fetal distress were able to predict normal outcomes for fetuses as well as discriminating true fetal distress (9)

Our patient had persistent tachycardia which was inferred to be an early sign of fetal distress.

After more than 30 years of experience with interpretation of fetal heart rate patterns, there is finally emerging evidence that some combinations of fetal heart rate characteristics can be meaningfully used to identify normal and severely compromised fetuses. True fetal distress patterns appear to be those where beat-to-beat variability is zero in conjunction with severe decelarations or persistent baseline changes or both (6)

J Whitridge Williams (1903) observed that a characteristic sign of impending asphyxia is the escape of meconium (10). Obstetricians, however have long realized that the detection of meconium during labor is problematic in the prediction of fetal distress or asphyxia (11)

Meconium passage by the fetus in-utero may signify normal gastrointestinal tract maturation under neural control. Meconium passage could also follow vagal stimulation from common but transient umbilical cord entrapment and resultant increased gut peristalsis (12). The pathological explanation proposes that fetuses pass meconium in response to hypoxia and that meconium therefore signifies fetal compromise (13)

Miller (1975) observed that perinatal mortality with meconium without other factors was 4-5 % but with other factors was 18.4 % especially with FHR abnormalities (14). Fongoh (1984) found that thick meconium staining of amniotic fluid was associated with lower Agar scores at 1 and 5 minutes and perinatal mortality was 80 per 1000 births in thick meconium stained amniotic fluid. However he found that light staining of amniotic fluid with meconuim was unpredictable in determining fetal distress (15).

Ramin and co-authors (1996) studied almost 8000 pregnancies with meconium-stained amnionic fluid at Parkland Hospital. They concluded that the high incidence of meconium observed in amnionic fluid during labor often represents fetal passage of gastrointestinal contents in conjunction with normal physiological processes. Such meconium however can become enviromentally harzadous especially with concomintant fetal acidemia and can lead to meconium aspiration syndrome (16).

Our patient had thick meconium stained amnionic fluid together with a non-reassuarring FHR which necessitated delivery by emergency caesrarean section.

Other available clinical methods for detecting impending or actual fetal asphyxia include quantification of fetal movements, fetal response to a stimulation (fetal scalp stimulation, or vibroacoustic stimulation), biophysical profile (BPP), contraction stress test and fetal scalp blood sampling. Other methods include fetal pulse oximetry continuous fetal tissue Ph measurements and percutaneuos umbilical blood sampling.

In cases of possible fetal compromise vaginal examination should be done to assess for rapid progression or cord prolapse. Intrauterine resuscitation may be accomplished by changing the position of the mother, correcting maternal hypotension by intravenous fluid administration, stopping administration of oxytocin, administration of tocolytics . administration of oxygen by mask at 10 Litres/ min and use of 10 % dextrose drip. In our patient all these were done except the administration of tocolytics.

Labor may be continued in the presence of reassuring signs of fetal status through fetal acoustic stimulation, scalp stimulation, or fetal scalp blood sampling. If fetal well-being cannot be documented, if the situation worsens, if the signs of probable fetal distress

persist for 30 minutes, of if there is continuous distress despite conservative treatment immediate delivery is indicated. Obstetric judgement must dictate how the delivery will be accomplished in accordance with the presentation, station, position, dilatation of cervix and presumed fetal status. Our patient underwent emergency caesarean section because she was in early labor with thick meconium stained amnionic fluid with persistent fetal tachycardia.

Our patient did not receive tocolytic therapy since it is not routinely used in our set-up. A single intravenous or subcutaneuous injection of 0.25 mg of terbutaline sulfate given to relax the uterus has been described as a temporizing maneuver in management of fetal distress (17).

Cook and Spinnato (1994) observed that tocolysis with terbutaline for fetal resuscitation improved fetal scalp pH values although all the women in their study were delivered by caesarean section (18). Other tocolytic agents are slowly getting their place in the management of fetal distress in labor (19).

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OBSTETRIC CASE No. 2

SICKLE CELL DISEASE-FAVOURABLE OUTCOME.

NAME:	B AO	DOA	26.11.2004
AGE	18 YEARS	DOD	06.12.2004
IP NO	0996095	LMP	15.03.2004
PARITY	0+0	EDD	22.12.2004
		MBD	36+WEEKS

PRESENTING COMPLAINT

B AO was admitted in labor ward with complains of labor pains for the last 6 hours, chest pain for the last one day and right lower limb pain for one day.

HISTORY OF PRESENTING COMPLAINT

She started experiencing regular abdominal pains 6 hours prior to admission at intervals of 10-15 minutes which were increasing in intensity. She also noticed thick mucoid discharge from the vagina.There was no drainage of liquor or vaginal bleeding. She had no dysuria nor frequency of micturition.

She experienced chest pain a day prior to admission which was confined to the sternum and left middle ribs. The pain was dull and intermittent. It was non-radiating and was aggravated by palpation. There was no cough, no haemoptysis or difficulty in breathing. There was no history of fever She also had left lower limb pain which was insidious in onset and was dull and deep which she described as "deep bone pain" There was no limb swelling and the gait and joint movements were normal.

PAST MEDICAL AND SURGICAL HISTORY

She was a known patient with sickle cell disease diagnosed when she was 10 years old. She had been admitted twice at the Aga Khan Hospital Nairobi in 1995 and in 1998 due to painful crisis as a result of sickle cell disease. She also suffered chronic suppurative osteomyelitis of the right femur in 1999 and was admitted at the Forces Memorial Hospital where she underwent sequestration. She had been on paludrine 50mg once a day and folic acid 5mg once a day.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a para 0+0 gravida 1. Her last menstrual period was on 15.03.04 with an expected date of delivery of 22.12.2004 giving her a maturation of 36+ weeks gestation by dates at the time of admission. She attended antenatal clinic at Kenyatta National Hospital Her blood group was B positive, VDRL negative, Hb-12.8g/dl and HIV test was negative. Her antenatal period was uneventful. She had her menarche at 13 years and her menstrual cycles were regular every 28 days with a flow of 3 days. There was no associated dysmenorrhoea. She had not used any form of contraception

FAMILY AND SOCIAL HISTORY

She was a single form four leaver. She was unemployed and lived with her mother who was a businesswoman. She had two siblings who were alive and well and were not sicklers. There was no history of chronic illness in the family. She neither smoked cigarettes nor took alcohol.

GENERAL EXAMINATION

She was in fair general condition. She was not pale or jaundiced, no lymphadenopathy but she was mildly dehydrated. Her blood pressure was 125/70 mmHg, pulse rate of 84 beats per minute, respiratory rate of 22 per minute and temperature of 36.8C.

ABDOMINAL EXAMINATION.

The abdomen was uniformly distended. The fiindal height corresponded to 36 weeks gestation. The lie was longitudinal and the presentation cephalic. The fetal heart rate was 140 beats per minute and was regular. She had 2 mild contractions every 10 minutes lasting approximately 20 seconds each. There was no hepatosplenomegaly.

PELVIC EXAMINATION.

She had normal external genitalia A digital vaginal examination revealed a cervix which was central, the cervical os was 2cm dilated and 80% effaced, it was soft approximately 0.5cm long. The descent was 4/5, the pelvis felt borderline. There was show on examination fingers.

RESPIRATORY EXAMINATION.

She had normal respiratory effort with a respiratory rate of 22 breaths per minute. There was mild tenderness on palpation of left 2^{nd} 3^{rd} $4^{,h}$ and 5^{th} ribs. The trachea was central. The chest expansion was normal. The percussion note was tympanic and on auscultation normal bronchovesicualr sounds were heard.

MUSCULOSKELETAL SYSTEM.

She had a surgical incision scar on the lateral aspect of the right thigh. She had a healed sinus scar on the medial aspect of the lower 1/3 of right thigh but there was no swelling. There was slight muscle wasting of the right thigh otherwise the joint movements were normal. The left lower limb was normal.

The cardiovascular and the central nervous system were essentially normal.

DIAGNOSIS

Latent phase of labor in a known sickler in painful crisis.

MANAGEMENT

She was admitted into labor ward and started on intravenous 5% dextrose to alternate with half strength darrows. She was also started on intravenous crystalline penicillin 2 mega unit six hourly and gentamicin 80mg intravenously 8 hourly and was also given a stat dose of pethidine lOOmg intramuscularly and to continue with pethidine lOOmg 8 hourly. She was given oxygen by mask. A hematologist was consulted and added subcutaneous heparin 7500 IU 8 hourly, Junior aspirin 75mg once a day and neurobion one tablet 8 hourly. She was also to continue with paludrine lOOmg and folate 5mg both once a day. Several investigations were ordered requested for and a summary is given below;

HAEMOGRAM

W.B.C	11.8 x 10/L	Na+	136 mmol/L
R.B.C	5.19 x 10/L	K+	4.0 mmol/L
Hb	10.9 g/dl	BUN	1.3 mmol/L
Platelets	112 x 10/L	Creatinine	71 umol/L
MCV	68 um		
HCT	35.5 %		
MCHC	21.1 pg		

Blood Slide for malaria parasites- Negative.

Widal test- negative

Urinalysis- Normal.

Random Blood Sugar - 5.2 mmol/L

LIVER FUNCTION TESTS

Total protein-	68g/dL
Albumin-	24
ALT-	37
AST-	31
ALP-	312
Total Bil.	6.0
Direct Bil.	1.3

PROGRESS IN LABOR WARD.

She was examined 3 hours later and the cervix was found to be 5cm dilated. ARM was done and clear liquor obtained. She was having moderate contractions 3 in 10 minutes lasting 30 seconds each. Descent was 3/5. A decision to augment her with syntocinon was made. She progressed well and 6 hours later she delivered SVD to a live male infant who scored 9/1 and 10/5 with a birth weight of 2700gm.

She was observed in labor ward for 12 hours then transferred to the postnatal ward.

She continued with her medication and was discharged on the 4th postnatal day to be seen in both the postnatal clinic and hematology clinic in 3 weeks.

POSTNATAL

She was stable and the baby was breastfeeding well. She had no major complaint and was referred to the hematology clinic for follow-up.

DISCUSSION

The patient was a para 0+0 known sickler who presented in labor in painful crisis. She gave birth to a live male infant who scored well. She had a satisfactory postnatal recovery and was discharged home.

Sickle cell hemoglobin (hemoglobin-S) results from a single B-chain substitution of glutamic acid by valine, because of Adenine for Thiamine substitution at the codon 6 Of the B-c globin gene.(l). Valine is hydrophobic while glutamic acid is hydrophilic and therefore HbS is less soluble in blood. When oxygen concentration is low, HbS, polymerizes, therefore forming tactoids, which make red blood cells to sickle. Sickling is however reversible if reduction of oxygen tension is only for a short period. Sickle cells are particularly liable to splenic sequestration and hemolysis, hence the chronic hemolytic anemia (1).

The gene causing sickle cell disease is inherited as an autosomal recessive trait irrespective of sex. Inheritance of the gene responsible for the production of HbS from both parents results in sickle cell disease and from one parent results in sickle cell trait. Sickle cell anemia (SS disease), sickle cell hemoglobin C disease (SC disease) and sickle cell-B-thalassemia disease(S-B-thalassemia disease) are the most common of the sickle cell hemoglobinopathies. Maternal and perinatal outcomes are altered markedly in women with sickle cell anemia. These adverse outcomes are related to the vascular complications of sickling but not the anemia. Maternal morbidity and mortality are all increased with these hemoglobinopathies (1.2).

Prevalence and incidence of sickle cell disease (SCD) varies both geographically and racially. In the U S 1 in 12 African-Americans has the sickle cell trait. The theoretical incidence of sickle cell anemia among African-Americans is 1 in 576, but the disease is not so common in pregnancy because of an earlier high mortality rate, especially during early childhood (1). In Sub-Saharan Africa 1-2% of infants are born with SCD (3). In Kenya, the disease is frequent in Nyanza, Western and Coast Province and is second to malaria as a cause of anemia. The patient presented was from Nyanza province.

Pregnancy is a serious burden to women with hemoglobinopathies. This is especially true for those with hemoglobin SS disease in whom the anemia often becomes intense, sickle

cell crises usually become more frequent and infections and pulmonary complications are more common (1). Sickle cell trait (HbAS) results in no detectable abnormality and crises are rare.

Hypoxia, acidosis, infection or dehydration may precipitate sickle cell crises and may be life threatening (1). Shortly before and after delivery, these patients are liable to severe bone pain crises that may be complicated by marrow and bone embolus and systolic hypertension with albuminuria" pseudotoxaemia" (1,4). A particular worrisome pulmonary complication is related to embolization of necrotic bone marrow fat and cellular debris and acute respiratory insufficiency may develop (1).

These patients also have high frequencies of urinary tract and other infections during pregnancy and in the puerperium, they are liable to infection especially wound sepsis (4).

Sickle cell disease in pregnancy puts the fetus at some risks like, genetic transmission, abortion, still birth, growth retardation, and preterm pregnancy.

Perinatal mortality can be as high as 33% but may be reduced to around 10% with good antenatal care and careful supervision of delivery and puerperium (4). The outcome for both mother and baby will improve with intense antenatal supervision by a team of obstetrician and hematologist.

Clinically the hallmarks of sickling episodes are periods, during which there is ischemia and infarction within various organs, " sickle crisis". In addition to painful crisis there may be aplastic, megaloblastic, sequestration and hemolytic crises (1). The patient presented had painful crisis.

Chronic and acute changes from sickling include bony abnormalities, renal medullary damage, autosplenectomy by adulthood in SS patients, splenomegaly in other variants, hepatomegaly, ventricular hypertrophy, pulmonary infarction, cerebrovascular accidents, leg ulcers, and propensity to infection and sepsis (1).

In absence of infection or nutritional deficiency, hemoglobin concentration, usually does not fall below 7g/dl. But any factors impairing erythropoesis or increasing red cell destruction or both aggravates the anemia.(l).

Screening consists of examination of blood for RBC indices (MCV), sickledex, hemoglobin electrophoresis, and Hb A2 and Hb F quantification. Management during pregnancy includes careful antenatal supervision and prophylactic antimalarials and supplemental folic acid (1,4). Supplemental folic acid lmg per day is given. Blood transfusion with red cell concentrates is indicated if the patient approaches obstetric delivery with Hb < 8g/dl. However prophylactic red cell transfusions and their use remain controversial, with benefits being slight if any and certainly outweighed by risk of complications from transfusion in the tropics (1,4,5).

Overt bacteriuria and acute pyelonephritis are increased considerably and careful surveillance for bacteriuria is important (6).

Pneumonia due to streptococcus pneumonia is common and the woman in advanced pregnancy may not tolerate severe pulmonary infections. Most authorities recommend polyvalent pneumoccocal vaccine for these women (1).

Acute infarction is usually accompanied by severe pain and because bone marrow is frequently involved, intense bone pain is common. Relief of pain is not afforded by heparinization or dextran.

Intravenous hydration is provided and for severe pain, meperidine or morphine are administered parenterally. Red cell transfusions administered after the onset of severe pain have no dramatic effect on the intensity or duration of the pain (1,7).

Because of the high incidence of fetal growth retardation and increased perinatal mortality, careful fetal assessment is necessary. Some workers have recommended weekly non-stress testing, beginning at 32 weeks, along with serial ultrasonography to monitor fetal growth and amniotic fluid volume.

Labor and delivery in women with hemoglobin SS disease should be managed the same way as for women with cardiac disease. The woman should be kept in propped-up or left lateral position, but not over sedated. Epidural anesthesia is usually ideal for pain relief but morphine or meperidine can be used. Compatible blood should be available. Obstetric delivery is often complicated by pelvic disproportion, the result of impaired growth during childhood, and in some African countries about half of the patient's are delivered by caesarean section. If a difficult vaginal or caesarean delivery is contemplated and the hematocrit is less than 20%, the hemoglobin concentration should be increased by packed erythrocyte transfusions, taking care to prevent circulatory overload, ventricular failure and pulmonary edema.

Newer therapies include, 5-azacytidine butyrate and hydroxyurea which selectively increases hemoglobin F production, and with increased hemoglobin F production, there is less sickling (8). The safety of these preparations in pregnancy is not yet clear.

Because of the chronic debility from sickle cell anemia, the more complications caused by pregnancy and shortened life span of women with sickle cell anemia, a sterilization or at least a very effective means of contraception is indicated even for women of low parity. Combined oral contraceptives are relatively contraindicated in women with sickle cell hemoglobinopathy because of the potential risk of thrombo-embolism. Intrauterine contraceptive devices are likely to increase the incidence of pelvic infections and should be avoided. Progesterone only pills, Norplant implants, Depo-Provera and barrier methods can be used (1).

Prenatal diagnosis of sickle cell disease through amniocentesis or chorionic villus sampling is available in some parts of the world, but not in our set-up. If prenatal diagnosis of sickle cell disease is made, genetic counseling and options can be given to a couple.

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OBSTETRIC CASE No. 3

ABDOMINAL PREGNANCY WITH FETAL DEMISE: LAPAROROTOMY

NAME:	W.N	DOA:	11.09.2004
AGE:	30 YEARS	DOD:	15.10.2004
IP.NO:	0986556	LMP:	16.04.2004
PARITY:	1+0 G 2	EDD:	23.01.2005
WARD:	GFB	MBD:	22 Weeks.

PRESENTING COMPLAINT.

The patient was admitted from home having had heavy per vaginal bleeding for the past 6 hours associated with lower abdominal pains. She also had cough and chest pain for the past one week.

HISTORY OF PRESENTING COMPLAINS.

She was well until one week prior to admission when she had cough which was productive of mucopurulent sputum but no haemoptysis. Cough was associated with generalized sharp chest pains. She took a cough syrup and analgesics and some capsules which were antibiotics. Six hours prior to admission she had heavy per vaginal bleeding with clots then immediately associated with lower abdominal pain. There was no history of trauma or excessive physical activity. No frequency or dysuria. No drainage of liquor

PAST MEDICAL HISTORY.

Non contributory

PAST OBSTETRIC AND GYNAECOLOGICAL HISTORY.

She was para 1+0. The last delivery was in 1996 via spontaneous vertex in a hospital to a live female infant who had a birth weight of 2900 grams and was still alive and well. Her menarche was at 15 years. Her menstrual cycles had been regular with a cycle length of 28 days and a flow duration of 3-4 days. There was no menorrhagia nor dysmenorrhoea.

She had used oral contraceptive pills from 1997 to 2003 when she stopped since she desired another child.

PRESENT PREGNANCY.

Her last menstrual period was on 16.04.2004 and her expected date of delivery 23.01.2005, giving her a maturity by dates of 22 weeks. She had not attended any antenatal clinic.

FAMILY AND SOCIAL HISTORY.

She was a housewife living in Kibera. The husband was a mason. She neither smoked cigarettes nor took alcohol. There was no history of chronic illness in the family.

GENERAL EXAMINATION.

She was sick looking with moderate pallor but no jaundice, cyanosis nor lymphadenopathy.

Vital signs

Respiratory rate 22 per minute

Pulse rate 112 beats per minute

Blood pressure 90/60 mmHg

Temperature 37.8 C

CENTRAL NERVOUS SYSTEM.

She was alert and conscious. Her neck was soft. Kerning" s sign was negative. Pupils were equally reacting to light and there were no lateralizing signs.

RESPIRATORY SYSTEM

Respiratory rate was 22 per minute, the hemithorax were equal and expanding on inspiration. The trachea was central. Percussion note was tympanic but there were scattered coarse crepitations especially on the right midzone.

ABDOMINAL EXAMINATION.

The lower abdomen was distended corresponding to a fundal height of 24 weeks. There was suprapubic tenderness but no guarding or rigidity. There was no hepatosplenomegaly or any other masses felt. The bowel sounds were normal. The fetal heart tones were not heard.

SPECULUM EXAMINATION.

She had normal external genitalia. There were blood clots in the introitus which were evacuated. The cervical os was parous open 2 cm. There was thin brown bloody discharge from the cervical os.

CARDIOVASCULAR SYSTEM.

She had a tachycardia of 112 beats per minute which was regular but reduced volume. It was non-collapsing. The blood pressure was 90/60mmHg, the neck veins were normal and the JVP was not raised. The first and second heart sounds were loud but there was no murmur. There were no thrills.

IMPRESSION

An initial impression of Inevitable Abortion with Intrauterine Fetal demise at 22 weeks gestation in a patient with Anaemia and Pneumonia was made.

INITIAL MANAGEMENT

The patient was counseled and informed that she had fetal demise and the plan was for delivery. Initial blood investigations were requested for and the patient was started on Dextran solution. A urethral catheter was inserted and clear urine drained. A pelvic/abdominal scan was requested for together with a chest X-ray. She was started on crystalline penicillin 2 mega unit 6 hourly, gentamicin 80mg 8 hourly and Flagyl 500mg 8 hourly. She was given Aspergic lg 8 hourly for pain and fever All drugs were given intravenously. An input-output chart was started The patient was then managed in the acute room in labor ward.

INITIAL INVESTIGATIONS RESULTS

PCV	31%
BUN	2.8 umol/L
Na+	143 mmol/L
K+	3.9 mmol/L
Creatinine	64 umol/L

Coagulation Profile

Prothrombin time test	20 seconds
Prothrombin time control	15 seconds
Prothrombin Index	75%

International Normalised Ratio (INR): 1.34

Activated Partial Thromboplastin Time (APPT) test- 42 seconds

APTT Control- 28 seconds

Comments: Slightly increased .APPT test but generally normal profile.

She was started on induction with 20 IU of syntocinon to run at 10 drops per minute to be gradually increased by 10 drops every half hour to a maximum of 60 drops per minute. Despite a gradual increase of syntocinon to 60 IU and the patient having uterine contractions there was no change in cervical effacement and dilatation and twenty four hours later since admission the patient had not expelled the fetus. The patient also received two units of blood because of increasing anemia.

An impression of Abdominal pregnancy was made and the patient was eventually taken for abdominopelvic scan together with the chest X-ray.

Pelvic/Abdominal Sonography Results

Scan showed a bulky non-gravid uterus with a length of approximately 12.6 cm. A fetus was seen in the abdominal cavity slightly to the right of the uterus. No cardiac activity was noted. Estimated gestation was not given.

Chest X ray- Report

Perihilar opacifications involving the right mid and lower lung zones. Features suggestive of pneumonia.

A confirmed diagnosis of abdominal pregnancy with Pneumonia was made.

SUBSEQUENT MANAGEMENT.

The mother was informed of the sonography results and the need for laparotomy. She gave an informed consent for emergency laparotomy. Blood was taken for grouping and cross-matching and four units of blood were requested. Premedication was given. Atropine 0.6mg stat before she was taken to theatre.

INTRAOPERATIVE FINDINGS.

On the operation table the patient was placed in supine position and general anaesthesia was given. The abdomen was prepared surgically and opened in layers through a midline subumbilical incision.

A fetus was found in the abdominal cavity on the right and posterior to the uterus surrounded by omental and placental tissues. The placental tissues were adjacent to the fetus and could not be delineated from the omentum. The umbilical stump was not identifiable There was approximately 500mls of blood in the peritoneum.

The fetus was gently separated from the omental tissues and delivered. It was macerated and weighed 1500 grams. No gross external anomalies were noted. The uterine tubes and ovaries were noted to be normal The abdomen was washed with warm saline and rifocin and surgicell left on the placental tissues. An abdominal drain was also left insitu. The patient received two units of blood intra-operatively and the blood loss was approximately 1600mls.She reversed well from general anaesthesia.

POSTOPERATIVE MANAGEMENT.

She was observed V* hourly until she was fully awake. She was started on strict input output charting, antibiotics and analgesics. She was then transferred back to the acute room in labor ward for continued management. On the first postoperative day, she was fully awake but moderately pale. The vital signs were within normal. Input out-put charting was adequate. She was started on oral sips and ambulation. She was also given two units of packed red blood cells. She was then transferred to the postnatal ward on the third postoperative day. Some investigations were done on the third post-operative day and patient was noted to have an elevated serum creatinine and BUN. Her input for the last 24 hours had been 3500mls against an out-put of 1750mls.

INVESTIGATION RESULTS

1. UREA ELECTROLYTES SERUM CREATININE

Na+	146 mmol/L.
K+	4.5 mmol/L.
Urea	20.9 mmol/L.
Creatinine	390.2 umol/L.

2 HAEMOGRAM

WBC	9.1 x 10/L
RBC	4.85 x 10/L
Hb	12.6 g/dl
Platelets	372 x 10/L
MCV	90 pg
HCT	43.7 g/dl

3 LIVER FUNCTION TESTS

Total protein	69 g/dl
Albumin	30g/dl

ALT 13 u/L

AST 9 u/L

ALP 210 u/L

Total Bilirubin 11.7 umol/L

Direct Bilirubin 2.0 umol/L

4. Urinalysis-Normal.

The renal physicians were consulted and she was put on Resonium A 1.5g per day low protein diet oral zantac 150mg once day and a strict input-output chart to be maintained. She was also to continue with intravenous antibiotics and daily urea and creatinine measurements. She did well on medication and by the 7th post-operative day the stitches were removed. Creatinine had come down to llOumol/ L and bun wasl2mmol/L. On the 12th post-operative day she was allowed home to be followed up in the renal unit and the gynecology out-patient clinic.

DISCUSSION

This was a 30 year old para 1+0 who had abdominal pregnancy and laparotomy was done. She also had anemia and pneumonia which complicated her clinical condition. Postoperatively she had renal failure which was managed successfully.

Almost all cases of abdominal pregnancy follow early rupture or abortion of a tubal pregnancy into the peritoneal cavity. Primary peritoneal implantation of the fertilized ovum is very rare. The incidence of abdominal pregnancy diagnosis depends on the presence of normal tubes and ovaries without evidence of trauma; in the absence of uteroplacental fistula and attachment of the conceptus exclusively to the peritoneal surfaces. Secondary abdominal pregnancies(more common) occur when the fetus escapes from the tube through a rupture or through the fimbriated end. In secondary abdominal pregnancy, the primary site of the gestation may have been tubal, ovarian or even uterine(3).

The incidence of abdominal pregnancy is increased after gamete intrafallopian transfer, invitro fertilization, endometriosis, tuberculosis and intrauterine devices (IUCD) may also contribute to an increased incidence(2).

The growing placenta typically penetrates the oviduct walls, maintains its tubal attachment but encroaches upon and implants on the neighboring structures while the fetus continues to grow in the peritoneal cavity. In other cases after tubal rupture, the conception re-implants elsewhere in the peritoneal cavity. In some cases a prior caesarean section scar will rupture early in pregnancy to give rise to a pregnancy under the vesico-uterine peritoneal fold. In the case presented the placenta was found to be attached to the gut and omentum.

The pregnancy usually develops normally if the implantation site provides sufficient blood supply to the placenta. Discomfort, genitourinary symptoms and actual pain are the rule as the pregnancy progresses. If undiagnosed and untreated, the fetus will die and suppurate, with abscess formation, form a true lithopedion or calcified fetus; develop into an adipocere;or result in undetermined retention of bony fetal parts with absorption of soft tissues. Massive intra-abdominal haemorrhage may ensue; fetal parts may extrude through the rectum, bladder or vagina or an abdominal fistula may form(3).

Fetal salvage is extremely poor in an abdominal pregnancy and the great majority succumb. A prenatal loss of 75% has been reported by some authors with incidences of malformed fetus ranging from 20-50%. Most of the fetal malformations are facial and or cranial asymmetry and joint abnormalities. Fetal lung hypoplasia is another abnormality resulting from oligohydramnios that will lead to fetal demise. Most common malformation are limb deformities and central nervous system malformation.

Because early rupture or abortion of tubal pregnancy is usual antecedent of an abdominal pregnancy, a suggestive history of spotting or irregular bleeding can be found in early pregnancy. High index of suspicion is required especially where there is a suggestive history of tubal rupture or abortion. Pregnancy complicated by unusual gastrointestinal symptoms such as nausea, vomiting, flatulence, constipation and diarrhoea may all be present in varying degrees. Fetal movements that are very marked or painful, easy palpation of fetal parts and movements, pregnancy described by multiparas as "different' or 'not right' and late in pregnancy;-lying fetus in abnormal presentation-often transverse. Abdominal massage over the pregnancy does not stimulate the mass to contract as it almost always does with advanced intrauterine pregnancies. On pelvic examination, the cervix is long, firm, displaced depending on the part of the fetal position and may dilate but appreciate cervical effacement is unusual hence failed induction should raise the index of suspicion of abdominal pregnancy.

Most laboratory values are normal as in intrauterine pregnancy unless tubal rupture or abortion occurs when a transient anaemia may result. An otherwise elevated level of alpha-fetoprotein in maternal serum suggests possibility of abdominal pregnancy. However amniotic fluid alpha-fetoprotein levels are usually normal.

Oxytocin stimulation tests are a valuable aid to diagnosis of abdominal pregnancy but some workers have found them to be unreliable and giving false positive results.

Sonographic diagnosis of an abdominal pregnancy may be hindered by bowel gas, adjacent soft tissues or lack of an acoustic window secondary to oligohydramnios^). Sonography is not a definitive diagnostic procedure for abdominal pregnancy since in

even ideal conditions, sonographic diagnosis of abdominal pregnancy is missed in half of the cases(8). In our patient, ultrasound was used to confirm abdominal pregnancy.

Magnetic resonance imaging (MRI) has been used for successful diagnosis and management of a viable abdominal pregnancy. The technique is able to confirm location of fetus, amniotic fluid, uterus and placenta before surgery and to follow placenta involution/dissolution after delivery(3). However Costa et al(8) maintain that computed tomography is superior to MRI but its use is limited due to fetal radiation, but computed tomography(CT)M Scan can be used in cases of fetal demise.

Treatment for abdominal ectopic pregnancy consists of immediate surgical removal of the fetus and membranes and ligation of the cord near the placenta. Some clinicians await fetal viability with in-hospital expectant management if pregnancy is diagnosed after 24 weeks(6). Such management carries a risk for sudden, life threatening intra-abdominal bleeding. Because of this risk, laparotomy is generally indicated when abdominal pregnancy is diagnosed. In cases where amniotic fluid is minimal or absent and in cases less than 24 weeks, conservative management is rarely justified because fetal survival is extremely poor.

The primary treatment of abdominal pregnancy is surgery. Before surgery it is usually good to have adequate blood supply available as surgery precipitates torrential bleeding. Preoperatively, two large intravenous infusion systems capable of delivering large volumes of fluid at a rapid rate should be functioning. The massive hemorrhage that often ensues with surgery for abdominal pregnancy is related to the lack of constriction of hypertrophied opened blood vessels after placental separation. Partial placental separation occasionally occurs spontaneously and mandates immediate laparotomy. Even if the fetus has been dead for several weeks bleeding may still be torrential.

Placental removal always carries risk of hemorrhage. Separation can occur either spontaneously or during the operation in an attempt to find the placental bed attachment, hence need to avoid unnecessary exploration of surrounding organs. In general, the infant should be delivered, the cord severed close to the placenta and the abdomen closed.

Unfortunately, ifthe placenta is left in the abdominal cavity, the placenta commonly causes infection, abscesses, adhesions, intestinal obstruction, wound dehiscence and even reversible maternal hydronephrosis (7,9).

Although the complication of leaving the placenta are troublesome and usually lead to subsequent laparotomy, they may be less grave than the hemorrhages that sometimes result from placental removal during initial surgery.

When the placenta is left, its involution may be monitored by ultrasound or B-hcg. In most cases placental function rapidly declines and the placenta is reabsorbed. The use of methotrexate is controversial. Its use has been recommended to hasten re-absorption but may cause accelerated placental destruction with accumulation of necrotic tissues and infection with abscess formation(2).

The maternal mortality is increased substantively compared with normal pregnancy with the rate being about 10%. About 50% of the fetus is alive at surgery but only about 20% survive(3).

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OBSTETRIC CASE No. 4

L NSENS1TIZED RHESUS-D NEGATIVE MOTHER-VAGINAL DELIVERY-LIVE BABY.

NAME	P.W	LMP: 29.10.2003
AGE	: 24	EDD: 05.08.2004
PARITY	: 0+0 G1	DOA: 18.07.2004
IP NO	: 0984296	DOD: 19.07.2004

PRESENTING COMPLAINT.

She was admitted in labor ward with labor pains for the last 6 hours. There was no history of drainage of liquor.

OBSTETRICS AND GYNAECOLOGY HISTORY.

She was a para 0+0 gravida I. Her last menstrual period was on 29.10.2003, and her expected day of delivery was 05.08.2004, fetal maturity by dates was 38 weeks. VIenarche was at 14 years. Her menses were regular lasting 3-4 days with a cycle of 28 days. She had used microgynon since 2000 to 2002 when she stopped since she desired to have a child.

ANTENATAL CARE

She attended a private antenatal clinic in South C in Nairobi at 6 months gestation. The antenatal profile was as follows:

Haemoglobin level	11.8g/dl
Blood Group	O Negative
VDRL	Negative
HIV Test	Negative
Indirect Coombs test	Negative
Husband's blood group	B Positive
Urinalysis	Normal

She had done an obstetric scan on 08.04.2004 which reported a single viable intrauterine pregnancy at 24 weeks gestation with adequate liquor. No fetal abnormalities were noted. She was seen in the same clinic on 06.05.2004 at 28 weeks gestation where a repeat indirect Coombs test was done and was found to be negative. She was given Anti-D 300ug. Her antenatal period was uneventful and at 36 weeks a repeat indirect Coombs test was negative and the patient continued with antenatal follow-up Her blood pressure at booking was 120/70mmhg and remained normal throughout pregnancy.

PAST MEDICAL HISTORY

There was no history ofblood transfusion nor previous admissions into hospital.

FAMILY AND SOCIAL HISTORY

She was married and employed as a secretary in a security company. Her husband was a businessman. There was no family history of chronic illness. She did not drink alcohol or smoke cigarettes.

GENERAL EXAMINATION.

She was in good general condition, no pallor , jaundice, edema cyanosis nor lymphadenopathy. Her blood pressure was 120/70mmhg, pulse rate 80 per minute, and temperature36.8 C.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moved with respiration. The fundal height was term, the lie longitudinal and cephalic presentation. The fetal heart tone were heard and regular at 146 beats per minute. The head was engaged with descent being 3/5 up. Contractions were palpable, 3 moderate contractions in lOminutes lasting 20 seconds each.

PELV IC EXAMINATION.

She had normal external genitalia. The cervical os was dilated at 6cm. there was no caput or moulding the membranes were bulging and the pelvis felt clinically adequate and no cord was felt.

The central nervous, cardiovascular and respiratory systems were found to be normal.

IMPRESSION.

An impression of a primigravida who is Rh- Negative in active phase of labor was made.

MANAGEMENT.

She was admitted to the labor ward and partograph started. ARM was done and clear liquor was obtained. She was reviewed 3 hours later and found to be 9 cm dilated and descent was 1/5 with no caput or moulding. She had strong contractions 4/ lOminutes lasting 40 seconds. The fetal heart rate was regular at 144 beats per minute. 4 hours since admission she was noted to have the urge to bear down and was transferred to the second stage room. She subsequently delivered by spontaneous vertex delivery to a live female infant weighing 3150 grams with an apgar score of 9/1 and 10/5.There were no any-abnormalities noted on the infant. Third stage was uneventful with the placenta delivered by controlled cord traction. Cord blood was taken for hemoglobin level, blood group and direct Coomb's test. The baby's blood was Rhesus positive, direct Coomb's test was negative. Haemoglobin level was 12.5g/dl. The mother was given Anti-D globulin 300ug on the 1st postnatal day and was discharged home to come again to the post natal clinic in six weeks time

FOLLOW-UP.

The patient turned up at the postnatal clinic as scheduled. She had no complaints. On examination she was normal and the bay was also normal. She was advised on family planning and referred to the Family Welfare Clinic.

DISCUSSION.

The patient presented was a primigravida who was unsensitized blood group O rhesus negative and delivered a rhesus positive son. Her husband's blood group was B rhesus **positive.**

In 1982 Ballantyne established the clinicopathological criteria for the diagnosis of hydrops fetalis. In 1932 Diamond and associates reported that fetal anemia that was characterized by numerous circulating erythroblasts was associated with this syndrome (1). In the red blood cells there are about 2500 recognized antigenic factors of which the most common are .ABO, Rhesus, Kell, Lutheran, Duffy, Kidd, P and MNS. In 1940 Landsteiner and Weiner discovered rhesus factor and in 1941 Lavine and associates confirmed that erythroblastosis was due to maternal isoimmunization. Finally Finn et al (1961) and Freda et al (1963) developed effective maternal prophylaxis(l). Since the development and implementation of antenatal Rh (D) immunoglobulin in 1960 by Finn and associates, there has been a marked reduction in Rh isoimmunization with its attendant complications (2).

Rhesus (CDE), blood group system antigens are inherited independent of other blood group antigens and are located on the short arm of chromosome 1(1). There is no difference in terms of sex distribution but there is a significant difference in terms of racial distribution. The incidence of rhesus (D) negative antigen is highest among the Basque population 30-50% followed by Caucasians 15% and nil among the mongoloids. The Afro-Americans have an incidence of 8% while African blacks have an incidence of 4%(3). Mulandi found an incidence of 4% among antenatal mothers in KNH (3). Whereas muroki found an incidence of 2.6% among patients admitted with abortion (4).

Individuals with rhesus factor are called rhesus positive and the others rhesus negative. The Rhesus blood group has five red blood antigens namely c C, D, e and E. No d antigen has been identified. The genes are transmitted in groups of three. The gene that makes a person Rh- Positive is D. It is inherited in a mendelian dominant manner. If it is present in either pair of chromosomes, the individual is Rh- positive. D negative is the absence of D' in either pair of chromosomes. A rhesus negative mother carrying a rhesus positive fetus may have fetal cells crossing into maternal circulation in different amounts. This causes maternal antibodies against rhesus factors to develop and cross back into

fetal circulation. This results in hemolysis of fetal blood cells and causes hemolytic disease of the newborn(1,3). Forty five percent of rhesus positive persons are homozygous for D and 55% are heterozygous(2). If the father is heterozygous half of his children are positive. The patient was rhesus negative while the husband was rhesus positive.

The Cc and e antigens have a lower immunogenicity than the D antigen that is responsible for severe hemolytic disease of the newborn. Fetal cells do enter the maternal circulation but in small amounts, which are destroyed by the maternal immune system before provoking an antibody reaction especially where the ABO blood group of the mother and fetus are incompatible. Isoimmunization may occur following incompatible blood transfusion or following feto-maternal hemorrhage between a mother and incompatible fetus. There are a number of predispositions to fetal maternal hemorrhage including spontaneous or induced abortion, amniocentesis, abdominal trauma, placenta praevia, abruptio -placenta, multiple gestation, fetal death in utero, manual removal of the placenta and caesarean section(2). The patient did not have any of these risk factors.

If a woman who is Rh (D) negative delivers an infant who is Rh (D) positive and ABO compatible she has a likelihood of 16% of isoimmunisation (5). Of these women 1.5-2% of the reactions occur antepartum, 7% within 6 months of delivery and the remainder early in the second pregnancy (2). The main cause of maternal sensitization is Rh (D) incompatible fetus. Feto-maternal hemorrhage may occur during pregnancy or at delivery. Even where there is no apparent risk factor fetal red cells can be detected in maternal blood in 6.7% of women during first trimester, 15.9% in the second trimester and 28.9% in the third trimester (2). The result of this antepartum feto-maternal hemorrhage is an overall rate of Rh sensitization of about 1 to 2 percent before delivery. However, antepartum sensitization rarely occurs before the third trimester (2,6).

Antigens on the fetal red cells are detectable as early as 30 days after conception hence the need for anti-D globulin even after an abortion. The number of red blood cells required to cause isoimmunization is uncertain however as little as 0.1ml of Rh (D) can cause sensitization (5). However the amount varies from patient to patient. This is probably due to the difference in the actual numbers of Rh positive RBCS, immunogenic capacity of the Rh-positive erythrocytes and the immune responsiveness of the mother.

Two characteristics affect whether alloimmunization will occur in susceptible Rh-negative women. First 30% of Rh-negative individuals appear to be immunologic "non responders" who will not become sensitized, even when challenged with large volumes of Rh-positive blood. Secondly incompatibility of ABO exerts a protective effect against the development of Rh sensitization. For ABO incompatible pregnancy this 16% risk is reduced to 1.5-2% (1,7). This is more pronounced when the mother is type O and the father is type A, type B, or type AB (3,6). In our patient the mother was type 0 and the father type B. thus this couples blood group incompatibility offers protection of their children against alloimmunization to about 1.5-2%.

The initial response of a rhesus negative individual to rhesus positive cells is formation of IgM. Subsequently IgG antibodies are formed and this crossES from mother to fetus and cause hemolysis, hydrops fetalis and kernicterus depending on extent of hemolysis. The initial isoimmunization reaction is minimal but subsequent reactions tend to be stronger and more severely affecting (1,3,4,8).

Early in the 1960s it was discovered that administration of anti-D immunoglobulin to Rh-negative individuals who had been infused with Rh-positive red cells prevented development alloimmunization. This was based on immunological principle called antibody-mediated immune suppression (AMIS) where passively administered antibody will prevent active immunization by its specific antigen. The widespread use of anti-D immunoglobulin to prevent alloimmunization has made the frequency of sensitized pregnancy among Rh-negative women to decrease. During early trials it was established that 300ug of Rh -immune globulin would reliably prevent alloimmunization in individuals who had received lOmls of Rh-positive cells and from this, it was established that lOug of Rh- immune globulin should be given for every 1 ml of fetal blood in the maternal circulation. Subsequently it has been established that 100 to 150ug of Rh-immune -globulin is sufficient for routine use. However, in the United States of America, the standard dose for Rh prophylaxis is 300ug within 72 hours of delivery. The 72 hours has been because early studies were on male prisoners and prison officials would only allow investigators to visit the volunteers at 3 -day intervals. Thus the use of Rh-immune globulin at intervals of more that 3 days after a challenge was never extensively evaluated.

In our patient the baby's blood group was found to be Rh-positive and the mother was given anti-D immunoglobulin 300ug on the 1st postnatal day. If the infant was Rh-negative no anti-D immunoglobulin would have been administered (1,3,5,6).

The management of isoimmunized patients aims at minimizing fetal and neonatal morbidity and mortality. Accurate assignment of gestational age using the menstrual dates and early ultrasound is crucial in management of Rh-alloimmunized pregnancy. The timing of amniocentesis, umbilical cord blood sampling, in utero treatment and delivery will depend on the gestational age. Management is usually individualized. Investigations for rhesus factor, ABO blood group and ruling out sensitization by indirect Coomb's test is mandatory.

If the woman is sensitized she will not benefit from anti globulin. If the woman is rhesus negative, testing for paternal ABO and rhesus blood groups may be useful (2). Unsensitized patient should be followed up antenatally by Indirect Coomb's test initially at first visit, then between 28-32 weeks, then 4 weekly interval to term and immediately post delivery (2). In the first contact with the patient the maternal antibody titre is quantified. The critical antibody titre level below which the fetus will not die from hemolytic disease is usually 1:16. Maternal ICT is monitored on a fortnight basis; titres between 8-32 are acceptable, however if they exceed 32 there is an associated risk of severe fetal compromise even death and amniocentesis with use of the OD 450 curve is indicated (9).

The amount of bilirubin in the amniotic fluid correlates with the amount of red blood cell destruction and hence fetal anaemia. Amniocentesis is performed when the critical titre is reached or if there has been a previous seriously affected fetus or infant. Bilirubin present in amniotic fluid is derived from fetal pulmonary and tracheal effluents and correlates with the degree of fetal hemolysis. The analysis of the change in optical density of amniotic fluid at 450nm curve on the spectral absorption curve (delta OD450) is used to determine amniotic fluid bilirubin, thereby helping to evaluate the degree of fetal anaemia. Data are plotted on a normative curve based upon gestational age. The original curve developed by Liley in 1961 was divided into three zones (10). Before 28 weeks of gestation the Liley,s curve is a less sensitive measure to determine need for intervention rather the Queenan curve is used (11).

The unaffected or mildly affected fetus fall into Liley's zone 1 and amniocentesis should be repeated every 2-3 weeks and delivery shouldn't be near term when the fetus has achieved pulmonary maturity. Moderately affected fetus fall into zone 2 and amniocentesis is repeated every 1 -2 weeks. Delivery may be required prior to term and enhancement of lung maturity can be achieved by use of steroids.

The severely affected fetus with a hemoglobin level less than 8.0g/dl may die in 7-10 days without intervention and falls into zone 3. Amniocentesis is done weekly and ultrasound is used to search for ascites or edema. Generally hemolysis and hydrops develop at about the same time or somewhat earlier in subsequent pregnancies. Doppler scan for the middle cerebral artery flow and umbilical artery (cordocentensis) hemoglobin* Severely affected fetus may require intra-uterine blood transfusion and when the fetus has sufficient pulmonary maturity for survival delivery can take place (1,3,8). A 30% drop in cord blood hemoglobin level of the expected for gestational age requires intervention either by transfusion in utero or delivery if the fetus is viable (12), however this is associated with some risk of fetal wastage and hence amniocentesis is preferred.

Intrauterine fetal transfusion is performed using " 0" negative , cytomegalovirus-negative. irradiated, glycerolized or irrigated packed cells (2).

Postpartum rhesus D immunoglobulin prophylaxis is a well established practice of proven benefit (1,3). The following are currently recommended for rhesus prophylaxis.

Every rhesus negative immunized woman must be given one prophylactic dose of rhesus immunoglobulin as soon as possible after delivery if the infant is rhesus positive. Administration should not be delayed beyond 72 hours.

If the pregnancy loss occurs at 12 weeks gestation or less, a 50ug dose of Rh-immune globulin is adequate to cover, the entire fetal blood volume. If the gestation is unknown or beyond 12 weeks, a full 300ug dose of Rh-immune globulin is indicated(1.36,13).

Rhesus negative non-immunized woman undergoing amniocentesis should be given one prophylaxis dose of immunoglobulin (300ug) unless the father is known to be rhesus negative(3,5).

Rhesus negative non-immunized woman should receive one prophylactic dose of immunoglobulin (300ug) at 28 weeks unless the father is rhesus negative(3,5) and should be repeated in another 12 weeks.

Rhesus negative non-immunized woman who has fetal transplancental hemorrhage exceeding 25mls of fetal blood should be given at least 300ug of rhesus immunoglobulin for every 25mls of fetal blood in her circulation unless the father is rhesus negative (5).

Some schools of thought recommend a repeat dose of anti-D at 34 weeks and after delivery while others recommend that a repeat dose only be given of the pregnancy exceeds 40 weeks of gestation at which time the antibody levels have started to wane (5). However at 40 weeks delivery is indicated. Anti-D globulin should be administered within the first 72 hours of delivery, however if this is not possible it has been shown to offer partial protection even when given up to 13 days post partum. Some authors recommend that it be given up to 28 days postpartum (2). Women who deliver within 2-3 weeks of antenatal anti -D globulin injection need not repeat the injection after delivery (14).

Other methods for infant treatment other than exchange transfusion include, plasmapharesis, large doses of promethazine, D-positive erythrocyte membrane in enteric coated capsules and immunosuppression with corticosteroids (1,6).

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OBSTETRIC CASE No. 5

GESTATIONAL DIABETES-VAGINAL DELIVERY-LIVE BABY.

NAME:	F A	DOA	30.08.2004
AGE:	28 YEARS	DOD	02.11.2004
IP NO:	0895484	LMP:	25.01.2004
PARITY:	3+0 Gravida 4	EDD:	01.11.2004
		MBD:	32 Weeks.

HISTORY OF PRESENTING COMPLAINTS.

F A was admitted via labor ward as a referral from Mathare North City Council Clinic with a one month history of polydipsia, polyuria and polyphagia. She also had a one week history of headache, joint pains, general malaise, dysuria , fever, frequency of micturition and lower abdominal pain. She had had one episode of vomiting prior to admission. A random blood sugar done at the clinic was 11,3mmol/L with a glucosuria of 3+.

OBSTETRICS AND GYNAECOLOGY HISTORY

She was a para 3+0 gravida 4.Her first delivery was in 1996, SVD in Pumwani Maternity Hospital, live male baby at term who had a birth weight of4000gm. The second delivery was also in the same hospital in 1999, SVD, at term, live male infant whose birth weight was 3900gm. The third delivery was in KNH in 2002 at 36 weeks, SVD, live male infant whose birth weight was 2850gm and had an Apgar score of 8 in 1 minute and 10 in 10 minutes. This baby passed away at two months due to pneumonia. In all the pregnancies the antenatal period was uneventful but her blood sugars were not done and she is not a known diabetic. Her menarche was at 15 years and she had regular periods every 28 days lasting 3-4 days. She had used IUCD from 1996 to 1998 and had it removed because of chronic back pain and recurrent per vaginal discharge. She then used oral contraceptive pills from 1999 to 2002 when she stopped because she desired to have another child.

HISTORY OF CURRENT PREGNANCY

Her last menstrual period was on 25.01.2004 with an expected date of delivery of 01.11.2004 giving her a maturation by dates of 32 weeks at the time of admission. She attended antenatal clinic at Mathare North City Council Clinic at 24 weeks gestation and had gone for two visits and was referred to KNH on this third visit. Antenatal profile had been done and the results were as follows -

Hb	12.6 g/dl
Blood Group	**B** Positive
VDRL	Negative
HIV	Negative

Prior to her referral to KNH, her antenatal period had been uneventful.

PAST MEDICAL HISTORY.

This was non-contributory

FAMILY AND SOCIAL HISTORY

She was a married tailor living in Mathare North. Her husband was a businessman. She did not smoke cigarettes nor drink alcohol. There is no history of diabetes in the family or any other chronic illness.

GENERAL EXAMIN ATION.

She was in fair general condition clinically febrile with mild dehydration. She was not pale, jaundiced or cyanosed. She had no edema or lymphadenopathy.

VITAL SIGNS

Temperature	38.1 C
Blood pressure	110/70 mmHg.
Pulse rate	98 beats per minute
Respiratory rate	20 per minute

ABDOMINAL EXAMINATION.

The abdomen was uniformly distended with a fundal height corresponding to 30 weeks gestation. The fetus was in longitudinal lie and cephalic presentation. The fetal heart tones were heard at a rate of 140 beats per minute. There was no evidence of hydramnios. There was mild suprapubic tenderness. There was no any other organomegaly or any other masses.

Respiratory, Cardiovascular and Central Nervous systems were essentially normal.

DLAGNOSIS

Gestational Diabetes Mellitus with Urinary Tract Infection at 30 weeks gestation.

MANAGEMENT

She was admitted in labor ward and started on intravenous fluids, (Hartman's solution) and intravenous Augmentin 1.2g 8 hourly and diclofenac 75mg 8 hourly for pain and fever. She was also started on subcutaneous insulin 10 IU to be given 8 hourly and to be titrated against blood sugar levels. She was started on diabetic diet. Blood and urine were taken for investigations and the results were as follows.

Blood slide for malaria parasites:	Negative
Random blood sugar:	12.3 mmol/L (Before 1st dose of insulin).
WBC	11.4 x 10/L
Hb	10.8 g/dl
Urinalysis	Sugar 3+, epithelial cells +, trace rbc's, ketonurial
Renal Function Tests:	BUN-3.0mmol/L, Creatinine-86umol/L.
	Na+- 146 mmol/L, K+- 4.5mmol/L.

Serial blood sugar levels to monitor control were requested for and a chart maintained. After observations and treatment in labor ward for 24 hours she was stable and was transferred to the antenatal ward to continue with treatment and other investigations.

SUMMARY OF INVESTIGATIONS

Fasting blood sugar- 11.8 mmol /L

OGTT	Blood Sugar	Glycosuria	Ketonuria
hour	12.4	++	+
1 hour	11.2	+++	++
2 hour	10.8	++	+

HAEMOGRAM

Wbc	8.6 x 10 /L	RBC	5.66 x 10 *fL*
Differentials	Neutrophils - 60 %	Platelets	134x1O/L
	Lymphocytes- 30%	Hb.	11.6 g/dl.
	Basophils- 1%		
	Monocytes- 0%		
	Eosinophils- 1%		

Urinalysis Culture and Sensitivity: Moderate growth of Group B b-hemolytic streptococcus sensitive to Augmentin +++.

Hb A 1C 10.6% (N-4 5-7.0)

Obstetric Scan: Live viable intrauterine pregnancy at 30 weeks 3 days by FL and BPD No fetal anomalies seen. Adequate amniotic fluid volume. Biophysical profile of 8/8 and Resistive Index of 0.630.

PROGRESS IN THE WARD.

Within a week the urinary tract infection resolved and the antibiotic was stopped. However serial blood sugars were not well controlled and she had stepwise increment of soluble insulin up to 24 IU 8 hourly by the third week. Fasting blood sugar ranged

between 6.0 mmol/L to ll.Ommol/L. On 15.10.2004 at 38 weeks maturation by dates she went into spontaneous labor and was taken to labor ward. She progressed well and delivered SVD to a live female infant whose birth weight was 3400gm with Apgar score of 8 in 1 minute 10 at 5 minutes and 10 in 10 minutes. The baby had no anomalies and was taken to the newborn unit for observations. After 48 hours the baby was discharged from the newborn unit to join the mother. The mother's blood sugar levels were not well controlled postpartum and a diabetologist was consulted and converted her to Lente insulin. She was discharged on the 14^{th} postnatal day on lente insulin to be seen in the diabetic clinic in two weeks. Unfortunately she was lost to follow-up

DISCUSSION

The patient presented was a para 3+0 who developed gestational diabetes at 30 weeks gestation by dates. She was managed on soluble insulin though adequate control was not achieved but she had a good fetal outcome.

Diabetes mellitus is a heterogeneous disorder characterized by hyperglycemia, which is a result of relative or absolute insulin deficiency. Insulin plays a critical role in carbohydrate, fat and protein metabolism. With insulin deficiency or functional impairment, blood glucose levels are elevated as a result of both decreased utilization of glucose by skeletal muscle, hepatic and adipose tissues and increased hepatic glycogenolysis and gluconeogenesis (1).

Glycosylated haemoglobin is increased when blood glucose levels are elevated over a period of time. Impaired utilization of aminoacids by muscles contribute to gluconeogenesis. Impaired lipolysis occurs with insulin deficiency, causing elevation of free fatty acids and an increased formation of ketone bodies (acetoacetate and beta-hydroxybutyrate) (I). The incidence of diabetes in pregnancy is about 1%, making it the most frequent metabolic disorder complicating pregnancy (1). The incidence of diabetes in pregnancy was found to be 1 in 343 in **KNH.** (2).

The diagnosis of diabetes in pregnancy depends on history, physical examination and laboratory investigations. Polydipsia and polyuria occur when blood glucose levels significantly exceed the renal resorption constant for glucose. An associated osmotic diuresis with dehydration and electrolyte loss may occur (1). Some workers have outlined the most commonly used criteria for the suspicion of diabetes mellitus. A history of one or more of the following features should sound a warning (3).

1. Bad obstetric history (BOH): Previous unexplained still births or neonatal deaths or habitual abortions or previous congenital malformation of the newborn.
2. A strong family history of diabetes, at least 1st degree relative or two 2nd degree relatives or a previous child with a birth weight of 4Kg or more
3. Abnormal screening tests (Abnormal glucose tolerance tests-GTT or glucosuria outside pregnancy).
4. Obesity which is progressive (90 Kg or more).
5. Unexplained polyhydramnios.
6. Unexplained hypertension or pre-eclampsia.
7. History of repeated infections, especially vulvovaginitis.

The diagnosis is confirmed by means of a glucose tolerance test. The patient underwent OGTT whose results confirmed gestational diabetes mellitus. In this test, the patient is instructed to have a normal diet for 3 days prior to the test, then she fasts overnight for at least 10 hours before the test. She should not smoke or have diuretic therapy before or during the test.

A carbohydrate load of 75 gm glucose in 20-350 mis of water is used. Blood sampling every hour for 3 hours is done. A diagnosis of diabetes is made if fasting blood glucose is 8mmol/L or the 2 hour value is > 1 lmmol/L. a diagnosis of impaired glucose tolerance is made if the fasting plasma glucose is less than 8 mmol/L and the 2 hour glucose is more than 8 mmol/L and less than 11 mmol/L. (3).

The Diabetes Data Group of the National Institute of Health proposed a new classification system based on the aetiological factors and insulin dependence as follows (4):

a. Type I - Insulin-dependent diabetes mellitus.
b Type II - Non-Insulin dependent diabetes mellitus
c. Type III - Gestational diabetes or carbohydrate intolerance.
d. Type IV - Secondary diabetes.

Another classification proposed by White almost 40 years ago is still generally accepted and remains a useful prognostic guide (5). Whites classification relates the onset of diabetes, its duration and the degree of vasculopathy to the outcome of pregnancy.

A revision made by Hare and White proposed class-A diabetes, to include women known to have diabetes before pregnancy and who are treated with diet only (5).

Practically speaking, women with pregnancies complicated by diabetes mellitus may be separated into one of two groups:

A. Gestational diabetes- Women with carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy.

B Pre-gestational diabetes- Women known to have diabetes before pregnancy.

Gestational diabetes is further classified as

A-1- With fasting glucose of< 5.8mmol/L- Post-prandial blood glucose >6.7 mmol/L

A-2-With fasting glucose > 5.8mmol/L and post-prandial glucose level > 6 7mmol/L

Whites' Classification of pre-gestational diabetes is as follows.

1 Class A- **With** any age of onset, any duration, no vascular disease and controlled on diet alone

2. Class B - With age of onset > 20 years, duration< 10 years, no vascular disease and therapy with insulin.

3. Class C - With age of onset 10-19 years, duration 10-19 years, no vascular disease and on insulin therapy.

4. Class D- With age of onset before 10 years or after 20 years, duration of > 20 years, with benign retinopathy and on insulin therapy.

5. Class F - With any age of onset, any duration, with nephropathy and on insulin therapy.

j. Class R- With any age of onset, any duration, with proliferative retinopathy and on insulin therapy.

I Class H - With any age of onset, any duration, with heart disease and on insulin therapy.

n general the more ,severe the degree of vasculopathy in pregnancy, the worse the fetal prognosis. 90% of all pregnant diabetic patients have gestational diabetes (GDM), whereas insulin dependent diabetes mellitus (IDDM) and non insulin dependent diabetes mellitus (NIDDM) account for the remaining 10% (5).

Type I diabetes is immune-mediated, and develop in genetically susceptible persons, its associated with HLA-D complex located on chromosome 6 (6). Type II diabetes has no HLA association, though it has a familial occurrence. It is caused by abnormal insulin secretion and insulin resistance in target tissues. (6).

In normal pregnancy, basal insulin levels are almost unchanged in the first and second trimester but increase as much as 50% in the third trimester which suggests insulin resistance at the time.(5,6).

The increase in insulin release in response to a glucose load becomes pronounced by the third trimester During the first trimester, insulin action is enhanced by estrogen and progesterone leading to an increased glucose use and lower fasting glucose levels, resulting in increased episodes of hypoglycaemia, in pregnant diabetics in early pregnancy.(1,5,6).

Human placenta contains insulin-degrading enzymes, insulinase, but this has not been shown to increase insulin clearance from the placenta. Insulin resistance in pregnancy has been demonstrated and is progressive as gestation advances. Insulin sensitivity is decreased in pregnancy by as much as 80% from non-pregnant state. (5).

Pregnancy has a diabetogenic effect on the mothers as demonstrated by glucose intolerance during pregnancy.

Elevated insulin concentrations during human pregnancy can be attributed to a variety of hormonal changes, like rising levels of maternal progesterone, human placental lactogen, free Cortisol and prolactin.

During normal pregnancy, the fasting blood glucose level decreases reaching a nadir by the 12th weeks of gestation and remaining unchanged thereafter till delivery.

Diabetes may be deleterious to pregnancy in a number of ways. A number of complications may arise. Both maternal and prenatal morbidity and mortality are increased compared to normal pregnant women. The likelihood of preeclampsia-eclampsia is increased fourfold even in absence of pre-existing vascular or renal disease. Some bacterial infections are more common in diabetic pregnancy. The fetus can be macrosomic with resultant difficult delivery, birth trauma and maternal injuries (6). The rate of caesarean delivery is increased with increased maternal risks of surgery. Hydramnios is common. Maternal diabetes adversely affects the fetus. The incidence of spontaneous abortions may be high in poorly controlled diabetics, probably due to higher malformation rates. Prenatal death is increased considerably. Major anomalies are increased at least 3 fold in fetuses of women with overt diabetes. The incidence of preterm delivery is increased 2-3 fold. Neonatal morbidity is common from birth trauma, RDS, hypoglycaemia and hypocalcaemia. The infant may inherit a predisposition to diabetes. Metabolic disturbances in diabetic patients are expressed in increased concentrations of circulating metabolic fuels including carbohydrate, protein and fat, these can be transferred to the fetus and contribute to the development of fetal macrosomia.

Gestational diabetes is defined as carbohydrate intolerance of variable severity with onset or first recognition during present pregnancy. The incidence of gestational diabetes mellitus varies and is estimated at 3-5% of pregnant women (5). In the past patient selection for an oral lOOg GTT was based on historic and clinical risk factors such as obesity, glycosuria, previous macrosomic infant, previous neonatal death or congenital malformation, family history of diabetes and hypertension during current pregnancy. By these selections only 63% of patients with GDM were identified (5). There is no international agreement as to the appropriate and globally acceptable diagnostic criteria for gestational diabetes. The second International Workshop-Conference on GDM recommended that all pregnant women receive screening for glucose intolerance at 24-28 weeks with 50g of oral glucose. A value of plasma glucose of >7.8mmol/L, indicates the need for a full

diagnostic GTT. Diagnosis of GDM is based on the results of the lOOg oral GTT. This method of screening has a sensitivity of 79% and specificity of 87% (8). SQ (*f «« • Vtffeft -a birth tTj.im. ffti»i,

The American College of Obstetrics and Gynecology recommend screening all pregnant women older than 30 years of age as well as women with any risk factor (6,9). Most authorities recommend testing at 24-28 weeks gestation, when insulin resistance is increased. Some investigators recommend screening for GDM at the first prenatal visit for all patients with risk factors, with repeat of a negative test at 24-28 weeks.

Other workers have recommended screening at 20, 28 and 34 weeks (11). Glycosylated haemoglobin and fructosamine levels are of limited value in screening for GDM, as they have low sensitivity and specificity (5). Screening has improved pregnancy outcomes in GDM patients. Close surveillance of the mother and fetus with close monitoring of maternal glucose levels is important in reducing morbidity and mortality.

After diagnosis of GDM, patients receive nutritional counseling, which is the mainstay of therapy in these patients. Blood glucose is monitored once or twice weekly. If fasting blood glucose levels are at least 5.8mmol/L or 2-hour post-prandial levels of at least 6.7mmol/L insulin therapy is begun (5).

Glycaemic control may be assessed with glycosylated hemoglobin periodically. Some workers have recommended prophylactic insulin therapy, even in those patients with GDM and are seemingly well controlled on dietary therapy. It has been shown to further reduce neonatal morbidity(12).

Antenatal testing with non -stress test weekly, maternal surveillance of fetal movements from 32 weeks gestation have been advised in some patients with GDM, especially if they are on insulin, have chronic hypertension or pre-eclampsia or have had a previous still birth.

Delivery may be delayed until spontaneous labor or 42 weeks in well controlled patients, but induction of labor as soon as pulmonary maturity is confirmed is recommended in poorly controlled patients (5).

Fetal weight estimation is prudent before attempt at vaginal delivery and in unfavorable cervix, cervical ripening is indicated. If estimated weight is > 4.5kg, caesarean delivery is recommended to prevent shoulder dystocia and birth trauma. If the weight is 4-4.5Kg, individualized management based on the size of the patient and patients' previous obstetric history is necessary.

Patients with GDM are at risk of developing diabetes, years after pregnancy. It is recommended that women with GDM be followed up postpartum to detect diabetes early. The risk of developing diabetes later in life in GDM is greatly influenced by body weight, with highest rates in obese patients. Obese patients should be advised to control their weight.

Management of the pregnant diabetic woman is a complex task that should start before conception. In the pre-pregnancy period, the patient and her partner are educated on diabetes care during pregnancy and the need for stringent glycaemic control. The patients' general medical status is assessed and signs of retinopathy, nephropathy, hypertension, and ischaemic heart disease looked for. Opthalmological evaluation, ECG and renal function tests are performed. Severe retinopathy is treated with laser coagulation before pregnancy. In-patients with coronary artery disease, termination of pregnancy is seriously considered Patients on oral hypoglycaemic drugs should discontinue them and begin insulin treatment. In our set-up, many patients present for booking when already pregnant and there are no effective pre-conception sessions.

The goals of pre-pregnancy care are to achieve optimum diabetic control even before conception, as a high incidence of congenital anomalies is related to hyperglycaemia in early pregnancy.

Congenital malformations in fetuses of diabetic patients are now responsible for about 40% of all prenatal deaths, replacing respiratory distress (RDS) as the leading cause of infant death (5). Cardiac anomalies are the most frequent malformations followed by CNS malformations (neural tube defects) and skeletal malformations.

Diabetes associated embropathy in humans is thought to be caused by hyperglycaemia, ketone bodies, hypoglycaemia, low levels of trace metals and somatomedin-

inhibiting factors, during organogenesis. Insulin has not been conclusively incriminated in causation of diabetic embropathy.

All patients with pre-gestational diabetes mellitus are evaluated for possible fetal anomalies. Patients are routinely evaluated by maternal serum alpha-fetoprotein (MSAFP), an early glycosylated hemoglobin (HbA1C) estimation and ultrasonography.HbA1C level > 9.5% is associated with significantly higher malformation rate. Ultrasound should be done at about 20 weeks gestation (5).

Over the last 2 decades, prenatal mortality and morbidity has decreased significantly from 14-35% to 3-5%. This has been after stringent glucose control programs have resulted in better fetal outcome, and advances in insulin delivery and monitoring of glucose levels. Insulin doses must be individualized and balanced with diet and exercises.

Diet therapy is considered a standard treatment of diabetes mellitus. All patients are seen by the dietician and individual meal plan adjustments made. The FDA recommends 35Kcal per Kg of ideal body weight and a diet composed of 20% protein, 30% fat and over 50% carbohydrates (5).

Restricted saturated fats and cholesterol and increased dietary fiber are suggested. A diet consisting of 3 meals and 1-3 snacks, with the last at bedtime is recommended. Patients' weight gains are assessed at each clinic visit (total weight gain of 10-13 Kg through the pregnancy is ideal). Intensive insulin therapy should begin before conception or as soon as possible thereafter. In the past, liberal hospitalization of diabetic patients was employed early in pregnancy, with routine readmission in the 3rd trimester. But no significant difference has been found in maternal blood glucose control, fetal hyperinsulinaemia, perinatal mortality and morbidity between outpatient approach and long term hospitalization.^).

Currently hospitalization of diabetic patients is an exception rather than the rule. Most of the patients are seen as outpatient at 1-2 weeks intervals. Hospitalization is currently reserved for poorly controlled or non-compliant diabetics or in the third trimester with infection induced hyperglycaemia, worsening diabetic nephropathy or frank preeclampsia.

Ophthalmologic and renal function tests (creatinine clearance, total urinary protein excretion) are performed in each trimester or more often if indicated. ECG is done at booking and repeated as necessary in those with vasculopathy. Assessment of blood pressure and proteinuria is done to detect early sign of pre-eclampsia. All pregnancies complicated by diabetes require extra assessment. At 1^{bl} trimester scan is used to date the pregnancy, establish viability and determine fluid volume status. A 2nd trimester scan is repeated at 18-20 weeks to rule out fetal anomalies. Subsequent scans are then performed at 4-6 weeks intervals to assess fluid volume and fetal growth.

In pregnant diabetics stillbirth occurs with increased frequency, particularly in the third trimester and a program of fetal monitoring should be initiated at 32-33 weeks.

Currently outpatient protocols for antepartum fetal surveillance include, 1-2 times/week NST, once weekly oxytocin challenge test or biophysical profiles. NST is the most widely used test for pregnancies complicated by diabetes mellitus.

Vlaternal assessment of fetal activity also seems to be a practical approach toward evaluation of fetal condition, decreased fetal movements less than 10 in 12 hours requires further testing. Counting of fetal movements in diabetics may begin as early as 28-29 weeks gestation. Doppler ultrasound may also be used to assess vascular resistance on umbilical vessels.

In the past, a policy of early delivery in pregnancies complicated by diabetes was almost the rule. Many authorities recommend delivery at 36-37 weeks. This resulted in very high caesarean section rates and more infants with RDS.

Its now widely recognized that if the pregnant diabetic patient and her infant are under stringent metabolic control and antepartum surveillance, delivery may be safely delayed in most cases until term or the onset of spontaneous labor (5, 13) This new approach has increased the incidence of spontaneous labor, resulted in a decrease in caesarean section rate and reduction of RDS.

Selection of the time of delivery is individualized considering the degree of glycaemic control, maternal complications and fetal well being. Some diabetics are selectively delivered at 38 weeks after fetal lung maturity has been confirmed.

These include patients in poor metabolic control, worsening pre-eclampsia, with suspected fetal macrosomia, growth retardation or polyhydramnios.

In certain rare cases, pre-term delivery may be necessary despite immaturity of fetal lungs. These include severe pre-eclampsia unresponsive to therapy or signs of severe fetal compromise. Other relative indications of delivery include worsening diabetic nephropathy leading to renal failure, worsening retinopathy not responding to laser therapy.

Whether induction or elective caesarean section is done , depends on favourability of cervix, and the estimated fetal size. Induction is not done if estimated fetal weight is 4 Kg or more, instead elective caesarean section is preferred to prevent traumatic vaginal delivery. If the cervix is unfavorable at term, it is ripened before induction with prostaglandin's, laminaria or intracervical balloon.

During labor and delivery, it is necessary to maintain maternal euglycaemia to avoid neonatal hypoglycaemia. A decrease in insulin requirement has been documented particularly in the first stage of labor. Therefore in patients undergoing induction of labor, the morning Insulin dose should be withheld and glucose levels determined once every hour In well controlled patients one unit of insulin per hour and 3-6gm of glucose per hour are usually required to maintain a glucose level of 3.8-5. Ommol/L. If the patient presents in spontaneous labor and had her morning insulin, additional insulin may not be necessary throughout labor and delivery, but a continuous glucose infusion will be necessary (125mis /hr of 5% dextrose).

When an elective caesarean section is planned in a diabetic patient, it should be scheduled early in the morning when the sugar levels are usually in the normal range. Infusion without glucose is preferred and glucose levels are monitored frequently.

After delivery, a dramatic decrease in insulin requirement arises and there is no need for stringent glucose control and levels < I lmmol/L are satisfactory In the first few days after delivery, it is preferable to give regular insulin subcutaneous before each meal on the basis of plasma glucose levels. After the patient is able to eat

regular meals, she may get one-half of the pre-pregnancy dosage of insulin in 2 daily injections.

Breastfeeding should not be discouraged, but the mother is advised to increase her caloric intake just before nursing because insulin requirements are lower after breast feeding and may result in hypoglycemia. Causes of maternal deaths have shifted from diabetic ketoacidosis to cardio-renal complications (Diabetic Retinopathy and Nephropathy)

Estrogen containing oral contraceptives and intrauterine device are best avoided if possible in women with overt diabetes. Combined oral contraceptives are likely to worsen diabetes or its vascular complications. The progestin implant (Jadelle) has minimal effect on carbohydrate metabolism and may be the ideal contraception in diabetic women. Progestin only oral contraceptives also may be utilized. Barrier methods are also excellent choices for reversible contraception, followed by sterilization once the woman wants no more children(6).

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OBSTETRIC CASE No. 6

PRETERM PREMATURE RUPTURE OF MEMBRANES-CONSERVATIVE MANAGEMENT-INDUCTION-LIVE BABY.

NAME:	R.L	DOA.	27.08 2002
AGE:	25 YEARS	DOD:	30.09.2002
IP NO	0986545	LMP:	02.02.2002
PARITY	1+0	EDD:	09.11.2002
		MBD:	30 WEEKS

PRESENTING COMPLALNT

The patient presented with a one day history of drainage of liquor.

HISTORY OF PRESENTING COMPLAINT

She was well until a day ago when while doing her household chores she experienced a sudden gush of fluid which drained on her thighs and feet. The fluid was clear. There was no vaginal bleeding or abdominal pains. She did not have any preceding history of frequency dysuria or vaginal discharge. The fetal movements were normal.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was a para 1+0. Her last delivery was in July 1998 SVD at term . to a live male infant whose birth weight was 3200gm and was alive and well. Her last menstrual period was on 02.02.2002 and an expected date of delivery of 09.11.2002. Hence she was at a gestation of 30 weeks at admission. She had attended antenatal clinic at a local City Council clinic for one visit where antenatal profile was done and was normal.

She attained her menarche at 14 years of age. Her menstrual cycle had been regular lasting 2-3 days every 26 days. She had used oral contraceptive pills from 1999 to mid-2000.

FAMILY AND SOCIAL HISTORY

She was a housewife. The husband worked for a construction company. She lived in Kariobangi. She neither smoked cigarettes nor took alcohol. There was no history of chronic illness in the family.

PAST MEDICAL HISTORY

This was not contributory.

GENERAL EXAMINATION

She was in good general condition with no pallor jaundice or edema. She was clinically afebrile. Her blood pressure was 125/70mmHg, pulse rate was 79 beats per minute and temperature of 36.9 C.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moving with respiration. The fundal height corresponded to 30 weeks gestation. There was no tenderness on palpation. The lie was longitudinal and presentation cephalic. Fetal heart tones were heard and regular at 140 beats per minute. No contractions were palpable. There was no hepatosplenomegaly or any other masses.

SPECULUM EXAMINATION.

Aseptic speculum examination revealed a closed cervix approximately 1cm long and was central. There was active drainage of clear liquor from the os with pooling of liquor in the posterior fornix. It was not foul smelling. The vaginal walls were normal.

The cardiovascular, respiratory and central nervous systems were essentially normal.

IMPRESSION

An impression of preterm premature rupture of membranes was made.

MANAGEMENT

The patient was informed of the need for hospitalization and was put on bed rest and given a fetal kick chart. The foot of the bed was raised using a stool. She was given sterile pads to monitor drainage of liquor. She was started on erythromycin tablets 500mg 6 hourly. Investigations were requested for and a summary is given below:

Blood Group	B Positive
VDRL	Negative
HIV Test	Negative

Hemogram. 29.08.2002		Repeat Hemogram 12.09.2004	
Hb	12.0g/dl	Hb.	11.8g/dl
W.B C	9.2 x 10 /L	W.B.C	8.4 x 10/L
Neutrophils	65%	Neutrophils	70 %
Lymphocytes	35%	Lymphocytes	30%
Eosiniphils	1%	Eosinophils	0%
Basophils	0%	Basophils	1%
RBC	3 8 x 10/L	RBC	4.1 x 10/L

Platelets	250x10/L	Platelets	300x10/L

Repeat Hemogram 24.09.2004

Hb.	12.5g/dl
W.B.C	7.4 x 10/L
Neutrophils	60%
Lymphocytes	40%
Eosinophils	1%
Basophils	0%
RBC	4.5 x 10/L
Platelets	310 x 10/L

Urinalysis Microscopy Culture and Sensitivity-09.09.2004

No growth obtained.

HVS Microscopy Culture and Sensitivity. 10.09.2004

Moderate growth of E.coli and Citrobacter species both sensitive +++ to Zinacef.

Obstetric Scan 29.08.2002

Single viable fetus in cephalic presentation. Fetal heart rate of 138 beats per minute. Age by biparietal diameter corresponded to 31 weeks and 2 days. Abdominal circumference corresponds to 30 weeks and 5 days and femur length corresponded to 31 weeks and 1 day. The liquor was reported to be adequate, the placenta was tundoposterior and no fetal anomalies were seen.

Repeat Obstetric Scan 26.09 2002

A single viable intrauterine fetus in cephalic presentation. Fetal heart rate of 140 beats per minute. Age by biparietal diameter corresponded to 35 weeks and 2 days. Abdominal circumference corresponded to 34 weeks and 5 days and femur length corresponded to 34 weeks and 4 days. The amount of liquor was reported to be reduced. The placenta was fundoposterior and no gross fetal anomalies were seen.

PROGRESS IN THE WARD.

The patient remained stable though she continued to have spotting of liquor and she could change pads twice in 24 hours. Her pulse rate ranged from 78 beats per minute to 85 beats per minute and her temperature range was 36.4C to 37.2 C. Fetal kick chart was adequate. In view of the HVS results she was put on intravenous Zinacef 750mg 8 hourly for 10 days. She also received intramuscular dexamethasone 12mg weekly. Upon receipt of the ultrasound report on 26.09.02 a decision was made to deliver her and she was informed and counseled accordingly. A speculum examination done on 27.09.02 revealed a closed cervical os approximately 0.5cm long and was central. There was little pooling of liquor in the posterior fornix. The patient was transferred to labor ward and started on syntocinon 5IU at 10 drops per minute at a constant rate until regular contractions commence then to be increased gradually every 30 minutes by 10 drops to a maximum of 60 drops per minute. She went into labor and 8 hours later she delivered vaginally to a live female infant whose birth weight was 2700gms with an Apgar score of 9 at 1 minute and 10 at 5 minutes. The placenta weighed 500gms. The baby was initially admitted to the New Born Unit where prophylactic antibiotics were started On the 2nd postnatal day the baby joined the mother in the ward and were both discharged on the 3rd postnatal day to be seen again in the postnatal clinic in 6 weeks.

She did not come to the postnatal clinic and she was lost to follow-up

DISCUSSION

The patient presented was a para 1+0 admitted at 30 weeks gestation by dates with a history of drainage of liquor for one day. She was managed conservatively and induced at 34 weeks gestation and delivered a live baby. In Kenyatta National Hospital conservative management is employed for any patient presenting with PPROM before 34 weeks , unless there are other factors necessitating delivery, thereafter active management is employed

Fetal membranes are composed of 2 layers (amniochorial membrane) derived from amnion and chorion.They are sealed and contain amniotic fluid by 12"' week of pregnancy.(l)

Premature rupture of membranes (PROM) is rupture of fetal membranes with leakage of amniotic fluid more than 8 hours before onset of labor regardless of gestation (2,3). Preterm premature rupture of membranes(PPROM) is rupture of membranes before 37 completed weeks. The patient presented was at 30 weeks gestation, therefore, she had PPROM.

Premature rupture of membranes (PROM) occurs in 10.7% of all pregnancies and 94% the fetus is mature while in 5% it is preterm (2,3). The incidence at Kenyatta National Hospital has been quoted as 9 3% in 1974 and 8.2% in 1980 (4,5). It has been shown that PROM contributes to 20% of all perinatal deaths (6).

The cause of PROM is not known and rupture usually occurs without warning in a woman whose pregnancy has appeared to be progressing normally. Risk factors associated with PPROM include, previous pre-term delivery, early and late pregnancy bleeding, cigarette smoking, maternal infections (e g urinary tract infection, general infections, intrauterine infectios), cervical incompetence, multiple pregnancies, polyhydramnios, nutritional deficits (e.g copper and Zinc deficiencies), digital pelvic examination and decreased tensile strength of membranes (2,6, 7)

The unifying factor for the risk factors is weakness in the chorioamnion membranes (2,6,7). Pseudomonas aeruginosa strains that produce collagenase has been found to decrease fetal membranes strength and elasticity. Other organisms associated with PPROM include Staph aureus, Strep agalactiae, Bacteroides melaninogenicus and Enterobacteriaciae spp that produce non-specific collagenase. Group B streptococci and E.Coli, which are implicated in chorioamnionitis, have been known to bind, invade and cross the chorioamnion membranes. Engulfing of the bacteria by amnion has been shown to activate the peroxidase-hydrogen peroxide system. Aminon, chorion, decidua and placental macrophages all contain peroxidase activity as do cervical mucus and endometrial cells. Some bacteria produce hydrogen peroxide, thus providing free radicals leading to local tissue destruction, necrosis and cleavage of the peptide bonds in collagen.

The diagnosis of PROM requires thorough history, physical examination and laboratory testing. History alone has an accuracy of 90% (8). A mother may describe a "gush of amniotic fluid", intermittent leaking of small amounts of fluid or increased perineal moisture. The next step is to do a sterile speculum examination for confirmation. The most reliable sign of rupture is direct observation of amniotic fluid flowing from the cervix into the vaginal vault. If fluid cannot be visualized application of slight fundal pressure or asking the patient to cough or bear down by valsalva maneuver induce leakage of amniotic fluid if the membranes are ruptured. The presence of meconium often verifies the diagnosis. In some cases amniotic fluid may be difficult to distinguish from urine, mucus and seminal fluid if flow of fluid from the cervix cannot be visualized.

In the event of uncertainly a number of diagnostic tests could be carried out. The most common used tests are analysis of vaginal Ph with nitrazine paper(which turns blue due to alkaline pH of amniotic fluid) and evaluation of vaginal secretions with arborization test. When the diagnosis is in doubt, other modalities including cytology, alpha fetoprotein detection on the draining fluid and intra-amniotic dye instillation using indigo carmine should be used as indicated (8).Complications of instillation of Indigo carmine include injuries to the fetus and risk of feto-maternal transfusion

Diagnosis of PPROM in the patient presented was made from history and speculum examination which revealed a pool of liquor in the posterior fornix with drainage from the cervical os.

The management of a patient with PPROM needs to be selective and individualized. There is general agreement that initial management of PPROM should include confirmation of rupture, determination of the presence or absence of bacterial infection, assessment of the gestational age, determination of fetal pulmonary maturity status, early detection of fetal distress and early detection of maternal and fetal infection. Fetal malpresentation, degree of cervical dilatation and the presence or absence of uterine contractions should also be considered. Also considered is the availability of efficient neonatal care unit. Most authors advocate for conservative management for a patient between 28 and 32 weeks and active management for patients after 36 weeks. Between 32 and 36 weeks there is no consensus as between 23 and 28 weeks of gestation (9).

The expectant management is aimed at prolongation of gestation as prematurity is then greatest risk. The need for bed rest, careful observation of the mother and fetus, daily heart rate monitoring and prompt maternal and fetal evaluation at the onset of labor usually imply that a patient with PPROM is hospitalized. Daily monitoring of liquor drainage, twice weekly white cell counts, culture of cervix and vaginal swabs and urine culture in all patients with PPROM where delivery is not imminent (9).

Most patients with PPROM deliver within 48 hours of membrane rupture. Approximately 90% term patients will progress spontaneously into labor with latency period of less than 48 hours, 80% of those at 33-36 weeks and 66% of those at 20-32 weeks will develop spontaneous labor (10). The patient presented was at 30 weeks and had drained liquor for one day and had no contractions. The drainage was very minimal subsequently up to 34 weeks when a decision to deliver was made. She was then induced with oxytocin and had a good fetal outcome.

Over the past decade antibiotics have emerged as an important treatment for PPROM. A number of well designed studies have shown improved neonatal outcomes with antibiotics alone and with antibiotics combined with corticosteriod therapy.Earlier studies had shown antibiotic therapy does not alter overall fetal outcome as measured by perinatal mortaliy (1). The NIH consensus development panel recommends the use of steroids in PPROM prior to 32 weeks in the absence of intra-amniotic infection. Use of steroids in the presence of infection will increase the rate of endometritis without corresponding benefit to the fetus (12). No studies have shown that tocolysis alone improve fetal outcome. The use of tocolytics in PPROM should be limited to 48 hours duration to permit admnistration of corticosteroids and antibiotics and to transfer the mother to a tertiary care centre where intensive neonatal care is available

Complications of PPROM include perinatal mortality, prematurity, chorioamnionitis, cord prolapse, placenta abruptio, oligohydramnios tetrad of pulmonary hypoplasia, peculiar faces, limb deformities and growth deficiency (11). Prophylactic transabdominal amnioinfusions with 150-350 mis warmed saline (5-50mls/min) at weekly intervals has been shown to have a positive effect on latency period in a patient with PPROM and olighydramnios. The amniopatch technique using infusions of cryoprecipitate has recently been used to try and 'seal the leaking membrane".

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OBSTETRIC CASE No. 7

UNDIAGNOSED CONGENITAL MALFORMATION-EMERGENCY CAESAREAN SECTION-LIVE BABY.

NAME	: L.L	DOA:	10.10.2004
IP No.	:09842391	DOD:	16.10.2004
AGE	:28 YEARS	LMP:	29.12.2003
PARITY	: 1+0	EDD:	06.10.2004
		MBD:	38 WEEKS

HISTORY OF PRESENTING COMPLAINT

The patient was admitted in labor ward with labor pains for the last 8 hours. There was no drainage of liquor.

PAST MEDICAL HISTORY

This was non contributory.

FAMILY SOCLAL HISTORY

She was a housewife. Neither smoked nor took alcohol. Her husband was a security officer.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a para 1+0, the last delivery being in the year 2003 by spontaneous vertex delivery in hospital. The outcome was a live normal male baby with a birth weight of 3500 grams who is still alive and well. Her last menstrual period was 29.12.03 and therefore an expected date of delivery of 6.10.04, giving a maturation by dates of 38 weeks. She had attended a total of 4 visits in a peripheral city council clinic for her antenatal follow-up. Her first booking was at 26 weeks gestation by dates. Her blood group was B +VE, VDRL was negative, HIV test was negative and her haemoglobin levels was 11.7g/dl In all the visits the fetus was noted to be in breech presentation. No obstetric ultrasound was done during the antenatal follow-up. She was not on any folate supplementation prior to and during pregnancy.

Her menarche was at 16 years, her menstrual flow was regular 3-4 days with a cycle of 28-30 days. She had not used any form of contraception.

GENERAL EXAMINATION.

She was in good general condition. Her blood pressure was 125/74mmhg, a pulse rate of 78 per minute and a respiratory rate of 19 per minute. Her temperature was 36.8C.

ABDOMINAL EXAMINATION.

The uterus was at 38 weeks by fundal height, the lie was oblique and the presentation was breech. The fetal heart rate was heard at 140 beats per minute. One mild contraction lasting 20 seconds was palpated during the examination.

PELVIC EXAMINATION.

The external genitalia was normal the cervix was 3cm dilated , 100% effaced and was soft and anterior. The descent was 5/5. The pelvis felt adequate

DIAGNOSIS

A diagnosis of a para 1+0 in active phase of labor with oblique lie and breech presentation was made and a decision to perform emergency caesarean section was made.

OPERATION

The patient was taken to theatre and caesarean section performed as described in the introductory pages of this book. The outcome was a live male baby who was in breech presentation and oblique lie and was extracted breech. Apgar score was 2/1 3/5 3/10 birth weight of 2250 grams. The baby was found to have several external anomalies including anencephaly, micropenis with absent testis bilaterally with the scrotal sac without ruggae. The baby was taken to the Newborn unit. The mother did well subsequently and was counseled on the pregnancy outcome. The baby was still alive by the 6$^{!h}$ postoperative day though was still in critical condition in the acute nursery.

DISCUSSION.

Between days 24 and 28 post conception, the neural plate closes to form the neural tube and the cranium. Incomplete closure of the former results in spina bifida and the latter in anencephaly. While anencephaly is incompatible with life, the embryo dying either in utero or shortly after birth, spina bifida can result in very mild to extremely severe clinical outcomes. In latter instance significant lack of closure can result in marked restriction in the quality of life, with paralysis of the bladder and lower limbs and hydrocephalus due to improper drainage of cerebral spinal fluid. Neural Tube Disease (NTD) have long been known to have a strong genetic predisposition. The strongest evidence of this is the marked increase in the risk of recurrence.(l, 2).

A woman with previously affected pregnancy has up to 10 fold increased risk of having recurrence 20 times for affected pregnancies and 40 times for 3 previous affected pregnancies. A marked difference is found between different ethnic groups with high levels in those of Celtic origin and very low levels in Afro-Americans. A non-genetic factor probable nutritional, could be responsible for NTD. Approximately 90% of index cases occur spontaneously without a previous occurrence in a family as was in our patient

Trials have been done where folic acid has been given to women before they become pregnant. Alternatively the prevalence of NTDs in women who just happened to be taking folic acid containing supplements during conception was compared to matched controls from the same community who also delivered a child at the same period but who were not taking supplements. Smithells et al showed reduction in NTDs of well over half that found in non-treated controls. Polyhydramnios is commonly found in these patients. (4)

Our patient was not on vitamin folate at preconception and during pregnancy

At .least 3-5% of all newborns have recognizable birth defect.(5). Neural tube defects have an overall incidence of 1-2/1000 births with a peak of 7/1000 births in South Wales. Neural tube defects are common in abortuses and account for 3% of all spontaneous abortions. Mati in Nairobi birth survey found the incidence of congenital malformations

to be 11.7/1000 births and the commonest were CNS malformations, which accounted for 45.2%.

Aketch in his study in KNH found the incidence of congenital malformation to be 1.9% and CNS malformations were the commonest 64%. In that study anencephaly accounted for 12.5% of all congenital malformations (5,6,7)

Anencephaly is one of the lethal congenital malformations. Half of the fetuses are still births and they accounted for 3 .7% of the overall perinatal mortality. Crowther in Harare found that 6.2% of the total, perinatal mortality rate was accounted for by lethal congenital malformation, the commonest being anencephaly(7,9). Of women carrying anencephalic fetuses, 60% have polyhydramnios.. This results from either transudation of fluids from exposed meninges into the amniotic cavity, increased diuresis due to lack of antidiuretic hormone or defective swallowing. Abdalla in her study on hydramnios at KNH found the incidence of polyhydramnios to be 0.14% and the incidence of congenital malformation was 30%, the commonest malformation being anencephaly(5,13). The patient presented had moderate hydramnios.

Anencephalic pregnancies may go beyond term especially if there is no polyhydramnios^. 12). This is due to reduced fetal substrate dehydroepiandosterone sulphate(DES) needed for placental estrogen synthesis. The low estrogens results in inadequate production of membrane phospholipid from which arachidonic acid is cleared for the synthesis of prostaglandin's E2 and F2. These are responsible for the intrinsic uterine contractions and effacement that occur during normal labor.

Clinical suspicion of anencephaly and other NTDs is obtained from the presence of polyhydramnios. In such cases there is difficulty in appreciating fetal parts on palpation or fetal heart sounds on auscultation. Malpresentation are common like was the case with our patient who had a breech presentation. The fetus is usually small for gestational age in most cases.

Diagnosis of NTDs relies on a detailed ultrasound examination of the fetus and biochemical examination of the amniotic fiuid(6,10). Anencephaly was the first congenital abnormality to be recognized in utero by ultrasound Diagnosis is by absence

of cranial vault, short neck, typical frog like appearance of the face, polyhydramnios is often present and can be recognized as early as twelfth week of gestation. With ultrasound, all cases of anencephaly and 80% of spina bifida are picked(6).

Biochemical tests include maternal serum alfa feto-protein and amniotic fluid alpha-fetoproteins which are elevated in open NTDs. Acetyl cholinesterase which is found in cerebrospinal fluid is increased in amniotic fluid in cases of NTDs. It can be used in distinguishing true positive from false positive in amniotic fluid levels of alpha-fetoprotein (6).

Prenatal screening programs by biochemical tests and ultrasound may permit diagnosis in early part of the second trimester.

Therapeutic abortion is usually performed, if the mother requests it, when continuation of the pregnancy will result in the birth of a child with severe abnormalities such as meroanencephaly(a now preferred term for anencephaly) because there is a rudimentary brainstem and functioning neural tissue that is always present in living infants (6,14).

Current evidence suggests that extra folate/folic acid is recommended for all women prior to conception and during the first 12 weeks of pregnancy and this prevents occurrence of NTDs

Women who have had a prior NTD-affected pregnancy are at high risk of having a subsequent affected pregnancy. When this women are planning to become pregnant, they should consult their obstetricians for advice (15).

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OBSTETRIC CASE No. 8

POST DATISM: INDUCTION-LIVE BABY

NAME:	J.M	DOA:	06.10.2004
IP NO	0987092	DOD	07 10.2004
AGE:	30 YEARS	LMP	13 12.2003
PARITY	4+0 GRAVIDA 5	EDD	20.09.2004
		MBD:	42 WEEKS

HISTORY OF PRESENTING COMPLAINT

J.M was admitted via casualty as a referral from Ruiru health centre with a diagnosis of postdatism.

PAST **MEDICAL HISTORY**

This was not significant.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a para 4+0 gravida 5. Her last menstrual period was on the 13.12.2003 with an expected date of delivery of 20.09.2004. All her previous pregnancies were uneventful all being hospital normal deliveries. The children were alive and well. In the current pregnancy she attended Ruiru health centre at a gestation of 26 weeks. The antenatal profile done was normal. Haemoglobin of 12.8g /dl. VDRL and HIV tests were negative. Her blood group was O positive. Her menarche was at 15 years. Her menstrual cycle was 28 days and the menses lasted 3-4 days. She had not used any form of contraception.

FAMILY AND SOCIAL HISTORY

She was a married business woman. She never smoked cigarettes nor took alcohol. There was no history of chronic illness in the family.

PHYSICAL EXAMINATION

She was in good general condition, not pale, nor jaundiced, no edema and she was afebrile. Blood pressure was 120/80mmHg, temperature 36.7 C , pulse rate of 79 beats per minute and a respiratory rate of 18 breaths per minute.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and was moving with respiration. Fundal height was term, the lie longitudinal, cephalic presentation, presenting part was 5/5 above the pelvic brim, fetal heart rate was 140 beats per minute. The respiratory, cardiovascular and the central nervous system were normal.

PELV IC EXAMINATION

She had normal external genitalia, cervix was parous with the os admitting tip of finger. It was central but firm. The Bishop score was 3 and therefore poor.

DIAGNOSIS

Postdatism with unfavorable cervix.

MANAGEMENT

A decision was made to induce the patient and this was communicated to her. 3mg of PGE2 pessary was inserted in the posterior vaginal fornix and the patient was then advised to rest in the bed. Five hours later she complained of labor pains and was transferred to labor ward. Pelvic examination revealed a cervix which was 4cm dilated and she had mild contractions 1 in 10 minutes. Artificial rupture of membranes was done and she was augmented with intravenous syntocinon 5 IU in 500mls dextrose to run at 10 drops per minute to be increased by lOdrops every 30 minutes up to 60 drops per minute. She progressed well and 5 hours after admission into labor ward she delivered by spontaneous vertex delivery to a live female infant with an Apgar score of 9/1 and 10/5 with a birth weight of 3600 grams. The infant was well with no external congenital anomalies. She was discharged home the following day to be seen in the post natal clinic

POST NATAL CLINIC

She did not attend the post natal clinic six weeks later and was lost to follow-up.

DISCUSSION

The patient presented was a para 4+0 gravida 5 who was admitted with postdatism. She was induced with prostaglandin E2, went into labor and eventually delivered SVD to a live healthy female infant.

A post date pregnancy is one that has gone beyond 42 weeks from the last menstrual period but currently the concept is being changed to 40 weeks though the range is from 40-41 weeks (1). Elfenesh in a study carried out at Kenyatta National Hospital and Pumwani Maternity Hospital found the prevalence of postdate pregnancy to be 4.9%(2).

The exact cause of post date pregnancy is not yet known but a few conditions have been associated with it e g fetal adrenal hypoplasia, absence of fetal pituitary gland, placenta sulfactase deficiency and extrauterine pregnancy.The common factor in these cases is the low levels of estrogen (3,4). It is thought that the low estrogen results in low membrane phospholipids from which arachidonic acid is cleaved for the synthesis of prostaglandin E2 and F2 which play a significant part in the induction of labor.

Other factors associated with postdatism are improved living standards and hereditary. This is because postdatism has been shown to recur and at times runs in families
The diagnosis of postdatism is difficult to arrive at but generally depends on taking a exhaustive history e.g of the last menstrual period, contraceptive history, coital frequency the latter useful only if pregnancy resulted from a single episode of coitus, history of quickening, perusal of the patients' earlier records i.e first trimester examinations, first trimester ultrasound reports e.t.c.

The management of postdate pregnancy depends on certainity of dates and presence or absecence of other obstetric conditions. However, generally, vaginal delivery is aimed at .Post datism is one of the commonest indications of induction of labor (4). It is usually recommended that surfactant test be done before induction of labor to ascertain lung maturity. If it is satisfactory it is followed by insertion of of prostaglandin E2 pessay 3mg in the posterior vaginal fornix followed by amniotomy and oxytocin infusion.

In women with Bishop score of 7 and above, amniotomy is carried out without prostaglandins. The patient presented had a poor Bishop score. Other methods of induction are use of laminaria tents, ballooned foleys catheter among others. Complications of post datism are intrauterine growth retardation, fetal distress, macrosomia as the fetus continues to grow with subsequent dystocia, oligohydramnios and placental insufficiency. There is also increased rate of caesarean section.

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OBSTETRIC CASE No. 9

PLACENTA PRAEVIA AT 35 WEEKS-CONSERVATIVE MANAGEMENT-ELECTIVE CAESAREAN SECTION-LIVE BABY

NAME:	P.M	DOA:	12.12.2002
AGE:	28 YEARS	DOD:	04.01.2003
IP.NO	0981887	LMP:	15.04.2002
PARITY	1+0 GRAVIDA 2	EDD:	22.01.2003
		MBD	35 WEEKS

PRESENTING COMPLAINT

P.M presented with a one day history of painless vaginal bleeding.
She was well until one day prior to admission when she experienced sudden fresh per vaginal bleeding. There was no associated lower abdominal pain and no passage of liquor. There was no history of trauma. **By** the time of admission the bleeding had **subsided.**

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a para 1+0 whose last delivery was in 1996 to a live male infant who is alive and well. Her menarche was at the age of 15 years and she had regular menses every 28 days with a flow of 3 days. She had no dysmenorrhoea. She had been using oral contraceptives.

HISTORY OF CURRENT PREGNANCY

Her last menstrual period was on 15.04.2002 with an expected day of delivery of 22.01.2003 giving her a maturation by dates of 35 weeks at the time of admission. She attended antenatal clinic in a private clinic at Mathare North. The antenatal profile was done; Hb-11 8g/dl, blood group A positive, VDRL negative and HIV test negative.

PAST MEDICAL HISTORY

This was not significant.

FAMILY AND SOCLAL HISTORY

She was a married businesswoman. She neither smoked cigarettes nor took alcohol. There was no history of chronic illness in the family.

GENERAL PHYSICAL EXAMINATION

She was in fair general condition. She had no pallor, jaundice, edema or cyanosis and was clinically afebrile. She had a pulse rate of 78 beats per minute, blood pressure of 115/75mmHg, respiratory rate of 19 per minute and a temperature of 37.1 C.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moved with respiration. The fundal height was 34 weeks. The fetal presentation was cephalic with a longitudinal lie and the fetal head was 5/5 above the pelvic brim. There were no palpable contractions. The fetal heart tones were heard and were regular with a rate of 142 beats per minute.

SPECULUM EXAMINATION

The external genitalia were normal. A speculum examination with Cusco's bivalve speculum revealed normal healthy cervix with a closed os. There was no active bleeding from the os. There were blood clots on the vagina, which were removed. The vaginal walls were healthy.

The respiratory system, cardiovascular and central nervous systems were normal.

URGENT ULTRASOUND

This showed a single normal intrauterine pregnancy in cephalic presentation. Fetal somatic and cardiac activity was present with a fetal heart rate of 140 beats per minute. The amount of liquor was adequate. The placenta was anterior and low lying covering the internal os completely. An ultrasound diagnosis of placenta praevia type 4 was made.

DIAGNOSIS

A diagnosis of antepartum hemorrhage secondary to placenta praevia was made.

MANAGEMENT

Since the patient was not actively bleeding and was hemodynamically stable, it was decided that she be managed conservatively. She was to be put on bed rest.

Blood was taken for group and cross matching and two units of whole blood kept on standby for her. She had an intravenous access left insitu. She was given sterile pads to monitor the vaginal bleeding. She was also started on fetal kick chart. Blood was taken for full hemogram and her hemoglobin was found to be 10.4g/dl. While in the ward she did not have any bleeding and the fetal activity remained normal.

After 37 completed weeks, on 01.01.2003, she underwent elective caesarean section and the outcome was a live female infant with an Apgar score of 9/1 and 10/10 and a birth weight of 3200 grams. The placenta was found to be Type 4 intraoperatively as shown by the ultrasound.

POSTOPERATIVE MANAGEMENT

She did well post-operatively and was discharged home on the fourth day on antibiotics and analgesics for removal of stitches on the seventh day in the nearest health center She was advised to come to the postnatal clinic after six weeks.

POSTNATAL CLINIC

She came after 6 weeks. The incision site had healed well and she had no complaint. Both mother and baby were doing well. She was advised on contraception and she chose oral contraceptives.

DISCUSSION

The patient presented with antepartum hemorrhage due to placenta praevia at 35 weeks. Antepartum hemorrhage is defined as vaginal bleeding which occurs at any time after the 28^{th} week of pregnancy and before the birth of the child. It may be as a result of obstetric or non-obstetric causes. Obstetric causes include placenta praevia, abruptio placentae, vasa-praevia, uterine rupture, bloody show and abnormal blood-clotting mechanisms. Non-obstetric causes include cervicitis, cervicasl polyps, cervical erosions, vaginal lacerations, benign and malignant neoplasms of the cervix and vagina.
Placenta praevia is defined as implantation of the placenta partly or wholly on the lower uterine segment. Four degrees of the abnormality have been recognized

TYPE I- Low lying placenta. The placenta is partly implanted on the lower segment but the placental margin does not reach the internal os. It is sometimes called a lateral placenta praevia.

TYPE II-Marginal placenta praevia. The edge of the placenta is at the margin of the internal os

TYPE Ill-Partial placenta praevia. The internal os is partially covered by the placenta.

TYPE IV- Complete or total placenta praevia. The placenta completely covers the internal os

Placenta praevia occurs in approximately 0.5% (1 in 200) deliveries. At Kenyatta National Hospital, Ojwang found an incidence of 1 in 400 deliveries.

The etiology of placenta previa is not known with certainity but it is thought to be multifactorial. Possible etiologic factors include scarred or poorly vascularized endometrium, a large placenta and abnormal forms of placentation such as succenturiate lobe or placenta diffusa. Some factors have been found to predispose to placenta praevia.

1. Advancing maternal age. Maternal age> 35 years
2. Black race
3. Multiple pregnancies; because of increased surface area of placenta or placentas
4. Prior caesarean delivery. Studies have found a linear increase in placenta praevia risk with the number of prior caesarean deliveries. In patients with four or more caesarean deliveries, the risk of placenta previa approaches 10%.
5. Smoking- Studies have found a two fold increase in the relative risks of placenta praevia with smoking. This is probably due to carbon monoxide hypoxemia causing compensatory placental hypertrophy (1,2,4).

The patient presented had the race as the only risk of the above risk factors.

Bleeding in placenta praevia may be due to mechanical separation of the placenta from its implantation site as a result of effacement and dilatation of the cervix, placentitis or rupture of poorly supported venous lakes in the decidua basalis that have become engorged with venous blood (3).

The timing of placenta praevia diagnosis has undergone significant changes. Initially third tnmester bleeding was a common presentation for placenta praevia but currently with the advent of ultrasonography, most cases of placenta praevia are detected before the onset of significant bleeding. Management of placenta praevia diagnosed in the second trimester differs from the same diagnosis in the third trimester. At 17 weeks gestation, evidence of placental tissue covering the cervical os will be found in 5 to 15% of all patients but more than 90% of this patients will have a normal ultrasound by 37 weeks gestation.This is called placental migration and is probably due to differential growth of the lower uterine segment during the second and third trimesters. Total placenta praevia is diagnosed in the second trimester in 26% of cases, while marginal or partial placenta praevia will persist in only 2.5% of cases. Placenta praevia should be suspected in all cases of patients with bleeding after 24 weeks gestation.

Bleeding from placenta praevia has its onset without warning, presenting without pain in a woman who had an uneventful prenatal course. This happened in our patient. The initial bleeding is rarely so profuse as to prove fatal. It usually ceases spontaneously. In some cases, particularly in those with a placenta implanted near but not over the cervical os, bleeding does not appear until the onset of of labor when it may vary from slight to profuse hemorrhage and may clinically mimic placental abruption. Haemorrhage from the placental implantation site in the lower uterine segment may continue after delivery of the placenta, because the lower segment is prone to contract poorly. Bleeding may also result from lacerations in the friable cervix and lower uterine segment especially following manual removal of a somewhat adherent placenta. In this patient the placenta was delivered manually at caesarean section and hemostasis was achieved and no postpartum hemorrhage was encountered.

The diagnosis of placenta praevia is mainly clinical. The cardinal symptom of placenta praevia is painless per vaginal bleeding. However spotting may occur during the first and second trimester. The diagnosis of placenta praevia can only be established firmly when a finger is passed through the cervix and the placenta is palpated. This examination is however only permissible when the woman is in an operating room with all preparations for immediate caesarean delivery ready. This is because even the gentlest examination of this sort can cause torrential hemorrhage. Clinical examination of the abdomen will reveal a soft non-tender uterus. The presenting part will be high and cannot be pressed in the pelvic inlet. In about 15% of cases, the fetus will present in an oblique or transverse lie. The fetal heart rate is unaffected.

Placental localization can be done by ultrasonography. The accuracy rate of placental localization with ultrasonography is over 95%. .Another method of placental localization is magnetic resonance imaging. Other methods which have become obsolete include arteriography, soft tissue radiography and isotope localization. Ultrasonography has no place in cases with severe hemorrhage where immediate delivery by caesarean section is indicated. In our patient, an ultrasound was done because at admission, the bleeding had stopped and the fetus was premature.

On admission, careful history taking and physical examination will usually clinch the diagnosis. A quick and thorough physical examination is done and a gentle speculum examination is done to assess the bleeding. An intravenous line is established with a wide bore canula, blood is taken for grouping and cross matching and at least three units of blood cross matched and a full hemogram requested for.

Once the diagnosis is made, the management decision depends on the gestational age, amount of bleeding, viability of the fetus, presentation, status of the cervix and whether or not labor has began.Depending on the above, the mode of management will either be expectant therapy or immediate delivery. Expectant management is indicated in a patient who is remote from term but with no active bleeding. It involves a period of close observation in the hospital until bleeding stops or the onset of labor or bleeding worsens. Maternal blood loss should be replaced in order to maintain the maternal hematocrit between 30 and 35 %. (1,2,3).

There is no consensus on how long the patient should stay in hospital once bleeding has completely ceased and the fetus is judged healthy. In some centers, prolonged hospital stay until term is practiced while in others the patient is usually discharged after bleeding has ceased and the patient and family counseled on placenta praevia and the need to transport her quickly to the hospital once bleeding recurs. In our set-up, the practice is to keep the patient on bed-rest in the hospital until term or when labor sets in since our patients may not be able to come to hospital urgently should bleeding start while they are at home.

During this time, the patient is on conservative management. She should be on bed rest and blood should always be available for maternal transfusion in the event of a sudden hemorrhage. Corticosteriods are indicated to accelerate fetal pulmonary maturation. They have been found to be effective in reducing the incidence of neonatal respiratory distress syndrome, intracranial hemorrhage and neonatal death.

In rhesus negative patients on epectant management, Rh immunoglobulin should be given to all who are not sensitized. A Kleuhauer-Betke test should be performed in all Rh-negative patients to detect the occasional patient with a feto-maternal hemorrhage of greater than 30mls. 35% of infants whose mothers require antepartum transfusion will themselves be anemic and require transfusion when delivered.

If bleeding is not heavy to warrant termination of expectant managemant or ceases altogether, the optimum time of delivery is determined by tests of fetal lung maturity. Amiocentensis is done at 37 weeks for surfactant test and if mature, then elective caesarean section is carried out.

In this patient the gestation was 35 weeks and she was managed conservatively on bed rest. She was scheduled for elective caesarean section.

Immediate delivery is indicated when:-

a. The hemorrhage is severe irrespective of gestation
b. There is persistent bleeding
c. There is established labor whatever the gestation
d. The patient has completed 37 weeks.

The mode of delivery in most cases of placenta praevia except type I is by caesarean section. The placental location and the development of the lower uterine segment will determine the type of incision on the uterus. In an anteriorly placed placenta and if the incison passes through the placenta, there is a strong possibility that the fetus will lose significant amount of blood enough to require transfusion. A classical incision may be required to secure sufficient room and to avoid incision through the placenta. In a posteriorly placed placenta a low transverse incision may be be best if the lower segment is well developed. In all cases the paediatric team should be informed to expect a high risk infant Hypovolemic shock should be corrected by intravenous fluids and blood before the operation is started.

The most common complication following caesarean section delivery in placenta praevia is uncontrollable hemorrhage following placental removal This is due to the poor contractility of the lower uterine segment. To control the bleeding, oversewing the implantation site with mattress sutures or packing together with oxytocin and prostaglandins will be necessary. In others bilateral uterine artery ligation may be necessary and in others internal iliac artery ligation will be required. If this conservative methods fail and bleeding is heavy then hysterectomy is necessary to save the mother. Other postpartum complications include puerperal infection and anemia.

If the bleeding is severe in undiagnosed placenta praevia, an indirect method is by the double set up examination. This is done in theatre in an operating room with full preparation made to perform emergency caesarean delivery should torrential bleeding follow the examination. In this, the patient is prepared and ready for caesarean delivery. A careful speculum examination is done. If no placenta is detected by speculum examination, a digital examination is carried out starting with vaginal fornices to detect fullness in them which suggests the presence of placenta extending down towards the cervix. Finally, the examining fingers should be carefully introduced into the cervical os to detect the placenta.

Vaginal delivery is reserved for patients with type I or type II anterior and a cephalic presentation. If vaginal delivery is elected, the membranes should be artificially ruptured prior to any attempt to stimulate labor. Tamponade of the presenting part against the placental edge usually reduces bleeding as labor progresses.

The maternal prognosis in placenta praevia is good in the presence of antibiotics, a blood bank, expertly administered anaesthesia and an appropriately done caesarean section. There is a marked reduction in perinatal mortality with expectant management. In most centers, the perinatal mortality rate associated with placenta praevia is 15-20%. Preterm delivery is a major cause of perinatal death. (1,3).

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OBSTETRIC CASE No. 10

CERVICAL INCOMPETENCE:MAC-DONALD STICH-LIVE BABY.

NAME	H.N	**LMP**	: 14.01.2004
AGE	**30 YEARS**	**EDD**	:21.10.2004
PARITY'	2+2 G 5	**D.O.A**	: 14.04.2004
IP.NO.	0861414	**D.O.D**	18.04.2004

PRESENTING COMPLAINTS.

She had no major complaint and had been admitted via the antenatal clinic for insertion ofMacDonald stitch. She had been seen in the antenatal clinic from 10 weeks gestation and the clinical findings were suggestive ofcervical incompetence.

PAST OBSTETRIC AND GYNAECOLOGICAL HISTORY.

She was a para 2+2 with one living child. In 1997 she had an abortion at 5 months and no evacuation was done.. In 1999 she had a spontaneous vertex delivery at 8 months to a live female baby who is still alive and well. In 2002 she had spontaneous vertex delivery at 8 months to a live male baby who passed away at 2 months due to pneumonia. In 2003 she had a spontaneous abortion at 5 months and evacuation was done. Both fetal losses started with drainage of liquor followed by abdominal pain and later expulsion of the respective fetuses. She had no history of trauma or febrile illness associated with the abortions. Her menarche was at 15 years and the menses lasted 4-5 days with a regular cycle of 26 to 28 days. She had not used any form of contraception.

PAST MEDICAL HISTORY

This was non contributory.

CURRENT PREGNANCY.

Her last menstrual period was on 14.01.2004 and expected date of delivery was 21.10.2004 giving her a maturity of 14 weeks. She had been booked in the antenatal clinic at KNH at 10 weeks and this was her second visit.

FAMILY AND SOCIAL HISTORY

She was a housewife. Her husband was a driver. She neither took alcohol nor smoked cigarettes. There was no family history of chronic illness.

PHYSICAL EXAMINATION.

She was a young woman in good general condition with no pallor, jaundice, cyanosis , edema or lymphadenopathy.Temperature was 36.8C, pulse rate of 76 beats per minute, respiratory rate of 19 per minute and a blood pressure of 115/70mmhg. The respiratory, cardiovascular and the central nervous system were essentially normal.

ABDOMINAL EXAMINATION.

She had slight lower abdominal distension. There were no areas of tenderness. The uterine fundus corresponded to 14 weeks gestation. There were no other palpable masses.

PELVIC EXAMINATION

Speculum exam: She had normal external genitalia with normal vaginal walls. The cervix was patulous and short but with no visible defect.

Digital exam: The cervix was short ,0.5cm long and central but was firm. The internal os admitted 1 finger but no defects were felt. There was no discharge on the examining fingers.

DIAGNOSIS

A diagnosis of a patient with fetal wastage secondary tc cervical incompetence was made and she was planned for insertion of MacDonald stitch.

INV ESTIGATIONS DONE.

Hemoglobin level	11.2 g/dl
VDRL	Negative
Random Blood sugar	4.6 mmol/L
Brucella titres	Negative
Blood Group	B positive
T3	2.04 mmol/L (0.9-2.5)
T4	102.8 mmol/L (30-150)
TSH	0.84 umol/L (0.3-7.0)

Urinalysis for microscopy culture and sensitivity was reported to be normal.

An ultrasound done showed a live single intrauterine pregnancy of 15 weeks gestation Cervix was reported to be short and patulous. The placenta was fundo-posterior Amount of liquor was adequate. No fetal abnormalities were seen.

MANAGEMENT

The patient was counseled about the diagnosis and the operation to be performed. An informed consent was obtained. She was starved from midnight. On the morning of the operation the vulva was shaved, she was pre-medicated with atropine 0.6mg intramuscularly, and wheeled to theatre.

Mac Donald Stitch Insertion.

In theatre, she was given general anaesthesia. In litothomy position vulvovaginal toilet was done with savlon solution and she was draped with sterile towels. The bladder was catheterized and clear urine drained. Examination under anaesthesia confirmed the earlier findings.

An Auvard speculum was inserted into the vagina to expose the cervix, which was then held with tenaculum forceps on the anterior and posterior lips. The cervix was gently drawn towards the introitus. Using a No. 2 mersiline silk suture on round body a purse string cerclage was inserted at the level of internal os. The needle was directed into the stroma to avoid the endocervical canal. Four bites were taken by introducing through position 5 o'clock out through 4 o'clock, in through 2 o'clock out through 1 o'clock in through 11 o'clock out through 10 o'clock and finally in through 8 o'clock and out at 7 o'clock. A knot was placed at 6 o'clock and tightened just enough to admit the tip of a finger to allow for secretions to pass. A 2cm strand was left in order to facilitate easy removal. There was slight bleeding after the procedure but no drainage of liquor. The cervix was wiped with a sponge dipped in betadine solution. The patient was reversed from general anaesthesia.

POSTOPERATIVE CARE

She was taken to recovery ward and observed half hourly until she was fully awake. She was the transferred to the antenatal ward. She was put on Amoxil 500mg 8 hourly, and ventolin 4mg 8 hourly. She was to be on complete bed rest while in the ward.

On the second post-operative day she had no complaint and was discharged home on the same medication for five days. She was advised to abstain from coitus and to report to hospital in case she developed lower abdominal pain, drainage of liquor or vaginal bleeding. She was to be reviewed in the antenatal clinic in two weeks. The stitch was to be removed at 37 weeks.

RE-ADMISSION.

She continued with antenatal clinic uneventfully during her six subsequent visits. The stitch was removed in labor ward on 27.09.2004 when she was at 37 weeks gestation. She came back in labor on the 10.10.2004 at 39 weeks. Labor progressed well and she delivered by spontaneous vertex delivery to a live female infant weighing 3200 grams and scored 7 in one minute and 10 in ten minutes. She and her baby did well and were discharged the following day to be seen in the post-natal clinic in six weeks.

FOLLOW-UP

She was lost to follow-up.

I

DISCUSSION

The patient presented was a 30 year old para 2+2 with fetal wastage due to cervical incompetence. She had Mac Donald stitch inserted at 14 weeks gestation and delivered a live baby at 39 weeks gestation.

In normal pregnancy the cervix remains closed and retains products of conception within the uterus. During the third trimester, the cervix softens in preparation for parturition. Cervical incompetence can be due to congenital or acquired factors.
Some women experience cervical effacement and dilatation with every pregnancy; others have one or more uncomplicated births at term before presenting with the typical manifestations of cervical insufficiency. With cervical insufficiency there is second trimester or early third trimester fetal loss characterized by painless cervical dilatation with prolapse and ballooning of membranes into the vagina followed by rupture of membranes and expulsion of an immature fetus(l). The patient presented had second trimester pregnancy losses though were not consecutive and not very typical of cervical incompetence.

The incidence of cervical incompetence ranges from 0.05-1% per 100 pregnancies (2). At KNH, Njagi reported an incidence of 1 in 90 deliveries (3). The risk of preterm delivery rises by four times after one preterm delivery and about 10% of preterm deliveries are caused by true cervical incompetence(4).

The classic presentation of cervical insufficiency is cervical dilatation and effacement in the second trimester with fetal membranes visible at or beyond the externanl os in the absence of contractions It may be asymptomatic or associated with one or more of the following: Vaginal fullness or pressure, vaginal spotting or bleeding, an increased volume of watery, mucus or brown vaginal discharge or vague discomfort in the lower abdomen or back.

A clinician should suspect cervical incompetence in a patient with recurrent second trimester pregnancy losses. Other, more subtle markers of reduced cervical resistance include soft cervical consistency on digital examination, a history of short labors, advanced dilatation before the onset of labor and progressively earlier deliveries with each successive pregnancy. Funneling of fetal membranes into or completely through the

endocervical canal (hour-glassing) or shortening of cervical length and dilatation of the internal cervical os are indicators of cervical incompetence on ultrasound examination.

The etiology of abnormal cervical function can be divided int two major categories: Congenital abnormality and trauma although factors such as uterine overdistension and biochemical abnormalities also play a role. Developmental causes of reduced cervical competence include: congenitally short cervix, mullerian duct abnormalities and in utero exposure to diethylstilbestrol.

Congenital shortness of the cervix appears to be the commonest cause of cervical incompetence. In a prospective study of 2189 women cervical length was estimated at 24 weeks using ultrasound. Cervical length was normally distributed with a mean of 35mm. The relative risk of preterm birth was 10 fold-higher(positive predictive value about 25 percent) in women whose cervical length fell below the fifth centile (22mm) compared to those at the 75th centile(<40mm) (7). The risk of cervical incompetence is highest among women with unicornuate or bicornuate uterus (8). Incompetence of the cervix may occur in women exposed to diethylstilbestrol in utero where there were resultant cervico-vaginal anomalies.

Traumatic causes of cervical incompetence may be physiological or iatrogenic and the later includes: Cervical lacerations following spontaneous vaginal delivery, prolonged second stage of labor, instrumental vaginal delivery, cervical injury at the time of caesarean delivery and surgical procedures involving the cervix (e g mechanical dilatation, cone biopsy and cervical amputation as done in management of CIN (8,9).

Over distension of the uterus as occurs in twin gestation or polyhydramnios may result in cervical shortening or biochemical changes. Although a shortened cervical length at 24 weeks of gestation is the most powerful predictor of preterm birth in twins, cerclage does not appear to improve the outcome of twin gestations. However this procedure may be of value in higher order multiple pregnancies (9). Over distension in the preceding pregnancy may result in cervical incompetence in subsequent pregnancies.

There can be also asynchrony between the cervix and the uterus probably due to a biochemical cause such that the cervix dilates withpuot perceptible contraction or vise

versa (1,2). In the patient presented there was no identifiable cause of cervical incompetence.

Cervical dilatation characteristic of cervical incompetence seldom becomes prominent before the sixteenth week of gestation. This is because before that period, the products of conception are not sufficiently large to efface and dilate the cervix except when there are uterine contractions (1).

Diagnosis of cervical incompetence is largely made from history and physical findings. In pregnancy, abdominal and especially endovaginal ultrasound has facilitated the diagnosis of cervical incompetence. It may show an open os with herniation of fetal membranes and is accurate in assessment of cervical length (3,4). Outside pregnancy several tests can be performed. Hysterogram may show isthmal funneling. Kagia at KNH demonstrated evidence of cervical incompetence in 82.6% of patients who had preterm deliveries six weeks later by use of hysterosalpingogram (10). Other tests include passage of a size 6-8 Hegar dilators through the cervix with ease, and traction test by use of a foley catheter ballooned with 1ml of water and traction of 600mg applied (1).

The treatment of cervical incompetence is surgical, consisting of reinforcement of the weak cervix by some type of purse-string suture. It is best performed after the first trimester but before cervical dilatation of 2-3 cm is reached (1). The best time for insertion is at 14 weeks so that early abortion secondary to causes like congenital and genetic abnormalities will be complete by then. Njagi found that Mac Donald stitch gave best results if it was done between 13 and 19 weeks gestation (3).

Ultrasound must be done to exclude fetal congenital anomalies and to confirm a living fetus before cerclage (1). If substancial dilatation of the cervix has occurred, or bulging of membranes has occurred, then the likelihood of a successful cerclage is lessened. An attempt can be made to replace the protruding membranes with a balloon which is the deflated and removed (2). The patient presented had cerclage done at 14 weeks gestation.

Three types of operation are commonly used during pregnancy including Mac Donald stitch, Shirodkar stitch and modified Shirodkar stitch. There is less trauma and blood loss with both Mac Donald and modified Shirodkar than with the original shirodkar which is

often preserved for previous failed Mac Donald procedure and structural cervical abnormalities (1,2). Mac Donald stitch can be removed and so can the modified Shirodkar unlike conventional Shirodkar, which was a permanent stitch requiring caesarean section for delivery. Transabdominal cerclage may be appropriate in rare circumstances. These include traumatic cervical lacerations, congenital shortening of the cervix, advanced cervical dilatation and previous failed cervical cerclage (2, 11). Disadvantages include need to perform two operations (one for suture placement and another for caesarean delivery), and risk to injury of uterine vessels and the ureter (2).

Contraindications to cervical cerclage include rupture of membranes, uterine bleeding, uterine contractions, chorioamnionitis, cervical dilatation greater than 4cm, polyhydramnios and known fetal anomaly (1,2). Complications of cerclage especially when performed after 20 weeks gestation are high. The complications include haemorrhage, rupture of membranes, infection (chorioamnionitis, septicaemia), induction of preterm labor, cervical dystopcia, cervical laceration or uterine rupture at time of delivery, vesico-vaginal fistula formation and fetal death (1,2).

MacDonald stitch is removed at 36 completed weeks of gestation, or if there is vaginal bleeding, drainage of liquor, or premature labor sets in. Our patient did not have any complications and the stitch was removed at 37 weeks.

The success rates with both Mac Donald and the modified Shirodikar techniques approach 85-90% (1). Njagi found the success rate leading to term pregnancy to be 55% and 64.2% in fetal survival (3). Most case series quote a viable delivery rate of 70 to 90 % after cerclage, compared to 10 to 30 per cent prior to the procedure (12).

The efficacy of surgical treatment in women who are incidentally identified to have a short cervix also requires investigation. The challenge is to identify those patients early in pregnancy that will benfit from prophylactic cerclage. However there is no reliable mechanism to accomplish this.

Conservative management requires patients to have bed rest at home and weekly ultrasound are done from 15 weeks of gestation and if shortening at or below the critical length of 15mm is noted emergency cerclage is done. All cerclages are to be removed if

there is evidence of preterm premature rupture of membranes, preterm labor unresponsive to tocolytics or attainment of 36 completed weeks of gestation.

Therapeutic bed rest is the most commonly employed non-surgical approach to the management of incompetent cervix, although its benefit has never been proved in randomized controlled trials (13). Patient compliance for bed rest is also poor Other suggested modalities include intramuscular hyroxyprogesterone plus a program of bed rest (14) , and the use of several types vaginal pessaries and inflatable balloons in an attempt to change the axis of the cervical canal, thereby altering the gravitational force of intrauterine contents on the cervix. There is generally no indication for tocolytic therapy unless preterm labor is also present.

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OBSTETRIC CASE No. 11

PARA 0+1 WITH UNDIAGNOSED TWINS IN PREGNANCY: CAESAREAN SECTION-LIVE BABY.

NAME.	H.K	DOA:	02.10.2004
IP NO:	0986585	DOD:	06.10.2004
AGE:	25 YEARS		
PARITY:	0+1		

HISTORY OF PRESENTING COMPLAINT

She was admitted to labor ward with history of lower abdominal pains and drainage of liquor for 6hrs. The pains were increasing in intensity. There was no per vaginal bleeding.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was para 0+1 with the first pregnancy having ended up in spontaneous abortion at a gestation of three months in 2002. She attained her menarche at 14 years and subsequent regular menstrual cycle every 28 days lasting for 3 days.

She reported no use of contraception.

HISTORY OF PRESENT PREGNANCY

Her last menstrual period was on 16.01.2004 with expected date of delivery of 16.10.2004.Fetal maturity by dates was about 38 weeks. She was attending antenatal clinic at Gichuru dispensary in Satellite Nairobi. The antenatal profile was done with a haemoglobin level of 11.3g/dl VDRL was negative. HIV test was negative and her blood group was B positive. Twin pregnancy was not suspected antenatally.

PAST MEDICAL HISTORY

This was not contributory

FAMILY SOCIAL HISTORY

She was a married businesswoman living with her husband who was a driver. She did not smoke nor take alcohol. There was no family history of twinning or any chronic illness.

PHYSICAL EXAMINATION.

She was found to be in good general condition with no pallor, jaundice or cyanosis. She had bilateral pitting bipedal edema. Her blood pressure was 110/75mmhg with a pulse rate of 77 beats per minute. Her temperature was 37.0 C. with respiratory rate of 21 per minute.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and the fiindal height was corresponding to a term gestation. The fetus was presenting cephalic and was felt to be a big baby. The fetal lie was longitudinal and one fetal heart rate was perceived which was 145 beats per minute and was regular. She had mild contractions, one in five to ten minutes.

PELVIC EXAMINATION

She had a normal external genitalia. The cervical os was 3cm dilated, 80% effaced thin and central. The descent was 4/5 the membranes were felt and were flat.

Cardiovascular, respiratory and the central nervous systems were essentially normal.

MANAGEMENT.

A partograph was started and the patient was examined 4 hours later. The contractions were moderate, 2 contractions in five minutes, the descent was 3/5 cervical dilatation was 5 cm. Artificial rupture of membranes was done. Clear liquor was obtained. The fetus was found to be in persistent occiput posterior position with second degree moulding. A decision to perform emergency caesarean section was made and the patient was informed and consent was obtained. Blood group and cross-matching was done. In theatre vulvovaginal toilet was done and the urinary bladder was drained lOOmls of clear urine. She was cleaned and draped. Under general anaesthesia a caesarean section was done as explained in the introductory pages. Intraoperative^ twin pregnancy was diagnosed and the first twin was female extracted in cephalic presentation with a birth weight of 2500gm and Apgar score of 6/1, 7/5 and 10/10. The second twin was extracted cephalic with a

birth weight of 1800gm and Apgar score of 7/1, 9/5 and 10/10 and both babies were taken to the newborn unit. The placenta was single with one thick dividing membrane.

POSTOPERATIVE CARE

Postoperatively vital signs were recorded half hourly till the patient was fully awake the every four hours. She was put on intravenous fluids Hartman's solution to alternate with 5% dextrose 3 litres over 24hrs. She was also put on crystalline penicillin 2 mega-units six hourly, gentamicin 80mg and flagyl 500mg eight hourly. Both mother and twins progressed well and were discharged home on the 4th post-operative day to be reviewed in the postnatal clinic after six weeks.

FOLLOW-UP.

Patient decided to be seen in the Health Centre at Satellite.

DISCUSSION.

This is a case of a mother with undiagnosed twins in labor where a caesarean section is performed for a different indication.

Multiple pregnancy can occur from the simultaneous release and fertilization of two or more ova(dizygotic multiple pregnancy) or from the early division of a fertilized single ovum(monozygotic multiple pregnancy).

The incidence of twins in western countries is 1:80 pregnancies, that of triplets 1:64000 and quadruplets 1:512,000. Twins are more frequent in Asia and Africa. Nigeria has the highest incidence in the world, with an incidence of 1:22-28 deliveries. Japan has the lowest incidence of 1:155 births. In Kenyatta National hospital various authors have reported it at 1:58 and 1:46. In a study carried out by Mutungi, she found that the peak incidence of twinning was in the age group 25-29 years. Our patient falls within this age group.

Slightly more than 30% of twins are monozygotic;nearly 70% are dizygotic. Monozygotic twinning occurs in about 2.3-4 of 1000 pregnancies in all races. Rate of monozygotism is remarkably constant and is not influenced by heredity, age of mother, ovulation induction drugs, parity and other factors. The incidence of dizygotic twinning varies from 1.3 in 1000 in Japan to 49 in 1000 in western Nigeria.Dizygotism is influenced by race, heredity, maternal age and parity, maternal size, oral contraception, family history of twinning, previous multiple pregnancy and assisted reproductive techniques.

Monozygotic twinning is further divided depending on the time of division after fertilization of the ovum. If division occur before the inner cell mass(morula) is formed the outer layer of the blastocyst is not yet committed to become chorion i.e. within the first 72 hrs after fertilization; two embryo, two amnions and two chorions will develop i e diamniotic, dichorionic monozygotic twin pregnancy There may be two placentas or a single fused placenta.

If division occurs between the fourth and eighth day after the inner cell mass is formed and cells designed to become chorion have already differentiated and those of amnion have not, two embryos will develop, each in separate amnion sacs. The two amnion sacs will eventually be covered by a common chorion thus giving rise to a diamnionic, monochorionic monozygotic twin pregnancy. However if the amnion has already become established which occurs about 8 days after fertilization, division will result in two embryos with a common amniotic sac, or monoamnionic, monochorionic and

t-nonozygotic twin pregnancy. If division is initiated even later i.e. after the embryonic disc is formed, cleavage is incomplete and conjoined twins are formed.

Early diagnosis of twin pregnancy may alter perinatal mortality. Twins account for disproportionately large share of adverse pregnancy outcomes attributed to preterm delivery. Oyieke in his study found that only 25% of twins were diagnosed before 32 -weeks gestation. Diagnostic ultrasound in early pregnancy will show two gestational sacs as early as the 6th week. It however should be noted that between one third and two thirds of multiple pregnancies end in a single birth. The incidence of loss of one fetus range fro 0.5-6.8% after demonstration of multiple pregnancy. In Oyieke's study, only 54% of the patients with multiple gestation were diagnosed before labor and 38% were diagnosed after delivery of the first twin

Sometimes it is possible to identify two fetal heart tones each at different rate and clearly distinct from the maternal pulse. Biochemical tests like chorionic gonadotropin in plasma and urine, on average are higher than those found with singleton pregnancy, but not so high to allow a definite diagnosis of multiple fetuses. Alpha-fetoprotein level in maternal plasma is commonly higher in pregnancies with twins. A high index of suspicion may lead to the diagnosis of twins clinically. In multiple pregnancy the uterus is bigger than the dates with a small fetus in proportion to the uterus size. There may be increased fetal activity and multiple fetal pans may be palpated. Maternal weight gain is greater than normal. The patient presented had no ultrasound done and evidence of uterine size larger than dates was absent though the fetus was felt to be big.

Accepted indications for caesarean section in twin pregnancy are no-cephalic presentation of twin 1 (23%), intrauterine growth restriction (IUGR) in dichorionic twins (<5%), twin 2 significantly larger (>500g) than twin 1, antepartum death of 1st twin (1-2%), placenta praevia (1-2%), chronic twin to twin transfusion syndrome (TTTS) (<1%) and mono-amniotic twins (<1%).

Contentious indications for caesarean section are maternal request (5-10%), unfavorable cervix, uncomplicated monochorionic twins and previous caesarean section. The two common indications for elective caesarean section are no-vertex presentation of twin 1

£Lnd maternal request. The latter indication is increasing where a high proportion of twins is a consequence of infertility treatment in older couples.

Antenatally twin pregnancy has increased iron and folate requirement and thus predisposes to maternal anemia. Other complications include pregnancy induced hypertension polyhydramnios, placenta praevia, malpresentations, placenta abruptio, cord accidents, postpartum hemorrhage, discordant fetal growth especially in dichorionic gestation, chronic twin to twin transfusion syndrome, preterm delivery and death of co-twins.

A policy of offering all women routine ultrasound examination scan at the end of the first trimester will facilitate the diagnosis of twins, thereby allowing more accurate diagnosis of chorionicity. Screening for aneuploidy by nuchal translucency and early detection of discordant structural abnormalities are additional advantages.

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DBSTETRIC CASE No. 12

DEEP VENOUS THROMBOSIS IN PREGNANCY-LIVE BABY

"NAME:	S.K	DOA	16.09 2004
AGE:	28 YEARS	DOD	07.10.2004
IP NO.	0989061	LMP	21.01.2004
PARITY:	2+0 Gravida 3	EDD	28.10.2004

PRESENTING COMPLAINT.

The patient presented with a four day history of swelling of the left lower limb, pain in the same limb and difficulty in walking.

HISTORY OF PRESENTING COMPLAINT.

The patient was admitted through labor ward with a four day history of pain and swelling of the left lower limb. The pain was mainly in the left inner thigh and calf region. There was no history of trauma to the leg and no history of pricks to the foot .The pain was made worse by walking. She never had such a problem before. She had no chest pains or breathing problems.

OBSTETRICS AND GYNECOLOGY HISTORY.

She had her menarche at 14 years. Her cycle was regular prior to conception, every 28 to 30 days with a duration of 3 to 4 days of normal flow with no dysmenorrhoea. She didn't report any dyspareunia or pelvic pain.

S.K was a para 2+0. She first conceived in 1992, and delivered a live female infant 3000g in hospital via spontaneuous vertex delivery. Her last delivery was in February 2000, SVD, to a live male infant whose birth weight was 2800g. She had normal puerperium in both pregnancies. The two children were alive and well. Her LMP was on 21.01.2004 with an EDD of 28.10.2004 giving her a maturation by dates of 35 weeks at the time of admission. She had been on intrauterine contraceptive device between 1992 and 1999. She had never been on any hormonal contraceptive.

1?AST MEDICAL HISTORY

This was not contributory.

ANTENATAL CARE

She started attending clinic at a private clinic at 15 weeks gestation and her antenatal period was uneventful. She had a total of 5 visits to ANC prior to admission. Antenal profile was done and the results were as follows:

Haemoglobin:	10.8g/dl.
Blood Group:	A positive
HIV	Negative
VDRL:	Negative

FAMILY AND SOCIAL HISTORY

S K was a 28 year old married cashier at a local supermarket. Her husband was a security officer. She neither took alcohol nor smoked cigarettes. There was no history of chronic illness in the family.

GENERAL EXAMINATION

She was in good general condition, not pale, afebrile, not jaundiced or cyanosed with no lymophadenopathy. She had no edema.

VITAL SIGNS

Blood pressure	110/70mmHg
Respiratory rate	19 per minute
Pulse rate	76 beats per minute
Temperature	36.8 °C

ABDOMINAL EXAMINATION

'The abdomen was uniformly distended, with a fundal height corresponding to 36 weeks gestation. The lie was longitudinal, the presentation cephalic and the fetal heart tone was heard and was regular at 140 beats per minute.

LOCAL EXAMINATION OF LEFT LOWER LIMB.

The left thigh and leg was swollen with shiny skin both on the calf and upper thigh and was warm and tender to touch.The right thigh and calf were normal. The circumference of the left calf 10cm below the tibial tuberosity along the line between the tibial tuberosity and the medial malleolus was 37.0 cm compared with 34.0 cm on the right side. The thigh circumference taken at the level of 20 cm from the tibial tuberosity and the anterior superior iliac spine was 49.0 cm on the left compared with 47.0 cm on the right.

The respiratory, cardiovascular and central nervous systems were essentially normal.

DIAGNOSIS.

Deep Venous Thrombosis in pregnancy at 35 weeks gestation.

INVESTIGATION RESULTS

HEMOGRAM

Hb	11 2g/dl.
WBC	7.4 x 10/L
Differentials	Neutrophils 63%
	Monocytes 7%
	Eosinophis 2%
	Lymphocytes 28%
Platelets	350 x 10/L

COAGULATION SCREEN

18 09 2004:	PTI 14 seconds test. Control 13 seconds. Index 93%. INR 1.08.
	KCCT Test 50 seconds. Control 39 seconds
23.09.2004	KCCT Test 58 seconds. Control 38 seconds

01.10.2004 PTI Test 20 seconds. Control 15 seconds. Index 75%.

PTI Ratio 1:33

KCCT Test 60 seconds. Control 32 seconds. Ratio 1:1.9

Doppler Ultrasound. 24.09.2004

Showed left lower limb partially occluding thrombus in the calf and femoral vein. An obstetric scan was done at the same sitting and showed a single live intrauterine pregnancy at 36 weeks 4 days which was in cephalic presentation with the placenta being fiindoposterior.

MANAGEMENT

She was put on anti-inflammatory, heparin infusion and heparinisation phase was maintained for 5 days. When symptoms subsided she was put on maintenance dose of heparin titrated against the KCCT. Maintaining the value of 1.5-2.5 above the control level. The heparin infusion was maintained at 10,000 IU 8 hourly intravenously. Her clinical condition was guided by daily limb measurements and pain relief.

The patient was stable and walking well with no pain and monitoring was continued. On 05 10.2004 she developed labour pains and was transferred to labour ward. Pelvic examination revealed a cervical dilatation of 3 cm which was well effeaced. She had one contraction lasting 20 seconds in 10 minutes. The heparin was stopped. Protamine sulphate was odered and kept ready Blood for grouping and cross match and two units of freshly donated blood was kept ready for her

Labor was monitored by partogram. She progressed well and delivered a live male infant who had an Apgar score of 9/1, 10/5 and 10/10 and a birth weight of 2900 grams. Her third stage was managed actively with injection of ergometrine and uterine massage after delivery. She lost approximately 400 mis of blood. Heparin was restarted together with Warfarin 5mg daily after 24 hours post partum. Heparin was stopped on the 3rd post-operative day and she continued with Warfarin.

POST PARTUM

She continued on Warfarin 5mg once daily. Coagulation profile on 09.10.2004 showed a PTI Test of 31 seconds Control 13 seconds. She was subsequently discharged on the 5,h postpartum day to be seen in the postnatal clinic in two weeks time.

FOLLOW UP

In two weeks time she was seen in the hematology clinic and a repeat PTI was within normal range and was continued on warfarin. At 6 weeks post natal she was seen and contraception discussed and she opted for a barrier method. She was advised to continue on warfarin till her next review in the hematology clinic. The risk of recurrence of DVT in her next pregnancy was explained and she was advised on early booking and probable prophylaxis.

S

PISCLSSION

presented is a para 1+0 gravida 2 mother who developed deep venous thrombosis of the 1 eft leg, admitted and treated with heparin, analgesics and bed rest, improved, went into labor, delivered and converted to warfarin before discharge home in stable condition.

Deep venous thrombosis (DVT) is a vascular occlusive disorder caused by the formation of a pathological thrombosis in the blood vessel. Thrombus is a solid mass consisting of platelets, polymorph nuclear cells and fibrin strands with strapped red blood cells.

The risk of venous thromboembolism is five times higher among pregnant women as among non- pregnant women of similar age (1). The incidence of venous thrombosis in pregnancy varies from lin 1000 to 1 in 2000 according to different studies. Venous thrombosis can occur both antepartum and postpartum. Antenatal venous thrombosis is more common than postpartum one. .Antepartum DVT occurs more commonly in the 2nd and 3rd trimester than in the first trimester (1,2,3).

At Kenyatta National Hospital the incidence of deep venous thrombosis was reported to be 0.16%(1.6 per 1000) of all pregnancy admission (4). Out of the 50 cases analyzed by Waweru, 61% were associated with pregnancy and 76% of these thrombosis occurred in the left lower limb.

Thromboembolic diseases can be placed in three categories i.e. superficial thrombophlebitis, deep venous thrombosis and pulmonary embolism (1,2,3). Thrombophlebitis means thrombosis in a vein preceded by injury due to trauma or inflammation of the vessel wall due to infection or hypertension.Phlebothrombosis means coagulation or thrombosis occurring in veins without antecedent inflammation and may be due to increase in clotting factors and platelets or decrease in fibrinolytic activity. When solid thrombus is dislodged, embolism occurs. A thrombus can be formed in the deep veins of legs and dislodged to the lungs (pulmonary embolism). Superficial thrombophlebitis is the most common thrombosis associated with pregnancy usually in varicose veins in the calf. DVT may be a sequel of superficial venous thrombosis.

During normal pregnancy, the plasma concentration and activities of several proteins involved in blood coagulation and fibrinolysis change. These changes may promote coagulation, decrease anticoagulation and inhibit fibrinolysis and this may increase the risk of thromboembolic events especially among pregnant women who have acquired or genetic risk factors for thrombosis.

Prevention of recurrent venous thrombosis and pulmonary thromboembolism is the main reason for accurate diagnosis and adequate treatment. The pathophysiology of venous thrombosis involves three inter related factors ("Virchow's triad"): damage to the vessel wall, slowing down of blood flow and increase in blood coagulability. In pregnancy all three factors occur. Venous stasis results due to increased distensibility of the veins by mechanical obstruction of the gravid uterus and the relatively reduced mobility of pregnant women. Physiological changes in adaptation to pregnancy results in increased venous distensibility and capacity. This is worsened by the increased prevalence of women working during pregnancy at jobs that they sit for long hours. A change in clotting factors occurs in pregnancy and reaches its peak at term. Increase in factors II (Fibrinogen), V, VIII and X together with decrease in fibrinolytic activity and elevated blood levels of platelets. Blood vessel injury may occur from labor process, operative vaginal delivery and pelvic infections (1,2,3,5).

^isk factors for venous thrombosis include; Age, (>35 years), cancer, surgery, tmmobilization, prior pelvic radiation therapy, obesity, varicose veins, leg edema, diabetes mellitus, hypertensive disorders, heart disease, prolonged labor, operative cjelivery and postpartum endometritis.

|3lood group "A" has been related to increased risk to DVT (2,7). Our patient did not l-iave any ofthese factors apart from pregnancy and may be blood group A.

These conditions not only predispose apparently healthy people to thrombosis but are 1 ikely to trigger thrombosis in people with inherited thrombophillic abnormalities. Inherited thrombophillia is a genetically determined tendency to venous thromboembolism. The well established prothrombotic abnormalities are deficiencies of antithrombin III, protein C, protein S and presence of factors V-Leiden, A mutation in coagulation factor V (Arg 506-Gln) that results in the resistance to activated protein C (1,3,6). Hyper homocysteinuria is also associated with occurrence of venous thrombosis. (10).

Deep venous thrombosis (DVT) can be divided into proximal (ilio femoral) and distal (calf) Proximal DVT involves popliteal, femoral and iliac veins and forms 80% of cases. Distal type of DVT involves calf veins and forms only 20% cases. Proximal vein thrombosis is associated with higher incidences of pulmonary embolism, which occurs in 50% of patients documented with DVT. Proximal extension of calf deep venous thrombosis occurs in 30% of cases (1,2,3). The patient presented had both calf and proximal deep venous thrombosis.

Almost 90% of DVT affects left lower limb among pregnant women compared with 55% among women whom are not pregnant (3). At KNH, Waweru (4) found that DVT was three times more common on the left then the right. This difference may reflect compression of the left iliac vein by the right iliac and ovarian arteries, which cross the vein on the left side only.

DVT in pregnancy can present or be associated with low abdominal pain due to periovarian collateral circulation or thrombosis. When coupled with the mild pyrexia and leucocytosis of thromboembolism, this pain can be mistaken for other intra-abdominal disorders such as urinary tract infection or appendicitis (3). Clinical evidence of DVT of the legs precedes pulmonary embolization in only half the cases Importantly nearly 40% of asymptomatic patients with DVT were found to have concomitant pulmonary embolism (1,2). Chest discomfort, shortness of breath air hunger, tachypnoea, or obvious apprehension are signs and symptoms that should alert the physician to a strong likelihood of pulmonary embolism. The most reliable symptom is breathlessness (1,2,3).

Most patients with DVT are asymptomatic, classical features include swelling, pain, tenderness, local cyanosis, fever and a positive Homan's sign. However three quarters of patients who present with suspected signs and symptoms of DVT actually have non thrombotic causes of leg pains that include leg trauma, cellulites, obstructive lymphadenopathy, superficial vein thrombosis, post phlebitis syndrome and ruptured bakers cyst.(1,2,5).

fine presently available techniques for the objective diagnosis of DVT include contrast j-tonography, non-invasive methods and biochemical assays. Venous ultrasonographic rnaging is most widely used. Proximal veins are compressed under gentle pressure with t1-ie ultrasound transducer(compression scanning) and the inability to compress the vein \ ridicate presence of DVT. Impedance plethysmography, real time B mode ultrasonography, magnetic resonance imaging (MRI) and computed tomography (CT scan) are some of the other tests that can be done. Venography remains the standard for confirmation but has the risk of inducing thromboembolisim itself. Radioactive iodine 1 aden fibrinogen scanning and D-dimer assays can be used. Various D-dimers are formed vvhen cross-linked fibrin contrast within thrombosis is proteolysed by plasma. Various dimmer assays available include ELISA, latex agglutination assays and whole blood sgglutination test. In our patient Doppler ultrasonography and compression studies confirmed our diagnosis of DVT on the left leg (1,2,3).

In the management of venous thrombosis, the options include anticoagulation, caval filters, fibrinolytic therapy and surgical thrombectomy. The last three therapeutic approaches have been assessed less extensively and rarely used. Anticoagulation therapy is the treatment of choice for most patients with established venous thromboembolism.

In. DVT, management is divided into acute and remission phases. Main objective of therapy is to stop growth of the existing thrombus, control pain and swelling with analgesics and bed rest with elevation of the affected limb to facilitate venous return and decreased edema. Physiotherapy and mobilization is undertaken after pain subsides and prophylaxis to those at risk to stop thrombus formation (1,2 9).

Heparin is the drug of choice in pregnancy. Main problem with heparin is the cost, it is parenteral and compliance. Heparin does not cross the placenta and is not secreted in breast milk due to its high molecular weight. In acute phase, intravenous heparin infusion 30,000-40,000 IU/day is given together with ancillary measures to improve venous return and reduces patient discomfort. Supportive measures include bed rest, elevation of the affected limb, local application of heat and analgesics. Action of heparin is immediate through acceleration of action activity of antithrombin III which in turn decreases the activity of factors IX, X and XI. Heparin has half-life of 60-90 minutes. It is never given intramuscularly since it causes bleeding in muscles and also when the platelets are less than 50000/ml. Its side effects include bleeding, allergic anaphylactic reactions, thrombocytopenia and transient alopecia osteoporosis, fat necrosis at injection sites, rare but dangerous adrenal hemorrhage and necrosis. The antidote of heparin is protamine sulphate at a dose of lmg for every 100IU of heparin. Protamine sulphate itself is an anticoagulant hence its advisable not to give more than lOOmg in a short period and it also has side effects of sudden fall in blood pressure, bradycardia, transitory flushing or feeling warmth. Heparin anticoagulant effect adequacy is monitored by activated partial thromboplastin timeAPTT or KCCT, which should be 1.5 to 2 times control value (1,2,3,5)

Warfarin can also be used for anticoagulation but should be stopped at 36 weeks and patient converted back to heparin. Its action starts after 3 days through inhibition of vitamin K dependent clotting factors synthesis in the liver. Warfarin crosses the placenta and being teratogenic is contraindicated in first trimester. It causes nasal hypoplasia, ophthalmologic abnormalities, mental retardation, allopecia, urticaria and severe dermatitis apart from bleeding. After 36 weeks, warfarin crosses the placenta and might cause bleeding tendencies in the fetus especially intracranial hemorrhage apart from

ruptio placenta. Warfarin is given 5-15mg daily orally depending on its anticoagulant jrapeutic level achieved by measuring prothrombin time level range 1.5-2.5 times titrol value and prothrombin index (PTI) should be 50%. Vitamin K is the antidote in >ses of 501 Omg intravenously but the process of antidote is slow taking up to 72 hours reach maximum effect. In our patient warfarin was indicated antepartum since she was ready at 35 weeks.

>uring delivery, heparin does not cross the placenta and its effects on blood loss at slivery will depend on a number of variables including the dose, route and last time of dministration, the magnitude of incisions and lacerations, the intensity of postpartum lyometrial contraction and retraction and the presence of other coagulation defects. In eneral. therapeutic heparin therapy should be stopped during labor and delivery. If the terus is contracted and there has been negligible trauma to the genital tract, it can be estarted within several hours. Otherwise a delay of 1-2 days may be prudent. Protamine .ulphate should be put ready during delivery in case of need.

.Anticoagulation should be continued throughout peuperium preferably using oral anticoagulant warfarin. Warfarin has no significant transfer across the breast therefore its safe to use during lactation. Many patients with underlying congenital or acquired thrombophilia will require antenatal prophylaxis, the timing of which will depend on the patient's history and thrombophilia disorder. In the patient presented, DVT was managed antenatally with heparin which was stopped during labor, restarted 24 hours after delivery and discharged home on warfarin to use in puerperium (1,2,5).

Recently, released for clinical use, though most costly, is the low molecular weight heparin (clexane). The conventional heparin is 16000 Daltons but clexane is 4000 Daltons. Clexane has been shown to be safe, effective for treatment of proximal vein thrombosis in non-pregnant patients. Its safety in pregnancy has not been well assessed. Its main advantage is low risk of complications; Fewer injections per day and does not require an antidote (1,2,9).

Prevention measures include early mobilization in postoperative period and purperium together with physiotherapy (11). Oral contraceptives and cigarette smoking should be discontinued if the patient is to be detained for major surgery and provision of anticoagulants to high-risk groups. Thromboembolic disease and those with artificial valves should have prophylaxis. In women with multiple thromboembolic events during previous pregnancies antenatal prophylaxis should start at least 4 to 6 weeks in advance of the gestation at which the previous episode occurred for example in our patient who presented with DVT at 35 weeks, she needs prophylaxis in the next pregnancy from between 28-31 weeks gestation.

Deep venous thrombosis deterrent stockings may be useful in pregnancy as they prevent over distension of veins and hence prevent endothelial damage. (1, 2, 3, 5).

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j^STETRIC CASE NO. 13

HIV INFECTION IN PREGNANCY

ELECTIVE CAESAREAN SECTION-LIVE BABY.

>JAME: P W

IP NO: 0998461

>VGE: 31

F»ARITY:0-K)

DOA: 18.03 2005

DOD: 22.03.2005

LMP 01.07.2004

EDD: 08. 04.05

MBD: 38 WEEKS

PRESENTING COMPLAINT

"The patient knew she was HIV positive and was admitted at 38 weeks gestation for **elective** caesarean section.

HISTORY OF PRESENTING COMPLAINT

She was diagnosed as having HIV infection on her first antenatal visit at 14 weeks gestation by dates as part of the antenatal profile. She was healthy with no complaint. She was counseled about the need to undergo elective caesarean section in order to reduce the mother to child transmission of the virus. She accepted and was to be followed up in Kenyatta National Hospital antenatal clinic and subsequently to undergo elective caesarean section.

ANTENATAL HISTORY

She attended her antenatal clinic at the Kenyatta National Hospital from 13 weeks of gestation. As part of her antenatal profile HIV test was done and it was positive. She continued with her antenatal follow up which was uneventful. She was started on AZT (Zidovudine) at 34 weeks gestation, 300mg twice per day, until she was admitted for elective caesarean section.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was a primigravida whose last menstrual period was on 01.07.2004 with a expectated date of delivery of 08.04.2005 whose gestation by dates was 38 weeks. Her menarche was at 13 years and her menstrual cycles have been regular. She used oral contraceptives for 3 years but stopped two years prior to conceiving because she wanted to have a child.

p AST MEDICAL HISTORY

-["his was non-contributory

NAMELY AND SOCIAL HISTORY

She was a single lady, a secretary for a company in the city. She took wine occasionally tout never smoked cigarettes. There was no history of chronic illness in the family.

CENERAL EXAMINATION

She was in good general condition. She was not pale jaundiced or cyanosed. She had no edema oral thrush or lymphadenopathy.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and was moving with respiration. The fundal height corresponded to term pregnancy, the lie was longitudinal and presentation cephalic.The fetal heart tones were heard at 142 beats per minute and were regular

CENTRAL NERVOUS SYSTEM EXAMINATION

She was conscious and alert orientated in time space and person. The neck was soft, kernig's was negative. Pupils were bilaterally equal and reacting to light. There was no neurological deficit.

RESPIRATORY SYSTEM

She was not dyspnoeic, trachea was central chest expansion was normal with good air entry bilaterally. There was no crepitations or rhonchi

CARDIOVASCULAR SYSTEM

She had a pulse rate of 89 beats per minute which was regular and of good volume. Her blood pressure was 115/65 mmHg. Jugular venous pressure was not raised. The heart sounds were normal, no murmurs were heard and no thrills were felt.

infection in pregnancy at 38 weeks.

INVESTIGATIONS DONE.

Blood group	A- Positive
"S/DRL	Negative
"Hb	12.8 g/dL
HIV Test	Positive
7Na+	138 mmol/L
K^{4}-	3 .9 mmol/L
BUN	2.7 mmol/L
Creatinine	66 umol/L

MANAGEMENT

The patient was admitted into the antenatal ward and gave an informed written consent to undergo elective caesarean section. 2 units of fresh blood was grouped and crossmatched and she was to starve as from midnight.

The next morning she was taken to theatre and an elective caesarean section was done The outcome was a live male infant whose birth weight was 3200grams and an Apgar score of 8/1/ 10/10. The baby was reviewed by the paediatrician and recommended to join the mother. The baby was also given syrup Nevirapine 6mg stat.She recovered well from anaesthesia and was subsequently transferred to the antenatal ward

On the first postoperative day she was found to be stable with no palor jaundice or edema temperature was 36.8 C. abdominal examination revealed clean dressing, uterus was well contracted at 18 weeks. Bowel sounds were present and she was advised to start on oral sips and to ambulate. The breasts were not engorged and were non tender. She had opted not to breast feed and the baby was on formula feeds.

the second postoperative day she was started on light diet and continued with intravenous antibiotics.

On the third postoperative day she was on full diet and medications were changed to orals. The dressing was opened and wound was inspected and found to be clean. It was painted with betadine and covered with gauze.

On the fourth postoperative day she had no complains and was advised to put a firm bra' and to use cold compresses to avoid breast engorgement She was discharged home to be seen in the postnatal high risk clinic after 2 weeks. She was seen as scheduled and she had no complaints. The baby was also stable. She was to continue follow-up and to have work up for possible initiation of antiretrovirals.

DISCUSSION

"The patient presented was a primigravida with HIV infection who underwent elective oaesarean section to reduce the likelyhood of mother to child transmission of HIV i nfection.

Aquired Immunodeficiency Syndrome (AIDS) is caused by the HIV virus, which belongs to a group of viruses called retroviruses, due to their ability to replicate through a DNA intermediate using an enzyme called reverse transcriptase. The HIV-1 and HIV-2 are also called lentiviruses " slow" viruses due to their slowly progressive clinical effects (1,2). The patient presented was only HIV-Positive but had no other clinical features indicative of AIDS.

Over 80% of cases of HIV infection in women occur in the reproductive age group making heterosexual and perinatal transmissions important modes of transmission. It is currently estimated that about 11 million women are infected with HIV majority living in Sub-Saharan Africa (1,2). The prevalence among the antenatal mothers in Nairobi has increased from 0% in 1980, to 2% in 1985 to 13.0% in 1991.

An incidence of 6.2% among the antenatal mothers was reported at Kenyatta National Hospital in 1991 (9,10,13). In Kenya the seroprevalence of HIV infection in pregnant women exceeds 20% in many areas. (14).

Generally there are three main modes of transmission of HIV, these ar sexual intercourse (both Homo and Heterosexual contacts). Exposure to blood or bodily fluids and mother to child transmission. 90% of HIV infection in children in Kenya is due to mother to child transmission, making it a major source of HIV infection in the paediatric age group (14).

The main pathology in HIV infection is an infection of the CD4 positive cells by the HIV virus, these include, the T-Helper, lymphocytes, macrophages, central nervous system and placental cells. The dissemination of the virus leads to the lysis of the cells and therefore the depletion of the cell mediated immunity; resulting in multiple opportunistic

tections including oral pharyngeal candidiasis, Pneumocystis carinii, cryptoccocus i^oformans etc.

laternal to child transmission of HIV has been known to occur, antenatally 20-35%, during labour and delivery 65-75% in the non breastfeeding population and 40-50% through breastfeeding in the breastfeeding population (14).

X/arious methods of management of HIV infected pregnant mothers hve been devised to reduce the rate of mother to child transmission. During labor the rate of transmission is reduced by minimizing vaginal examinations, and cleaning the vulva with 0.25% solution of chlorhexidine, delaying the rupture of membranes to only about 4 hours to delivery, avoiding giving an episiotomy routinely, difficult operative vaginal deliveries e.g forceps and vacuum deliveries and invasive fetal monitoring procedures e.g fetal scalp sampling.

After delivery nasopharyngeal suction should be avoided instead wipe the baby with a dry towel and discouraging breastfeeding and encouraging formula feeds.

Before labor there should be provision of voluntary conselling and testing of HIV so that the HIV positive patients may be provided with the prophylactic antiretrovirals e.g Zidovudine and Nevirapine.

Vitamin A supplements have been shown to have some preventive aspect, when given to HIV positive pregnant mothers (1,2,3,4,5).

Elective caesarean section alone decreases the chances of mother to child transmission by up to 50%, but an emergency caesarean section after onset of labour is of no significance as far as prevention of MTCT is concerned (5). The patient presented underwent elective caesarean section, and therefore, did not require the precautionary measures taken during labour and delivery.

As noted earlier, 65 .75% of the mother to child transmission occurs during labour and deliverv. hence indicating the importance of an elective caesarean section before onset of labour During the caesarean section, precautionary measures includes avoiding milking the cord, holding the baby at the level of the mother i.e avoiding holding the baby very

Inigh, avoiding nasogastric suction of the baby unless there was meconium and encouraging early formula feeds (3,5,7). As part of preventing MTCT the infant is not breastfed and is given AZT 2mg/kg six hourly for 6 weeks and fed on formula feeds or home modified animal milk or raw animal milk.

The antiretroviral drugs hve been used successfully in reducing the rate of MTCT. Several protocols or regimes have been employed. Nevirapine 200mg is given stat at the onset of labour and after delivery the infant is given an oral dose of Nevirapine at 2mg /kg before 72 hours are over (14).

AZT (Zidovudine) is also used antenatally to reduce the rate of mother to child transmission. In the short course, the patient is given oral AZT at a dose of 300mg twice a day from 34 weeks of gestation, and the 300mg every 3 hours during labour till delivery. In the long course, AZT is given 300mg twice a day from 14-16 weeks till onset of labour, intrapartum the patient is given 2mg /kg AZT intravenously for the first hour then 1mg /kg/hour till delivery (14). In same occasions, the HAART therapy is used. The patient presented was on the short course AZT prophylaxis.

The rate of transmission is favoured by low CD4 count, high viral load, advanced AIDS, preterm delivery, urinary tract infection, Herpes Zoster, low birth weight, intrauterine growth restriction, chorioamnionitis and puerperal sepsis (1,4). Equally the effect of pregnancy on HIV is not quite clear, however, pregnancy is immunosuppressive and some authors feel there is a likelihood of development of clinical illness, while others believe there is no effect (11, 14).

After delivery, the mother should be evaluated by the physicians by checking the viral load, CD4/CD8 count for preparation for initiation of HAART Nutritional advise especially use of micronutrients and prophylaxis against tuberculosis and **Pneumocystis** carinii pneumonia is considered.

In general, in order to reduce the maternal to child transmission of HIV, the general transmission rate in the community must be reduced, by emphasizing on voluntary counseling and testing , advocating behavioural change and use of antiretrovirals.

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^ BSTETRIC CASE No. 14

C>NE PREVIOUS SCAR-SUCCESSFUL TRIAL OF SCAR-LIVE BABY.

>JAME	S.P	**LiMP:** 5.11.2003
/kGE	28 YEARS	EDD: 12.08.2004
PARITY	1+0	D.OA: 22.07.2004
IP NO	: 0987187	D O.D: 24.07.2004

PRESENTING COMPLAINTS

The patient came to labor ward with complaint of labor pains for the last 6 hours. The labor like pains increased in intensity and frequency. There was no drainage of liquor.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was a para 1+0. Her last delivery was in 1999. She underwent an emergency caesarean section due to fetal distress at Nazareth Hospital. The outcome was a live female infant who weighed 2900gm and the child was alive and well. Her last menstrual period was on 05.11.2003 and her expected date of delivery was 12. 08.2004.She was at 38 weeks gestation on admission. Her menarche was at 13 years of age and her cycles were regular every 28 days lasting 3-5 days. She had used microgynon from 1999 to 2002 and then stopped since she wanted another child.

ANTENATAL CARE

She attended a private clinic at Tigoni from 18 weeks gestation by dates and made three visits before the current admission. Her antenatal period was uneventful. At 36 weeks a clinical pelvimetry was done and deemed suitable for a trial of scar. Her antenatal profile was as follows:

Maternal height	5 feet 6 inches
Blood Group	B positive
VDRL	Negative
Haemoglobin level	12.1 g/dl
HIV Test	Negative

fAST MEDICAL HISTORY

This was not contributory

FAMILY AND SOCIAL HISTORY

She was a married businesswoman. She neither smoked cigarettes nor took alcohol. There was no chronic illness in the family.

GENERAL EXAMINATION

She was in good general condition. She had no pallor, jaundice, edema or cyanosis. She was not dehydrated. Her blood pressure was 110/75mmhg. Pulse rate was 78 per minute, respiratory rate of 19 per minute and a temperature of 36.9C.

ABDOMINAL EXAMINATION

She had an old pfannestiel scar and stria gravidarum. The fundal height was term. The fetal lie was longitudinal, with cephalic presentation and the presenting part was 3/5 up. There were palpable strong uterine contractions 3 in lOminutes each lasting 40 seconds. The fetal heart tones were heard at approximately 145 beats per minute. There was no area of tenderness on the abdomen.

PELVIC EXAMINATION.

She had normal external genitalia. The cervix was 6cm dilated and was central and well effaced. The membranes were bulging. .ARM was done and clear liquor obtained, no cord was felt The fetal head was in the left occipitoanterior position. There was no caput no moulding. The pelvis was clinically adequate.

IMPRESSION

An impression was made of a para 1+0 with one previous scar at term in active phase of labour, for trial of scar.

MANAGEMENT

The basic management of trial of scar was explained to the patient. Blood for grouping and cross-match was taken and 2 units of compatible blood was requested for and made

^.^ailable. An intravenous line was established and 500ml of 5% dextrose infusion commenced.

3he was started on the partogram where half hourly pulse, blood pressure, fetal heart rate, uterine contractions and liquor colour observations were made.2 hourly vaginal examinations and determination of the descent of the presenting part and cervical dilatation were made and also charted. Trial of scar was to be abandoned if the patient developed persistent pulse rate more than 100 per minute, fetal or maternal distress, vaginal bleeding, poor progress or obstruction of labor

Three hours later she had 4 strong contractions every 10 minutes lasting 40 seconds. The fetal heart rate was regular at 144 beats per minute. The presenting part was 1/5 above the pelvic brim. The scar was non-tender. Vaginal examination revealed the cervix to be 9cm dilated, with no caput or moulding and the liquor was clear. Partogram was continued and she was nursed in the lateral recumbent position.

She progressed well and after 4 hours of labor since time of admission, she was noted to have urge to bear down with contractions. She had a spontaneous vertex delivery to a live male infant weighing 3100 grams with an Apgar score of 9 in 1 minute and 10 in 5 minutes. Intramuscular ergometrine 0.5mg was given with the delivery of the anterior shoulder. The placenta was delivered by controlled cord traction. It weighed 600grams and was normal and complete. The lower segment was explored and found to be intact. The uterus was well contracted. The estimated blood loss was 300mls.

The patient was observed for 2 hours in labor ward and the vital signs remained stable. Thereafter she was discharged to the floors. The following day the mother and the baby were discharged home in good condition and were to be reviewed in the postnatal clinic in 6 weeks time.

POSTNATAL-CLINIC FOLLOW-UP

The patient did not show up for postnatal visit and therefore was lost to follow-up

DISCISSION

S P was a 28 year old paral+0 with one previous scar. She presented in labor and progressed to deliver by spontaneous vertex delivery to a live male infant.

Trial of scar is defined as an attempt at vaginal delivery following prior caesarean section. It is said to be successful when vaginal delivery is achieved and failed when repeat caesarean section is done (1).

Caesarean section rates have steadily increased world wide. In the USA rates of 25% in 1998 were reported with about 30% due to repeat caesarean deliveries(2). The reported incidence at KNH in the early eighties was 17.8% with 59% of the caesarean sections being repeat sections (1). A further review in 1989 showed an incidence of 21.1%(3). At Nairobi Hospital, incidence rates were as high as 28% reported in 1998. Generally the rate of vaginal delivery after caesarean section has increased from 7% in 1985 to 18% in 1994 (4).

Repeat caesarean delivery is a major contributing factor to the increase for occurrence of uterine rupture more so if the previous caesarean section was classical. Other studies have demonstrated no difference in the outcome of labor between women with and those without previous caesarean section scar with regard to rupture of the uterus. In KNH an incidence of ruptured uterus was found to be 0.22-0.52%(5,6).

For a successful trial of scar proper selection of the mother should be done. Factors that influence the success include the indication for the primary caesarean section, history of a previous vaginal delivery, number of previous caesarean sections, probability of uterine rupture and previous maternal and perinatal outcome

In our unit, a trial of scar is allowed when the following criteria inclusive of the Walton (3) criteria are met:

- Primary section should have been for non-recurrent condition
- Only one scar should be tried
- There should be no medical condition complicating the pregnancy
- There should be no history of previous uterine rupture

- The maternal pelvis should have a true conjugate of 10.5cm or more
- The pelvis must be clinically adequate
- Estimated fetal weight between 2500-3500gm.
- Previous lower uterine caesarean section.

To assess maternal pelvic dimension X-ray pelvimetry and clinical pelvimetry should be done However there is evidence that X-ray pelvimetry cannot accurately predict which women will deliver vaginally. In KNH Munyanja and Ogutu concluded that X-ray pelvimetry was not better than clinical examination and that it was only of value to prevent a mother with contracted pelvis from undergoing an emergency caesarean section following unnecessary trial of labor(7,8). In addition radiological pelvimetry is maternal focused, and is thus a static examination and does not assess the dynamic changes in the pelvis and fetal dimensions during labor. Moreover radiation exposure to the fetus possibly places the infant at risk of developing childhood leukemia(9).

In the patient presented, X-ray pelvimetry had not been done antenatally, but clinical pelvimetry was done at admission. There was no medical or surgical condition to prevent trial of scar.

In a patient who is undergoing trial of scar, a blood sample should be drawn for grouping and cross-match and 2 units of compatible blood made available. The patient must be carefully monitored and the obstetrician must be available throughout labor. Facilities and personnel must be available to perform emergency caesarean section. The patient must be appropriately counseled regarding the risks and benefits of the trial of scar.

During labor analgesia and epidural anaesthesia may encourage more vaginal births after caesarean section. In regards to it the use of analgesia has been shown that it rarely impairs ability to diagnose uterine rupture(10,11).

Less than 10% of women with scar deliveries experience pain and bleeding. The most frequent sign of uterine rupture is fetal heart deceleration. After delivery some authors recommend exploration of the lower uterine segment, others feel it is unnecessary if there

i s insignificant bleeding after delivery. The patient did not develop any of the above signs.

Induction and augmentation of labor have also been successfully done in mothers with previous scars. Oxytocin in low dose may be used, however prostaglandin's have been associated with uterine rupture (12). Induction of labor and augmentation of labor is not routinely done in our unit for a mother with a previous scar.

In the past 15 years there has been ample proof for a trial of labor in most women, after lower transverse caesarean section. Therefore encouraging vaginal birth after caesarean section would reduce the caesarean rates associated morbidity and mortality(l). The morbidity associated with caesarean section include, wound sepsis, wound dehiscence, post-operative pain, anaesthetic risk, and iatrogenic prematurity. Patients also stay longer in hospital and subsequently pay higher hospital bills(13).

Previous history of contracted pelvis, prior classical or T-shaped incision or other transftmdal uterine surgey, medical or obstetrical complications that precludes vaginal delivery and inability to perform emergency caesarean delivery because of unavailable surgeon, anaesthesia, sufficient staff or facility have been reviewed in various studies and found to be contra-indications to trial of scar (14).

Various authors have reported successful vaginal delivery after previous caesarean section deliveries. At KNH a success rate of 73 .9% was reported in 1982 (6). In a study done by Walton in 1978 (3), 74% of his study group had successful vaginal delivery, 20% had failed trial of labor due to arrest in dilatation, 5% had ruptured uteri, and 9% developed fetal distress and had caesarean section.

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OBSTETRIC CASE NO. 15

CARDIAC DISEASE IN PREGNANCY-VAGINAL DELIVERY-LIVE BABY.

:SAME	: Q.N	**LMP**	: 24.02.2004
.AGE	: 24 **YEARS**	**EDD**	: 01.12.2004
FARITY	: 0+0 G 1	**D O A**	: 30.09.2004
IP.NO	:0986556	**D.O.D**	18.11.2004

PRESENTING COMPLAINT

Two months history of cough, difficulty in breathing and leg swelling.

HISTORY OF PRESENTING COMPLAINTS

The patient was a referral from a peripheral city council clinic with a suspected diagnosis of cardiac disease in pregnancy. She had presented with two months history of cough which was productive of clear sputum. There was no haemoptysis. There was no history of fever. The cough was associated with progressive difficulty in breathing that was worse on lying down and on exertion. She also had paroxysmal nocturnal dyspnoea, easy fatiguability and palpitations. She later developed progressive swelling of both feet up to the knee joint. There was no history of facial puffiness, abdominal swelling or altered micturition or bowel habits.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a para 0+0 gravida 1 with her last menstrual period being on 24.02.2004 and an estimated date of delivery of 01.12.2004 and gestation by dates of 32 weeks. She attended Kayole city council clinic where she was referred on her first visit due to suspected heart disease. No antenatal profile was available.

Menarche was at 14 years of age. Menses were regular lasting 3 days after every 28 days. She had not used any form of contraception.

PAST MEDICAL HISTORY

She had no any other chronic illness nor previous surgery.

FAMILY AND SOCLAL HISTORY.

She was a housewife. Her husband was a casual labourer in Kayole. There was no family history of chronic illness. She neither smoked cigarettes nor took alcohol.

PHYSICAL EXAMINATION.

f5he was in fair general condition not in distress. She had mild palor with moderate fipedal pitting edema, no jaundice, cyanosis, no finger clubbing and no splinter •haemorrhages. The blood pressure was 120/80mmHg, the respiration rate was 20/minute **and** the temperature was 37C.

CARDIOVASCULAR SYSTEM

The pulse rate was 78/minute of good volume and regular Jugular venous pressure was raised There was no chest deformity. The precordium was hyperactive and the apex beat was displaced to 6^{th} intercostals space anterior axillary line. First and second heart sounds were heard. There was a pansystolic murmur heard loudest at the apex and a mid-diastolic murmur.

RESPIRATORY SYSTEM

The chest moved symmetrically with respiration. There was good air entry bilaterally. There were coarse basal crepitations.

MUSCULOSKELETAL AND NERVOUS SYSTEM.

They were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended with ftindal height corresponding to 30 weeks gestation. The lie was longitudinal, the presentation cephalic and was ballotable and 5/5 up The fetal heart tone was 148 beats per minute and was regular. There was no hepatosplenomegaly.The palpable normal liver edge was non-tender.

IMPRESSION.

Cardiac disease in pregnancy with possible valvular involvement

INVESTIGATIONS

ANTENATAL PROFILE

Blood group	0 Positive
VDRL	Negative
Hb	9.1 g/dl
WBC	9.5 x 10/L

Platelets 384 x 10/L

Urinalysis for microscopy culture and sensitivity was normal

Electrocardiogram showed normal sinus rhythm.

Echocardiogram was done and reported as follows:

Thickened mitral valve with reduced mobility of the tips

Normal aortic and pulmonary valves

Pulmonary pressures of 50-60mmHg.

The left atrium is enlarged 4cm

Borderline left ventricle size

Good left ventricular function

Ejection fraction 62%

The conclusion was predominant mitral stenosis and minimal mitral regurgitation with secondary pulmonary hypertension but good left ventricular function.

DIAGNOSIS

Cardiac disease New York Heart Association (NYHA) grade IV at 32 weeks gestation.

MANAGEMENT

The patient was initially admitted to labor ward for stabilization. She was put on bed rest in a propped up position. She was given oxygen by mask and started on digoxin 0.25mg once daily, lasix 40mg once daily and haematinics. She was also transfused 1 unit of packed cells. She was then transferred to antenatal wards to continue with the treatment. While in the ward she was on bed rest in a propped up position and was given oxygen as required. She was reviewed by the cardiology team and an electrocardiogram and an echocardiogram were done.

INVESTIGATIONS.

Haemogram

Hb 9.2 g/dl

WBC 94 x 10/L

Platelets 336 x 10/L

Subsequent haemograms were within normal range.

(_Jrea and Electrolytes

BUN	3.1 mmol/L
Na +	138 mmol/L
K +	4.2 mmol/L
Creatinine	68 mmol/L

Subsequent urea and electrolytes were normal.

Urinalysis

Proteins	Nil
Glucose	Nil
Nitrites	Nil

No growth obtained.

The patient was informed of need for hospitalization until delivery and then until stable. Her hospital stay was uneventful.

While in the ward her pulse rate, blood pressure, respiratory rate and temperature were monitored at least four times daily while the chest was auscultated daily for basal crepitations and any changing murmurs. She had weekly haemogram, urinalysis and urea and electrolytes and serum creatinine. On 4.11.2004 four weeks post admission at a gestation of 37 weeks she experienced labor pains. No drainage of liquor or urinary symptoms was noted. She was transferred to labor ward.

MANAGEMENT IN LABOR WARD

She was assessed in labor ward and found to be in good general condition. Her temperature was 37.0 C, blood pressure 115/75mmhg, pulse 90 per minute and regular, respiration 20 per minute. The chest had mild bilateral basal crepitations there were no new murmurs. The fundal height was at 36 weeks with palpable moderate contractions 3/10 minutes each lasting 20 seconds. The cephalic presentation was 4/5 up. The cervix was 3cm dilated, well effaced and the membranes were intact. The pelvis was clinically adequate.

She was propped up and given oxygen by mask. She was given Tramal lOOmg intramuscularly for analgesia, buscopan 40mg intramuscularly, and started on

prophylactic antibiotics, crystalline penicillin 2 mega units and gentamicin 80mg intravenously. An emergency tray containing aminophylline, digoxin,fiusemide,sodium t? icarbonate and calcium gluconate was prepared and placed nearby. A vacuum extractor vvas also kept in readiness for use during second stage.

.A partogram was started with Vi hourly maternal vital signs observations. ARM was performed at 6cm cervical dilatation and clear liquor was obtained. The patient progressed well in labor and 7 hours later from time of admission she was fully dilated and transferred to the delivery room. She was placed in semi-fowler position with legs supported by stirrups. Vulvovaginal toilet and aseptic catheterization was done and clear urine obtained. Local anaesthesia(8ml of lignocaine 2%) was infiltrated into the left side of the perineum. A left mediolateral episiotomy was performed. The medium (50mm) ventouse cap was applied to the fetal vertex and gently a vacuum was created (Increased at a rate of 0.1 mm/kg every minute until a maximum of 07mm/kg); by gentle traction with the first uterine contraction, the baby was easily delivered, a live female infant who weighed 2950 grams and an Apgar score of 8 in 1 minute, 9 in 5 minutes and 10 in 10 minutes. No fetal anomalies were noted.

She was given a bolus of 80mg intravenous frusemide immediately after delivery. The placenta and membranes were delivered completely. Clots were expelled and uterine massage yielded a well contracted uterus hence no need for oxytocin. The estimated total blood loss was 350mls. The episiotomy was repaired in layers after inspection revealed no cervical or vaginal tears. Post delivery vital signs were normal.

POSTPARTUM PERIOD

In the first 24 hours the vital signs were noted Vi hourly while the patient was in labor ward. They were found to be normal and the patient was transferred to the post-natal ward. She was given digoxin 0.25mg once daily, lasix 40mg once daily, Ampiclox 500mg three times a day and Ranferon lOmls once daily. She did well postnatally and was breast feeding.. She was discharged home on above treatment after a fortnight of observation and was counseled on contraceptive methods and need to plan on completing her reproductive life early. She was to be seen in cardiac and postnatal clinic in two weeks.

f OSTNATAL VISIT

3 he had no complaints and the baby was fine and breast feeding. She was not in failure •asA. the uterus was well involuted. She was continued on lasix and digoxin and referred to i he cardiac clinic. For family planning she chose to use progesterone only oral contraceptive pills (microlut) and was referred to the family planning and welfare clinic.

^ISCUSSION.

I"tie patient presented was a 24 year old primigravida with newly diagnosed mitral v-3lvular disease at 32 weeks gestation. She was managed in the ward until she delivered a live baby vaginally.

Extensive haemodynamic changes occur in pregnancy, which are normally well tolerated fc>y healthy women. In contrast this adaptations pose a challenge to those with cardiac disease placing them at risk of disability and even death.(1,2).

Cardiovascular disease is the most important non-obstetrical cause of disability and death in pregnancy and it complicates 1-2% of all pregnancies(l, 3, 4). Sequiera and Ojiambo in 1969, at Kenyatta National Hospital found an incidence of 0.5% with 95% of cases being due to Rheumatic Heart Disease(RHD). Thirty five percent(35%) of RHD cases had mitral stenosis (5). In a later study Ngotho (1982) reported an incidence of 0.99% again with 86.4% due to Rheumatic Heart Disease and 12.9% congenital heart disease(5). These results are similar to other studies from the African region where Rheumatic Heart Disease predominates(6). Other rare causes of cardiovascular disease are hypertensive heart disease, coronary, thyroid, syphilitic, idiopathic cardiomyopathy, cor pulmonale, constrictive pericarditis, various forms of heart block and isolated myocarditis(1).

Rheumatic heart disease is the commonest heart disease in pregnancy in our set-up in contrast to the developed world where congenital heart disease predominates(7). However, due to prompt and effective treatment of sore throat caused by streptococcal infection, the incidence of RHD is declining worldwide. In addition with improving medical services and advancement in cardiac surgery some women with congenital heart abnormalities will not only survive to reach the age of childbearing but also carry a pregnancy to term successfully(5,8,9).

Bhatt (1978) found that the majority of his cases had combined mitral valve disease(8) The dominant lesion in RHD is mitral stenosis 90%. The rest are mitral regurgitation 6 6%, aortic regurgitation 2.5% and aortic stenosis 1%. The most common lesions in congenital heart disease are atrial septal defects (ASD) and patent ductus arteriosus (PDA), accounting for 50% of cases. Ventricular septal defects (VSD), Tetralogy of Fallot and pulmonary stenosis (PS) together account for 20%(8,9).

pregnancy increases the difficulty of recognizing and defining heart disease. The rise in -^rdiac out-put and associated vasodilatation cause changes in the circulation that may r-nimic heart disease(8). The symptoms and signs associated with changes include, easy t~3-tiguability, chest discomfort, dyspnea, orthopnea, palpitations, peripheral edema and syncope.(3,10).However the indicators of heart disease during pregnancy are symptoms o f severe progressive dyspnoea, progressive orthopnea, paroxysmal nocturnal dyspnoea. Haemoptysis, syncope with exertion and chest pain related to effort or emotion.. In addition clinical finding of cyanosis, finger clubbing, persistent neck vein distension, a systolic murmur greater than grade III/IV in intensity, diastolic murmur, cardiomegally, sustained arrythmia, any signs of pulmonary hypertension and fixed split second heart sound. (1, 10).

The management of heart disease in pregnancy is dictated by functional capacity of the heart and special emphasis should be placed on prevention and early detection of heart failure. The severity of heart disease is usually graded according to the New York Heart Association (NYHA) classification. The grading is of prognostic significance. This clinical grading depends on cardiac response to physical activity with no relationship to the extent of the heart lesion. The grades are useful in the management protocol of patients(l,3).

Grade I	Uncompromised. No symptoms limiting ordinary physical activity.
Grade II	Slightly compromised. Slight limitation with mild to moderate Activity. No symptoms at rest.
Grade III	Markedly compromised. Marked limitation with less than ordinary activity. They get excessive fatigue, palpitations, dyspnea or angina on minimal activity.
Grade IV	Symptoms at rest or with minimal activity. Symptoms of frank congestive cardiac failure. Patients with previous heart surgery.

The disease grade is not fixed and may change suddenly. The management calls for team approach involving obstetrician, cardiologist and anaesthesiologist.

The American College of Obstetricians has developed heart disease classification based on the risk of death during pregnancy. The aim was to aid in counseling the woman as regards conception or continuation of pregnancy. (1, 11).

GROUP I

Includes conditions that with proper management, were associated with negligible maternal mortality<l%. They include ASD, VSD, PDA, pulmonary or tricuspid valve disease, corrected tetralogy of fallot, biosynthetic valves, mitral stenosis, NYHA classes I and II and Marfans syndrome with normal aorta.

GROUP II

These include conditions that carry a maternal mortality risk of 5-15%(moderate risk). They include mitral stenosis with atrial fibrillation, NYHA classes **III** and IV, artificial valve, aortic stenosis, uncorrected tetralogy of Fallot, uncomplicated coarctation of the aorta and previous myocardial infarction.

GROUP III

These have a major risk of complications and maternal mortality of 25-50%. In this group, prevention or interruption of pregnancy is generally recommended. They include, pulmonary hypertension, complicated coarctation of the aorta and Marian's syndrome with aortic involvement.

According to this classification the patient presented was in group II as she had mitral stenosis and NYHA class IV. She presented late in pregnancy at 35 weeks. It is noteworthy that maternal mortality increases with maternal age and the mortality is more likely in those cardiac conditions where pulmonary blood flow cannot be increased as in mitral stenosis. Eisenmenger syndrome, and primary pulmonary hypertension.

NYHA grade I and II patients are managed as outpatient after initial clinical evaluation. They are seen frequently by both the cardiologist and obstetrician as their grades may

jnange to higher grades and present with complications. At 36 weeks they are admitted to l-i«e ward to await delivery. Grades IH and IV patients are usually confined in the wards i-ntil delivery. (1, 3, 12, 13).

Restriction of maternal physical activity tends to avoid cardiovascular compromise and t improves utero-placental perfusion (14). The supine position should be avoided as pressure on the inferior vena cava reduces venous return. Excessive weight gain should prevented when possible. Haematinics are recommended for the prophylaxis of anaemia or its vigorous treatment when it occurs. Patients with prosthetic valves will require anticoagulation.

Respiratory infections must be treated with antibiotics and oxygen liberally given if respiratory difficulties develop. Persistent basal rales accompanied by cough are signs of onset of failure.

It is imperative to await the spontaneous onset of labor since induction is associated with significant haemodynamic changes (abnormal fluid retention should be prevented when possible) that could precipitate cardiac failure and in case of failed induction caesarean section carries an added risk of pneumonia, infective endocarditis and pulmonary edema and embolism (12). However caesarean section should be performed if there is an obstetric indication (6).

Relief from pain and apprehension without undue depression of the infant or mother is especially important during labor and delivery. Epidural anaesthesia and narcotic analgesics are preferable. The mother should be kept in a semi-recumbent with a lateral tilt position in bed and oxygen given by mask if need be. The patient should be started on parenteral antibiotics to prevent subacute bacterial endocarditis. As for grade III and IV patients, digoxin and frusemide are administered. Monitoring of vital signs, auscultation of lung bases are important to detect signs of congestive cardiac failure. Our patient was given tramadol for analgesia.

Increase in pulse rate above 100/minute and respiration rate above 24 per minute particularly when associated with dyspnea, may suggest impending ventricular failure.

[intrapartum heart failure is treated with morphine, oxygen and frusemide. In patients with prosthetic valves on heparin, it is stopped during labor.(l, 8, 15)

-X tray containing aminopylline, digoxin, morphine, sodium bicarbonate and frusemide is *-cept ready for use if need arises. Vaginal delivery should be aimed at with shortening of second stage by use of elective vacuum extraction or obstetrical forceps to decrease maternal strain. The patient should not bear down forcibly as this may increase the -venous pressure. Active management of third stage with ergometrine should be avoided and syntocinon used early if bleeding is excessive otherwise uterine massage is encouraged to minimize drug usage.

A bolus of frusemide 40-100mg is given late in third stage to offset the anticipated cardiac out-put increase from the sudden mobilization of extra vascular fluid and sudden release of blood from the lower extremities as the gravid uterus is emptied and vena-caval compression is released. The patient was given 80mg of frusemide immediately after delivery.

Close obstetric and medical surveillance must continue particularly during the first 24-48 hours, the most dangerous time for CCF, pulmonary edema and infection guarded against especially infective endocarditis. Early ambulation is necessary to prevent deep venous thrombosis and the attended risk of pulmonary embolism. A period of 10 to 14 days post natal observation and antibiotics is recommended (12). Anticoagulation is resumed 6 hours post vaginal delivery or 24 hours post abdominal delivery in patients with prosthetic valves.

Before discharge contraception should be discussed with the patient. Barrier methods and progesterone only pill is advisable for those who desire another child or do not opt for tubal ligation (1,3,13). The patient was encouraged to complete her family size early before the onset of age induced deterioration in cardiac function. When the family size is complete tubal ligation is the optimal choice. Alternatively vasectomy can be offered to the spouse if desired. Our patient opted for progesterone only pills since she had not attained desired family. She was also followed up in the cardiology clinic.

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PREGNANCY OUTCOMES IN MOTHERS WITH ADVANCED HUMAN IMMUNODEFICIENCY VIRUS DISEASE.

NUMMARY

Objective

To determine the impact of advanced HIV disease on immediate maternal and fetal outcomes.

Design

Prospective cohort study

Setting

Kenyatta National Hospital, Nairobi, Kenya, between September 2004 and April 2005 inclusive.

Subjects

Cases comprising of 68 pregnant mothers with advanced HIV disease (WHO Clinical stage 3 and 4) and controls comprising of 68 pregnant mothers who are HIV negative.

Results

Mothers with advanced HIV disease had statistically significant more anemia (55% Vs. 16%), sexually transmitted diseases (56% Vs. 15%), chorioamnionitis (14.8% Vs. 2%), premature rupture of membranes (31% Vs.9%), puerperal pyrexia (16% Vs.2%), maternal mortality (5% Vs. 0%) than HIV negative mothers.

Meconium staining of liquor occurred in 32% of mothers with advanced HIV disease and 21% in seronegative mothers. This difference was not statistically significant. Caesarean section rates were lower in mothers with advanced HIV disease(10%) compared to their seronegative counterparts (42%). This difference was statistically significant ($p<0.001$).

The mean gestational age at delivery was lower in mothers with advanced HIV disease compared to the seronegative controls and this difference was statistically significant ($p<0.001$).

Infants born to mothers with advanced HIV disease had significant lower birth weights (58% Vs. 21%) than their seronegative counterparts($p<0.001$). There were more stillbirths in mothers with advanced HIV disease than in the seronegative group (4% Vs 2%) but this difference was not statistically significant. Lower Apgar scores were recorded in infants born to mothers with advanced HIV disease and this difference was statistically significant ($p<0.003$).

External congenital anomalies were similar in the two groups (5 9% Vs.5.9%). Neonatal sepsis occurred in 11.8% in infants born to mothers with advanced HIV disease compared to 4% in infants born to HIV negative mothers. This difference achieved statistical significance($p<0.003$). Early neonatal deaths occurred statistically significantly more in infants of mothers with advanced HIV disease than infants of the seronegative group (20.1% Vs. 7.3%. $p<0.025$).

Conclusion

This combined data indicates that pregnancies complicated by advanced HIV disease tiave significant adverse outcomes, both maternal and fetal. There is increased risk of •both maternal and fetal mortality and morbidity.Pregnant mothers with advanced HIV disease have poorer fetal and maternal outcomes compared to their HIV negative counterparts. Statistical detectable biologic effects upon pregnancy occurs in mothers with advanced HIV disease.

Recommendations.

Pregnant mothers with advanced HIV disease should be managed as high risk. Aggressive antenatal maternal and fetal surveillance should be employed Labour and delivery should be closely monitored and should ideally occur in controlled enviroment where high risk maternal and neonatal care is readily available.

ABBREVIATIONS

AIDS	Acquired Immunodeficiency Syndrome
HAART	Highly Active Antiretroviral Therapy
Hb	Hemoglobin
rav	Human Immunodeficiency Virus
MSL	Meconium staining of liquor
MTCT	Maternal To Child Transmission
PPROM	Preterm premature rupture of membranes
PROM	Premature rupture of membranes
PPCP	Presumptive **Pneumocystis** carinii pneumonia
RDS	Respiratory distress syndrome
SPSS	Statistical package for social sciences
SVD	Spontaneous vertex delivery
VDRL	Venereal Disease Research Laboratory
WHO	World Health Organization

DEFINITIONS

-Advanced HIV disease,	Pregnant mothers in WHO clinical stage 3 or stage 4.
prematurity:	Birth before 37 completed weeks
I_ow birth weight:	Birth weight less than 2500 grams.
Early neonatal death:	Infant death within one week of birth.
Still birth:	Baby born dead
Low Agpgar Asphyxia:	5 minute Apgar score < 4.

INTRODUCTION

l-Tuman immunodeficiency virus (HIV) is the cause of the condition called acquired i mmunodeficiency syndrome (AIDS). AIDS was first recognized in the United States in 1980-1981 when homosexual men were found to have unusual infections and tumors suggesting an underlying deficiency in their cell mediated immunity. HIV was shown to toe the cause of AIDS in 1983-1984. Although evidence exists to show that HIV has been present in humans for 15-20 years, the exact origin of the virus is not yet known. HIV belongs to the family of viruses called retroviruses and subfamily lentiviruses. Retroviruses are single stranded RNA viruses that contain the enzyme reverse transcriptase. This enzyme is an RNA-directed DNA-polymerase that enables the RNA virus to produce a DNA copy of itself in order to become integrated and replicate in host cells.

HIV causes a chronic infection that leads to profound immunosuppression. The hallmark of this process is the depletion of CD4+ lymphocytes, and this predisposes the patient to develop a variety of opportunistic infections and certain neoplasm's. There are various clinical classifications and staging of HIV infection. The WHO clinical classification (see appendix 1) is widely used in Sub-Saharan Africa.

HIV infection has changed the face of reproductive health especially in the developing countries. Scientists are still trying to elucidate the effects of HIV infection on pregnancy and the effects of pregnancy on a HIV infected mother. There is also the issue of mother to child transmission and efforts to interrupt such transmission.

This study attempted to describe the effects of advanced HIV disease (WHO Stage 3 and 4) on pregnancy outcome. It was a case control study involving 68 mothers with advanced HIV disease and 68 mothers who were HIV negative. The mothers were matched for age, parity and gestation. The mothers were selected using a strict criteria and were followed up until delivery and 1 week thereafter or when the final fetal and maternal outcomes were determined. It is hoped that the findings of this study will add to the knowledge of the interaction between advanced HIV infection and pregnancy and to assist in the formulation of guidelines in the management of pregnant women with advanced HIV disease.

LITERATURE REVIEW

Differences exist in results of studies on pregnancy outcomes in HIV infected women from Africa compared to those from Europe and the USA. Whereas most studies in Africa have shown adverse outcomes, most western studies have shown little or no demonstrable impact of HIV infection in pregnancy both on maternal and neonatal outcomes (2).

Majority of these studies were conducted on asymptomatic HIV infected pregnant women. Few studies have been conducted on pregnant women with advanced HIV disease. Most studies concentrating on pregnant women with advanced HIV disease have been conducted in Europe and USA though the samples have been very small.

A'number of reasons could explain the differences in the results. The published work from the developed world has included very small numbers of pregnancies and majority of the studies hence lack statistical power to detect a true difference in birth weight or pregnancy-complications. In African studies control subjects are often loosely matched, for example because they delivered on the same day, were matched for age and parity alone, or were simply matched with all other pregnancies. This makes it difficult to attribute differences only to HIV status.

Furthermore there exists statistically significant differences in pregnant HIV infected women and seronegative pregnant controls which might be relevant. This includes age. unmarried status, pnmipanty, number of sexual partners, prostitution, alcohol consumption during pregnancy, gonorrheal and chlamydial infections, positive syphilis serology and travel to other African countries.

It is thus clear that control groups differ from cases, not only in HIV status, but also in ways, which are classically recognized as likely to affect birth weight or pregnancy outcome. In

several studies, attempts have been made using linear logistic regression to allow for these differences but not all potential confounders can be included, and residual features distinguishing HIV-infected women are likely to remain to confound the analysis.

Many African women have entered pregnancy in a more vulnerable nutritional state, so that any effect of HIV is more apparent, and HIV disease in Africa may be more likely to have a weight-loosing pattern. The load of other infectious diseases in Africa may contribute to the differences in study results. Malaria is endemic in several of the countries where HIV has been studied. If HIV infection resulted in a higher malarial parasite load, this could explain some of the association with adverse pregnancy outcomes, as this organism is known to be associated with increased rates of prematurity, low birth weight and neonatal death (3).

As opposed to the developed world studies, the African studies included a larger percentage of women with symptomatic HIV disease hence the possibility of an increase in adverse outcomes. In one African study 18% of the pregnant women had .AIDS **(4).** while women who reported symptoms, a non-specific guide to illness, comprised 53% and 17% in other studies (5,6).

HIV has been associated with increased pregnancy wastage in sub Saharan Africa In theory pregnancv wastage could result from various explanations. Interference with the fetal-maternal immune relationship could lead to early fetal loss. It has been postulated that early viral infection of the fetus could lead to pregnancy loss, intrauterine growth restnction and congenital anomalies. Maternal immunodeficiency could predispose to chonoaminionitis. with associated risk of preterm labour and stillbirths. It is possible that the fetus could also be damaged by recurrence of other infections such as cytomegalovirus or toxoplasmosis. Poor nutritional status with advancing disease together with the increased load of concurrent infections could impair the woman's ability to maintain her pregnancy successfully.

One of the earliest studies to report an increase in spontaneous abortion in HIV infected pregnant women concluded that this was probably due to ascertainment bias (7) More western data continue to show a higher rate of spontaneous abortion in HIV-seropositive women, but the difference is small and is no longer statistically significant. A small study in New York did not show any increase in spontaneous abortion in HIV infected women (8).

However these findings were in contrast with the findings of Langston and co workers who studied the outcomes of pregnancies in 124 HIV infected pregnant women over a 4 year period. The majority of the women were asymptomatic with only two having been diagnosed with .AIDS. There were 14 fetal losses observed (a rate of 11%) in this cohort of women. The losses occurred between 8 and 32 weeks of pregnancy with half of these experienced by 20 weeks gestation. HIV testing of fetal tissues revealed presence of HIV nucleic acid in 7 of the 14 fetuses, with all of the infected fetuses demonstrating thymus gland abnormalities. The results of this study suggest that HIV infection of the fetus occurs early in gestation and can be toxic and result in a higher fetal loss rate (9).

Africa studies on very large numbers of HIV infected women have shown a higher proportion of pregnancies ending in spontaneous abortion. In a large study from Malawi. 6605 women w ere tested for HIV and a history of spontaneous abortion was reported more often by HIV-seropositive than seronegative women (15% vs. 7%) (10).

In one of the largest studies to focus on spontaneous abortion, Temmerman and colleagues tested 195 women admitted to a hospital in Nairobi Kenya, with abortion. Controls were selected from antenatal clinic attendants. Spontaneous abortion was independently associated with HIV antibody status (odds ratio 2.2). maternal syphilis seroreactivity (odds ratio 4.3) and vaginal colonization with group B b-haemolytic streptococcus (11).

>\s with all other studies addressing this issue there were methodological problems The abortion and control groups were strictly not comparable and selection for the control group had potential for bias. Syphilis correlated with HIV, and may have been responsible for the spontaneous abortion. However attempts were made to account for this and other variables and HIV still retained a statistical significance.

Similarly a history of spontaneous abortion was the endpoint of studies in Malawi and Rwanda Such a history was more common in women infected with HIV. but neither HIV status nor syphilis serology was known for the time at which spontaneous abortion occurred **(12,13).**

What theoretical basis might there be for a relationship between HIV infection and spontaneous abortion" Maternal illness itself could be responsible, but direct viral infection is also a possibility. First trimester trophoblast tissue can be infected with HIV in vitro (14,15). Lewis and co workers claim that villous trophoblast stained for HIV-1 in pregnancies of 8 weeks gestational age (16).

One plausible mechanism for early spontaneous abortion might be changes in decidual immune cells, which could affect implantation and subsequent trophoblast proliferation. Studies on endometrial histochemistry might shed further light in this area.

It is uncertain whether HIV is a direct cause of spontaneous abortion. Most published information suggests a trend in that direction, but the studies are either retrospective or there is doubt whether the findings could be explained by a correlated variable such as positive sy philis serology or other infectious disease. On theoretical and observational grounds, an increased rate of abortion seems plausible but the increase is likely to be small.

In published controlled studies in Europe and USA, HIV infection has not been associated with a difference in birth weight (7,8,17). One study using multivariate analysis, suggested there might be a small, but statistically significant decrease in birth weight associated with maternal HIV infection (18).

In contrast to the developed world, nearly all-African studies seem to show decreased birth weight in pregnancies where the mother is HIV-infected. Temmermann and colleagues found an association between HIV-1 infection and low birth weight (mean birth weight 2913g Vs. 3072g; p=0.003) in a cohort of infants of 315 seropositive and 311 seronegative women followed prospectively through 6 weeks postpartum **(19).** A pregnancy and HIV study group in Kigali Rwanda also reported low birth weights of infants of serpositive mothers compared to seronegative controls (25.5% vs. 14.8%) **(20).** Similarly a report in Zaire noted infants of seropositive women were more likely to have lower birth weights **(21).** However. Taha T E.. Dallabeta G.A. and colleagues in Malawi did not find a significant difference in the mean birth weight between infants of sero-positive mothers and infants of sero-negative controls (22).

There is some evidence that the decrease in fetal size is related to the stage of maternal disease **(23).** An important and consistent observation is that birth weight seems unrelated to the infant's eventual HIV status **(4,24,25)** Anticardiolipin antibodies have been thought to be responsible for the decrease in birth weight in HIV infected pregnant women. Raised levels of circulating anticardiolipin antibodies have been found in women infected with HIV. independent of clinical status **(26,27).** Raised levels have also been shown in low risk obstetric populations, to be associated with adverse pregnancy outcomes, including pre-term deliver, and intrauterine growth restriction **(28,29)**

Prematurity and intrauterine growth restriction are major determinants of child survival. Early uncontrolled studies had reported a high incidence of preterm labour and intrauterine growth restriction in HIV infected women **(30,31).** The first controlled study showed both case and control groups to have a high incidence of preterm labour and intrauterine growth restnction (7). However there were no differences according to HIV serostatus. Several subsequent western studies did not show differences in pregnancy complications and outcomes as far as prematurity and intrauterine growth restriction is concerned. However because of small numbers, all these studies lacked statistical power **(8,17,32,33).** Several large studies in Africa yielded conflicting results. Some showed HIV infection to be associated with preterm delivery and intrauterine growth restriction **(4,23,34).** and other studies did not **(6,13,2135).**

Temmermann and co workers conducted a large case control study on adverse pregnancy outcome in Nairobi. 372 women who delivered a pre-term baby. 324 who delivered a baby small for gestational age. 120 who had an intrauterine death, and 69 with an intrapartum death, were compared for HIV status with 711 controls. HIV seropositivity was more common in the case groups but so were other potentially confounding features, such as pnmiparitv. lack of antenatal clinic attendance and maternal syphilis infection. However linear logistic regression retained HIV status as a statistically significant associated outcome with modestly increased odds ratios of 2.1 (pre-term birth), 2.3 (small for gestational age), 27 (intrautenne death) and 2.9 (Intrapartum death) **(36).**

HIV infection might influence pregnancy outcome due to an increased rate of chorioamnionitis. This was recently reported in one large controlled study, where pregnancies form 475 serpositive women were compared with those of 615 seronegative women matched for age and parity. Not only was chorioamnionitis more common in the former group, but also babies were more frequently premature and the neonatal death rate

•was increased (4). Bulfamante and colleagues described a higher incidence of chorioamnionitis, ophalitis, villous immaturity and plasma cells infiltrates in 91 placentas from HIV serpositive women (37).

In a further study Nyong'o go and coworkers reported on 638 mothers delivering preterm and 862 term controls. Maternal HIV seropositivity was associated with prematurity (odds ratio 1.9). In a subset where the placenta was examined, moderate to severe chorioamnionitis was found in 20% of preterm and 8% of term placentas (odds ratio 3.4). Within preterm deliveries the placentas showed chonoamnionitis in 37% of cases where the woman was HIV positive and 18% of matched seronegative cases. In addition to preterm delivery, chorioamnionitis in association with amniotic fluid infection could account for a higher rate of stillbirths (38).

Nearly all AIDS defining opportunistic infections have been reported in pregnancy, including tuberculosis, esophageal. bronchial and pulmonary candidiasis. disseminated cytomegalovirus infection, cryptococcal meningitis and disseminated histoplasmosis. Pneumocvstis carinii pneumonia stands out as the most frequent HIV related infection in pregnancy in the developed countries (39, 40, 41).

Bongain and colleagues in France reported two cases of fatal **Pneumocystis** carinii pneumonia in the third tnmester of **pregnancy** in patients who were asymptomatic at the beginning of the gestation (42). A large survey **covering** more than 1000 HIV-infected women who delivered in 1989 in the USA confirmed that **Pneumocystis** carinii pneumonia is the most frequent opportunistic infection in seropositive pregnant women in North America, as in France. Pneumocyctis carinii was also found to be the leading cause of pregnancy related deaths from AIDS in the USA (43).

Outcome for pregnant women with **Pneumocystis** carinii pneumonia appeared worse than **those reported** at the time for non-pregnant women Among 16 women who died of **Pneumocystis** carinii pneumonia 4 died during pregnancy, 6 within **a** week **after** delivery, 2 **within a month,** and 8 within a year after delivery. The mean interval between diagnosis and **death** was 59 **days** (SD +/- 42 days) compared to a survival of 187 days (+/- 208 days) in a CDC **reference** population of 190 non-pregnant women of reproductive age, matched for **intrav enous drug** use. These retrospective data suggest that pregnancy worsens the prognosis **of Pneumocystis** carinii pneumonia **(44,45).**

In a New York cohort. Minkoff and co workers reported an increased incidence of serious infections in 9 out of 16 HIV seropositive pregnant women with CD4 counts of less than 300 cells/mm. These infections included 6 women with Pneumocystis carinii peneumonia I woman with central nervous system toxoplasmosis, 1 woman with bacterial pneumonia and 1 woman with post casesarean pelvic abscess **(46).** These findings were in agreement with a subsequent report he published on HIV seropositive women with **Pneumocystis** carinii pneumonia **(40).**

It seems Iikelv that there are no major differences attributable to HIV infection in labour and delivery outcome. In immunocompromised women it is plausible that premature rupture of membranes may occur more frequently and in such women the risk of chorioamnionitis may be increased. In the Edinburgh cohort of women exposed to the risk of HIV infection, meconium staining of the liquour was more common compared with neighbourhood controls but was **equally** common in HIV seropositive and risk exposed seronegative women. The rates of induction of labour, use of epidural anaesthesia or oxytocin, assisted vaginal delivery, episiotomv and caesarean section were not related to serostatus and were similar to neighbourhood controls and the general hospital populations (8).

Temmermann and colleagues did not find any significant difference in apgar scores between infants of seropositive and seronegative mothers **(19).** though Mmiro in Uganda found that infants of seropositive mothers tended to have lower apgnar scores (23). Differences in caesarean section rates in seronegative and seropositive HIV infected women have been a subject of various studies. In one controlled study. Selwvn and colleagues found 36% of 25HIV-infected women had caesarean section as opposed to 14% of seronegative women, a result, despite the very small numbers, which approached statistical significance (p=0.06) (8).

In contradistinction Minkoff and co workers found no excess in caesarean sections in their larger prospective study (12% compared with 18% for seronegative controls) (17). In two large studies of vertical transmission, The Italian Multicentre Trial 1988 and The European Collaborative study 1992, Caesarean section rate among HIV infected pregnant mothers was found to be high. 23% and 26% respectively **(24,25).** However both studies were uncontrolled and the reason for caesarean section was not given. Reluctance to do fetal scalp sampling or to apply scalp electrodes could have been one of the reasons for high caesarean section rates.

Whether maternal mortality is increased in pregnant women with AIDS is a question yet to be answered. Sub Saharan African studies have shown a higher maternal mortality in pregnant women with adv anced HIV disease **(23).** Similarly the risk of postpartum wound infection and postpartum endometritis may be greater One study in Rwanda found that HIV seropositive mothers were more likely to have postpartum haemorrhage **(20)** However Mmiro and co workers in Uganda studying a cohort of 564 serpositive and 709 seronegative mothers found that the absolute risk of a complication to an individual infected woman was quite low; in particular there was no evidence of a clinically significant increase of a prenatal or perinatal sepsis or haemorrhage **(23).**

A dysmorphic syndrome associated with HIV has been reported. This syndrome included growth failure, microcephaly, flattened nasal bridge, oblique eyes, prominent forehead, triangular philthrum and patulous lips (47). Subsequent reports have not confirmed this findings and HIV dysmorphic syndrome was not seen in the large European collaborative study (28.48,49).

At present it seems doubtful that there is a specific syndrome and the original observations may have been due to confounding factors such as drug and alcohol use or ethnic group VIost controlled studies have not specifically reported on structural congenital anomalies Braddick and co workers and lepage and associates did not find any association between congenital anomaly and HIV serostatus (6, 13). Prospective studies, though uncontrolled, have reported on over 1000 babies with more than 18 months followup and have not emphasized unusually high incidences of congenital abnormality. Together with the belief that very early intrauterine infection is uncommon, this makes it unlikely that there exists a HIV associated embryopathy (24,25,50).

Many authorities believe that the more the advanced maternal HIV disease, the more the likelihood of adverse pregnancy outcome. Larger on-going natural history studies or HIV-infected cohorts of pregnant women, such as the National Institutes of Health (NIH)-sponsored Women and Infants Transmission Study are hoped to provide more definitive answers regarding the relationship between HIV infection and pregnancy (51).

STUDY JUSTIFICATION

Pregnant mothers with advanced HIV disease form a high risk obstetric group. It is significant to find out whether their pregnancies are at risk of excess morbidity and mortality compared to healthy HIV negative mothers. Worldwide it is not yet agreed whether infection with HIV results in adverse pregnancy outcome (1). Western studies, mostly on asymptomatic HIV infected mothers, have shown little or no demonstrable impact of HIV infection on pregnancy outcome (7,8,17,31).

Western studies which have focused on pregnant mothers with advanced HIV disease are small in number had have included small numbers of pregnant mothers. They hence may lack statistical power to detect a true difference both in fetal and maternal outcome characteristics (40,43,44,45,46). In contrast a majority of Sub-Saharan Africa studies have shown adverse outcomes in pregnant mothers infected with HIV (4,10,22,23,36). Rates of spontaneous abortion, low birth weight, prematurity,intrauterine growth restriction, chorioamnionitis,fetal mortality and maternal mortality have been found to be increased in HIV infected mothers and their infants.

Though most .African studies reported adverse outcomes a few showed no significant difference in pregnancy outcome between HIV infected mothers and their healthy HIV negative controls (13) This study focuses on advanced HIV disease and hopefully will include a larger number of mothers Though most authorities believe that the more advanced the disease the worse the pregnancy outcome , this study will go along way to ascertain whether this statement is true (51).

STUDY BROAD OBJECTIVE

To determine the impact of advanced HIV disease on immediate maternal and fetal outcomes.

SPECIFIC OBJECTIVES.

To determine and compare the following in mothers with advanced HIV disease and those who are HIV uninfected.

1. Course and conduct of labor and delivery
2. Fetal outcome.
3. Maternal outcome.

STUDY METHODOLOGY

A. STUDY HYPOTHESIS

Mothers with advanced HIV disease have poorer fetal and maternal outcomes compared to HIV uninfected mothers.

STUDY DESIGN

Case control study

INCLUSION CRITERIA

I. HIV POSITIVE CASES.

1. Pregnant mothers who accept to be tested for HIV and who have given informed consent to participate in the study.
2. Admitted in KNH antenatal wards.
3. Clinically are in stage 3 or stage 4 of HIV disease according to the WHO clinical staging system for HIV infection and AIDS.
4. Those that are in the third trimester of gestation by dates.
5 Those who are not on HAART regime
6. Those mothers who are, or are not on PMTCT regime

II. HIV NEGATIVE CONTROLS.

1. Pregnant HIV negative mothers who have given informed consent to participate in the study.
2. Those that are matched to the cases by age ± 5 years and gestation by dates ± 4 weeks.
3. Those that are in the third trimester of gestation by dates.
4. Those mothers who have been followed up in the antenatal clinics of KNH.

D. EXCLUSION CRITERIA.

1. Those mothers who do not consent to participate in the study.
2. HIV positive cases and HIV negative controls who do not eventually deliver in KNH.
3. Cases and controls whose in-patient file is lost or clinical information recorded in the files is partially or completely lost.

4 Those cases who are already on HAART.

jyfPLE SIZE

e **minimum sample size was estimated using the formula**

$$- \frac{Z^2 P. (1-P,)}{(1-P)^2}$$

» = Confidence limit probability to reflect a true difference was taken to be 90 %.

Z = 1.96 which is the value assuming a 95% confidence limit.

Ira study conducted by Ryder and colleagues on perinatal transmission ofHIV-1 to infants of seropositive women in Zaire. (New Engl. **J.** Med. 1989; **320:** 1637-1642), he found that the difference in birth weight between HIV negative pregnant mothers and mothers who were HIV positive with AIDS was 23.1 %. The sample size N is then:

$$N= \frac{1.96^2 \setminus 0.231 X(1-0.231)}{(1-0.90)^2}$$

N= 68.2418

N= 68

Therefore to detect a 23% difference in birth weight between HIV positive pregnant mothers with AIDS and HIV negative mothers, with a 95% confidence and 90% power

we require 68 HIV positive mothers with AIDS and 68 HIV negative mothers, hence a total sample size of 136 mothers.

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MATERIALS AND METHODS

RECRUITMENT , DATA COLLECTION AND ANALYSIS.

The cases were recruited from the antenatal wards of Kenyatta National Hospital. They were recruited by the principal investigator assisted by two nurses General information regarding the research was explained to the participant. Having accepted to participate, the mother was counseled by the principal investigator and the nurses and then signed an informed consent form. Her in-patient file was then be marked by a serial number for identification.

HIV testing is part of the routine antenatal tests done in Kenyatta National Hospital. However if the mother had not had the test, then blood was obtained for the test and any other test required for the research. Questionnaire I was then administered by direct interview and other necessary information obtained from the antenatal card.

Once a case was identified, a control matched for age ± 5 years and gestation by dates r4 weeks was randomly interviewed from the antenatal clinic. Questionnaire I was then administered. The cases and controls were then followed up by the principal investigator and the nurses until delivery or until final pregnancy outcome. Questionnaire II was then filled and completed one week after deliver)' when the final status of the mother and the infant was determined.

If a case was discharged before the final outcome of the pregnancy was known, then she was dropped from the study but could be re-recruited if admitted in labor ward for delivery.

The raw data from the questionnaires was then verified, compiled and entered into the computer Data analysis was done using the SPSS program Totals, means and percentages were calculated. The compiled data was then cross tabulated according to:

- Sociodemographic characteristics
- .Antenatal characteristics
- Labor and delivery characteristics
- The immediate postpartum period
- Fetal outcome at birth.

The analyzed data was summarized in histograms, pie charts, tables and graphs To compare the two groups chi-squared test was used for proportional variables and the t-test for continuous variables.

STUDY **LIMITATIONS**

1. This was a hospital based study and some findings may not be extrapolated to the general population.
2. This was a short follow-up study and would miss out on the long term effects of advanced HIV infection on pregnancy outcome.
3 Viral loads and CD4 cell counts were not done and this may have an effect on outcome

ETHICAL CONSIDERATIONS

HIV infection and AIDS has profound socioeconomic and health implications once the diagnosis has been made.

The study participants were informed of the nature of the study and its intended use. On' accepting to participate in the study the participants filled a consent form thereafter they were counseled by a trained counselor.

The counseling focused on issues of mother to child transmission of the virus, breastfeeding, contraception, safe sex practices, follow up and treatment of associated illnesses.

To reduce the risk of mother to child transmission the mothers received AZT 300mg 12 hourly from 34 weeks of gestation then 200mg stat of Nevirapine at the onset of labor thereafter AZT 300mg 3 hourly until delivery.

The infant received Nevirapine 2mg/kg stat within 72 hours of birth then AZT 2mg/kg 12 hourly for 1 week.

The participants were also encouraged to be followed up in KNH high risk post natal clinic for their ailments after the study in order to benefit maximally from the health services available in Kenyatta National Hospital. There was no financial inducement to participate in the study Patients had a right to decline to participate and they continued to receive medical attention without any discrimination whatsoever.

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RESULTS

Mothers who met the study criteria were interviewed. The sample size was 136 mothers, 68 who had advanced HIV disease and 68 who were HIV negative. They were recruited from the antenatal wards and the antenatal clinic in Kenyatta National Hospital.

TABLE 1: SOCIODEMOGRAPHIC CHARACTERISTICS.

	CHARACTERISTICS	ADVANCED HIV N=68	HIV NEGATIVE N=68	PVALUE
Occupation	Employed	25 (36.8%)	49 (72.1%)	0.03
	Unemployed	43 (63.2%)	19(27.9%)	
Marital Status	Married	52 (76.5%)	55 (80.9%)	0.281
	Unmarried	16(23.5%)	13 (19.1%)	
Level of education	<= Primary	60 (88.2%)	51 (75%)	0.004
	> Primary	8 (11.8%)	17 (25%)	

There was statistically significant difference in the levels of education between the two groups with mothers who were HIV negative being more educated than mothers with advanced HIV disease ($p<0.004$). There were more employed women in the HIV seronegative group than mothers with advanced HIV disease and this reached statistical significance ($p<0.03$). There was no difference in the marital status among the two groups.

TABLE 2: COMPLICATIONS DURING THE ANTENATAL PERIOD.

	ADVANCED HIV		HIV NEGATIVE		P VALUE
	NO	%	NO	%	
VDRL positive	2	3	1	1	0.308
j PPROM	48	73	22	32	<0.001
PROM	21	31	7	10	0.003
Haemoglobin <l0g/dl	37	55	11	16	<0.001
Sexually transmitted diseases- Trichomoniasis & Candidiasis	28	42	5	7	0.004

Mothers with advanced HIV disease had more complications during the antenatal period than HIV negative mothers. The difference was statistically significant for anemia, preterm premature rupture of membranes (PPROM), premature rupture of membranes at

term (PROM) and sexually transmitted diseases. There was no difference in the VDRL status in the two groups.

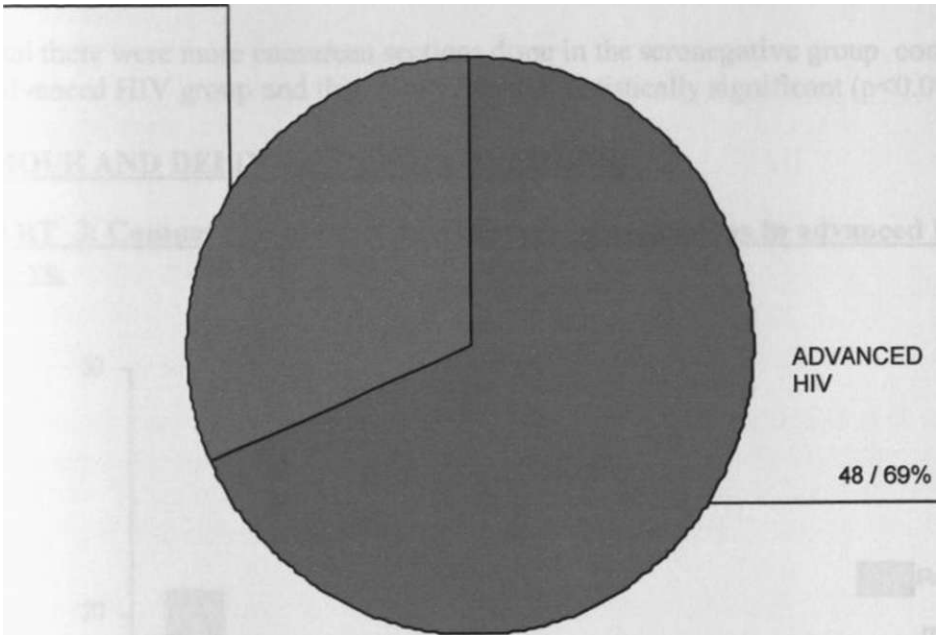
DISEASE COMPLICATIONS IN ADVANCED HIV DISEASE IN PREGNANCY. CHART 1.

Pulmonary tuberculosis and oropharyngeal candidiasis were the most common disease complications associated with advanced HIV disease in pregnant mothers (28% & *21%* respectively). Prolonged fever for more than a month as a symptom was found in 34% of mothers with advanced HIV disease. Presumptive pneumocyctis carinii was found in 3%, cryptoccocal meningitis in 5%. bacterial meningitis in 6%, septicemia 5% and severe bacterial pneumonia in 16% of mothers with advanced HIV disease.

LABOUR AND DELIVERY CHARACTERISTICS

CHART 2: DELIVERY < 37 WEEKS

HIV NEGATIVE



The difference in gestation at delivery between the two groups was statistically significant ($p<0.001$). There more mothers with advanced HIV disease who delivered at less than 37 weeks compared to HIV negative mothers.

TABLE 3: ONSET OF LABOR

	ADVANCED HIV	HIV NEGATIVE
Spontaneous onset	58	39
Induction	2	10
Caesarean section	3	14

Onset of labor was spontaneous in a greater number of mothers with advanced HIV disease. More seronegative mothers were induced compared to mothers with advanced HIV disease. More seronegative mothers had caesarean section compared to mothers with advanced HIV disease and this difference was statistically significant ($p<0.001$)

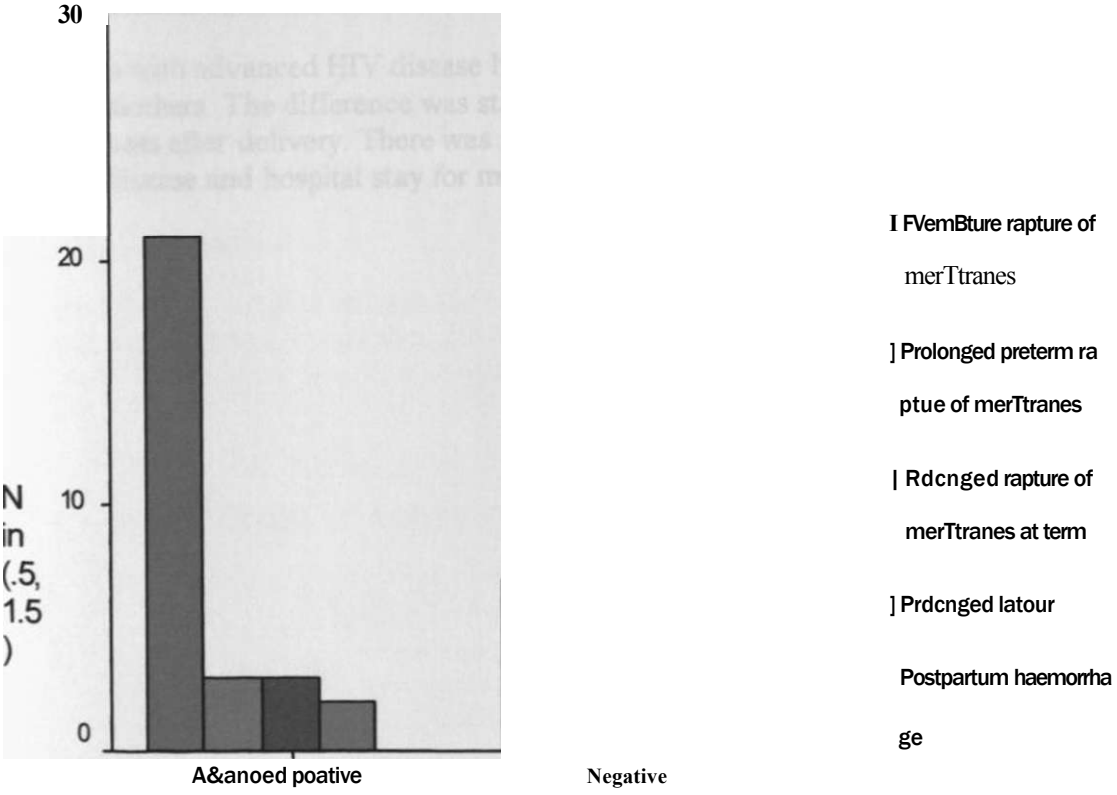
TABLE 4: MODE OF DELIVERY

TYPE OF DELIVERY	ADVANCED HIV	HIV NEGATIVE
Vaginal SVD	51	32
Vaginal Vacuum	0	2
Vaginal Breech Delivery	3	1
Caesarean section	9	28

In total there were more caesarean sections done in the seronegative group compared to the advanced HIV group and this difference was statistically significant ($p<0.001$).

LABOUR AND DELIVERY COMPLICATIONS.

CHART 3: Comparison of labor and delivery complications in advanced HIV vs. HIV -ve.



There was statistically significant difference in PPROM and PROM between the two groups, with more mothers in the advanced HIV disease group. There was no difference in terms of prolonged labor both at term and preterm and postpartum hemorrhage.

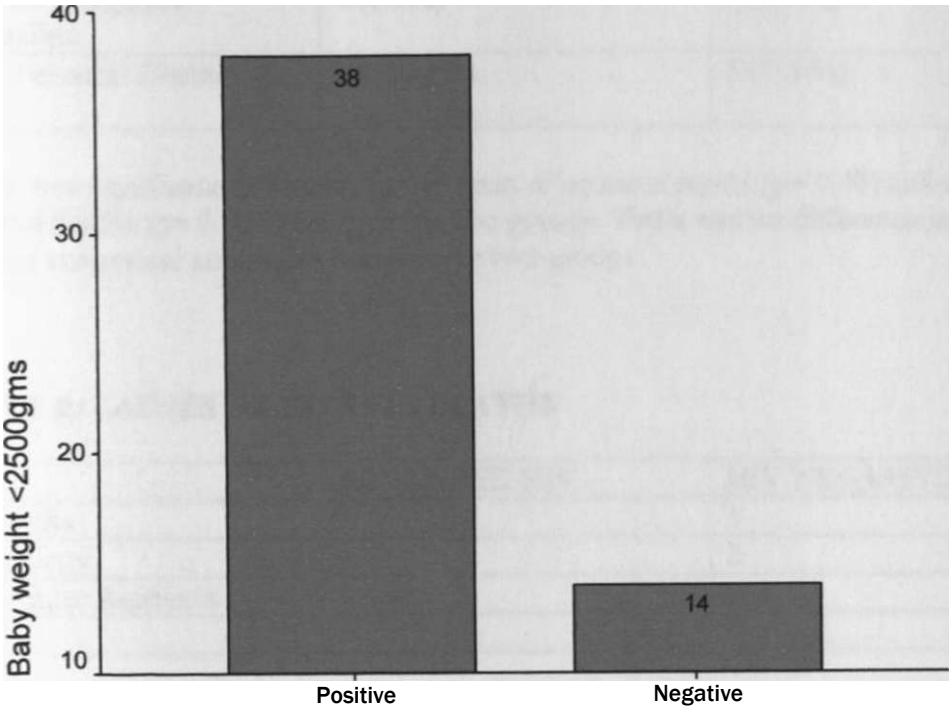
TABLE 5: MATERNAL PERLNATAL COMPLICATIONS

	Advanced HIV N=68		HIV NEGATIVE N=68		P Value
	No.	%	No.	%	
Meconium staining of liquor (MSL)	22	32	14	21	0.12
Maternal Pyrexia after delivery temp. > 38 C	15	23	6	9	0.032
Hypertensive disease. DBP > 90 mmHg.	9	14	8	12	0.745
Chorioamnionitis	15	22	3	4	0.004
Maternal Hospital stay > 1 week after delivery	23	44	10	27	0.099

More mothers with advanced HIV disease had perinatal complications compared to seronegative mothers. The difference was statistically significant for chorioamnionitis and febrile illness after delivery. There was no statistically significant difference in hypertensive disease and hospital stay for more than a week.

CHART 4. BIRTH WEIGHTS

Birth weight <2500gms



The difference in birth weights between the two groups was statistically significant. 38 (56%) infants had birth weight less than 2500gm at delivery in the advanced HIV group compared to 14 (21%) in the seronegative group ($p<0.001$).

TABLE 6: APGAR SCORE AT 5 MINUTES

APGAR SCORE	ADVANCED HIV	HIV NEGATIVE	P VALUE
> 7	44 (69%)	60 (90%)	0.003
4-7	18(28%)	8(12%)	0.02
<4	11(17%)	5 (8%)	0.089

The mean Apgar score at 5 minutes was 6.75 for infants born to seropositive mothers and 8.37 for infants of seronegative mothers ($p= 0.001$)The Apgar scores at 5 minutes were better in the seronegative group with those scoring >7 and 4-7 having statistically significant difference compared to the advanced HIV group.

TABLE 7: EARLY NEONATAL COMPLICATIONS

	ADVANCED HIV	HIV NEGATIVE
Neonatal sepsis	5(11.8%)	3 (4%)
External Congenital .Anomalies	4 (5.9%)	4 (5.9%)
Early Neonatal Deaths	14(20.1%)	5 (7.3 %)

There was significant difference in the rates of neonatal sepsis (p= 0.03) and early neonatal deaths (p= 0.025) between the two groups. There was no difference in rates of external congenital anomalies between the two groups.

TABLE 8: CAUSES OF INFANTS DEATHS

	ADVANCED HIV	HIV NEGATIVE
Still births	j	1
Prematurity	4	2
I Low Apgar Asphyxia	3	1
i RDS	4	1

Prematurity was the leading cause of infant deaths in both groups, followed by RDS and low Apgar asphyxia. The rate of stillbirth was not significantly different in the two groups.

TABLE 9: FETAL OUTCOME

	.ADVANCED HIV N = 68	HIV NEGATIVE N = 68	P Value
Gestational Age Mean ± S D	31.0642 ±3.9649	32.2448 ±3.8946	0.1789
Babies bom <37 weeks	48 or 73%	22 or 32 %	P<0.001
Birth Weight Meant SD	2375.1489 ±785.0214	3021.4261 685.5782	P< 0.001

A total of 60 babies were born before 37 completed weeks. There were 48 from the advanced HIV group and 22 from the seronegative group. Mothers with advanced HIV disease were found to be predisposed to preterm deliveries and this difference was statistically significant (p<0.001).

TABLE 10: INFANT STATUS 1 WEEK POST DELIVERY

	Advanced HIV	HIV Negative	P value
Alive and well	42 (61.8%)	55 (80.9%)	0.06
Unwell	9(13.2%)	7(10.1%)	0.89
Deceased	17(25%)	6(8.8)	0.025

More infants born to mothers with advanced HIV disease were deceased at the end of follow up compared to those bom to seronegative mothers. This difference was statistically significant (p= 0.025).

TABLE 11: MATERNAL STATUS 1 WEEK POST DELIVERY

	Advanced HIV	HIV Negative	P value
Alive and well	37 (54.4%)	58 (85.3%)	0.054
Unwell	23 (33 8%)	10(14.7%)	0.099
Deceased	6 (9%)	0 (0%)	0.028

There were six maternal deaths in the advanced HIV group and no maternal deaths in the HIV seronegative group This difference was statistically significant (p= 0.028). Three mothers passed away due to complications of pulmonary tuberculosis, two from bacterial meningitis and one from presumptive Pneumocystis carinii pneumonia (PPCP). A total of 33 mothers were unwell at the end of follow up with a higher proportion being in the advanced HIV group (33.8% Vs. 14.7%. p= 0.099).

DISCUSSION

This was a prospective cohort study whose aim was to document the impact of advanced HIV disease on the immediate outcome of pregnancy Worldwide most of the studies done on HIV in pregnancy have focused on interruption of maternal-fetal transmission of the virus. A number of outcome studies have also been conducted. In the Western countries and also in some African countries most studies compared pregnancy outcome in asymptomatic HIV mothers and their seronegative counterparts. Few pregnancy outcome studies have been done on mothers with advanced HIV disease.

This study recruited 68 pregnant mothers with advanced HIV disease (WHO Stage 3 and 4) and 68 HIV negative mothers. Overall this study found that pregnant mothers with advanced HIV disease have poorer fetal and maternal outcome compared to HIV negative pregnant mothers.

Mothers with advanced HIV disease had more complications during the antenatal period compared to seronegative mothers. Mothers with advanced HIV booked with significantly lower hemoglobin than their seronegative counterparts (55% vs. 16%). This compares favourably with what Sukwa et al found in Ndola Zambia (52). Sexually transmitted diseases occurred more significantly in mothers with advanced HIV disease (42% vs. 7%). This agrees with what Temmerman and colleagues found in their study (4.4% vs. 2%) (53). Burns et al (54) found that vaginal colonization with candidiasis occurred frequently in pregnant mothers infected with HIV virus. Our study had the same findings but the difference did not achieve statistical significance (25% vs. 15%. P=0 261).

Syphilis is more common in HIV positive women in Africa with an incidence of up to 33% being reported (55). In our study an increased risk among mothers with advanced HIV disease was found though the difference was not statistically significant (3% vs. 1% p=0.308). This rate is similar to what Gichangi (56) reported at Pumwani Hospital in Nairobi. It compares well with the 3.25 reported by Temmerman at Langata where she also found that the HIV positive mother was 2.5 times more likely to have a

positive VDRL test (53). Mmiro in Uganda (23) found that HIV positive mothers were more likely to have been tested for syphilis (22.0% vs. 14.5%). In our study a significant number of mothers with advanced HIV disease had PROM and PPROM. Omondi, L B Kumba (57) found that HIV positive mothers tended to have PROM (14.9% vs. 5%). Similar findings were observed by Mmiro in Uganda (23). This has adverse implications for MTCT.

The mean gestation at delivery for mothers with advanced HIV disease was 35 5 weeks compared to 38.2 weeks for HIV negative mothers. This was statistically significant ($p<0.001$). Therefore advanced HIV disease predisposed to prematurity Prematurity is associated with high neonatal mortality and morbidity in our set-up. Kumba (57) had similar findings.

In our study a total of 40 caesarean sections were performed, 12 (17.6%) from the advanced HIV disease group and 28 (41.2%) from the seronegative group. This difference was statistically significant ($p= 0.03$). This could be due to increased rates of anemia in mothers with advanced HIV disease and also due to their poor general conditions which increases their surgical risk. Among the HIV positive mothers that Minkoff studied in Brooklyn, (58) the caesarean section rate was much higher In.Rwanda caesarean section was associated with morbidity and mortality especially when CD4 counts were low (20).

There were more women in the advanced HIV group who were delivered SVD compared to their seronegative counterparts (75% vs. 47%) in our study. Again this could be due to the effect of anemia and high rates of associated infections which predispose to preterm labor and delivery. HIV itself could also be the cause of early labor and delivery. In his study, Kumba (57) found that 82% of seropositive mothers had vaginal delivery compared to 64% of seronegative mothers. No vaginal vacuum was done in the advanced HIV group with only one being done in the seronegative group.

In a study by Nyongo (38) moderate to severe chorioamnionitis was found in 20% of preterm and 8% of term pregnancies. Within preterm deliveries the placentas showed chorioamnionitis in 37% of cases where the woman was HIV positive and 18% of matched seronegative cases. In our study 22% of mothers with advanced HIV disease had chorioamnionitis compared to 4% in the HIV negative group ($p= 0.004$). This achieved

statistical significance. Meconium staining of liquor in labor was not significantly different in the two groups (p= 0.12). In contrast to our study, Kumba (57) found that HIV positive mothers were less likely to have meconium stained liquor than the HIV negative controls (9.9% vs. 18.8%. p= 0.07). Postpartum maternal pyrexia between the two groups was statistically significant (p = 0.032). More mothers in the advanced HIV group suffered postpartum pyrexia (Temp > 38 C).

Estimated blood loss after delivery was not significantly different between the two groups in our study (p = 0.195), though mothers with advanced HIV disease had a slightly higher mean compared to their seronegative counterparts (362.92 mis vs. 315 15mls). This could be due to the effect of anemia.This is in agreement with what Mmiro (23) found in his study in Uganda. Leroy (20) in Rwanda found that postpartum hemorrhage was more likely to occur in the HIV positive women. This was in contrast to our study which showed no difference.

Pulmonary tuberculosis was the leading disease associated with advanced HIV according to our study. This was followed by orophareangeal candidiasis, severe bacterial pneumonia, bacterial meningitis, septicemia, cryptoccocal meningitis and presumptive **Pneumocystis** carinii pneumonia. This is in contrast to the Western study which found that **Pneumocystis** carinii pneumonia was the most frequent HIV-AIDS related infection in pregnancy (45). Our study recorded 6 maternal deaths in the advanced HIV group and no maternal deaths in the HIV negative group. This difference was statistically significant (p = 0.028). This finding compare well with the Western studies which also recorded **higher** maternal mortality in pregnant mothers with AIDS(40). Three mothers passed **away** from complications of pulmonary tuberculosis, two from bacterial meningitis and one due to presumptive **Pneumocystis** carinii pneumonia.

Many studies in developed countries have shown little difference in birth weights of babies born to HIV positive mothers. However, Minkoff (58) found high rates of low birth weight Low birth weight is mostly reported in Sub Saharan African countries and especially among symptomatic mothers or mothers with .AIDS In our study the difference in birth weight was significant, with mothers with advanced HIV disease giving birth to more low birth weight babies compared to the seronegative group

($p<0.001$). This finding is similar to those of Temmerman in Nairobi (53), Mmiro in Uganda (23), Leroy in Rwanda (20), Chamiso in Ethiopia (59) and Taha in Malawi (22). However Lepage (13) in Rwanda found no diference in birth weights between HIV positive and HIV negative mothers.

The 5 minute Apgar score was poorer in infants born to mothers with advanced HIV disease and this difference was significant ($p = 0.003$). Similar differences were reported by Mmiro in Uganda (23) and Chamiso in Ethiopia (59). Temmerman and colleagues did not find any significant difference in Apgar scores between infants of seropositive and seronegative mothers (19).

Though there were no significant differences in neonatal sepsis, congenital anomalies and stillbirth rates between the two groups, early neonatal deaths occurred frequently in the advanced HIV group. This difference was statistically significant ($p =0.025$). Most mothers gave birth to live babies (95.6% for advanced HIV disease and 98.5% for the HIV negative). There was a 4% stillbirth rate among the cases and 1% among the controls. Mmiro (23) reported a still birth rate of 3.6% among the cases and 1.6% among the controls.

Majority of the babies had no complications after delivery, but the babies of mothers with advanced HIV disease had more complications compared to their seronegative counterparts. Significantly more babies of seropositive mothers had prematurity (73% vs. 32%). This data indicates that delivery in mothers with advanced HIV disease should ideally take place in controlled conditions where close observation and high risk infant care is readily available.

Overall, these data show that pregnancies in mothers with advanced HIV disease is at an increased risk with a significant risk of maternal and fetal mortality and morbidity and should be managed as high risk. There was statistically significant increase in maternal mortality and neonatal and perinatal mortality. It is clear that pregnant mothers with advanced HIV disease have poorer fetal and maternal outcomes compared to their HIV negative counterparts. Statistically detectable biologic effects upon pregnancy occurs in mothers with advanced HIV disease.

CONCLUSION.

1. Advanced HIV infection in pregnancy is associated with maternal anemia, sexually transmitted diseases, premature rupture of membranes both at term and preterm and chorioamnionitis.
2. Maternal mortality is increased in pregnant mothers with advanced HIV disease
3. Early neonatal death is common in infants bom to mothers with advanced HIV disease compared to their seronegative counterparts. Congenital anomalies and stillbirth rates are similar in the two groups
4. Infants born to mothers with advanced HIV disease have an increased tendency to be premature and to have low birth weight compared to infants bom to HIV negative mothers.
5. Pregnant mothers with advanced HIV disease have poorer fetal and maternal outcome compared to pregnant HIV negative mothers.

RECOMMENDATIONS.

1. High risk antenatal surveillance should be employed in the management of pregnant mothers with advanced HIV disease. Anemia should be sought for and treated aggressively. Sexually transmitted diseases especially chlamydia, gonorrhoea and syphilis should be screened for at the first antenatal visit. HIV testing should be made part of routine antenatal testing in the antenatal clinics.
2. Haematinics and multivitamins should routinely be given to pregnant mothers with advanced HIV disease.
3. Delivery of pregnant mothers with advanced HIV disease should take place under controlled conditions, in which close observation and high risk maternal and neonatal care is readily available.

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GYNAECOLOGY CASE No. 1

MULLERIAN AGENESIS: MAYER- ROKITANSKY-KUSTER-HAUSER SYNDROME

Name:	**Grace Wanjiru**	**DOA:**	**23.08.2005**
Age:	**25 years**	**DOD:**	**26.08.2005**
Ip. No.	**1040741**		
Parity:	**0+0**		

Presenting complaints

Grace presented with complaints of lack of menstrual flow since birth, lower abdominal pains and difficult painful coitus for the previous 3 months.

History of presenting complaints

She complained that she had not received her menses which to her knowledge she ought to have received by her age. She has had normal breast, axillary and pubic hair development. Her voice remained feminine. She had not noticed any discharge from the nipples nor had she noticed any abnormal hair growth on her body. She reported no history of persistent headaches and her vision remained normal.

She had cyclic lower abdominal pains approximately every 2-3 weeks The pain was mostly dull in nature but occasionally it was colicky and radiated to the back. The pain was relieved by analgesics but had no aggravating factors and occasionally it resolved spontaneously. Urinary and bowel habits had been normal. She did not report any abdominal swelling , vaginal bleeding or abnormal vaginal discharge. Her appetite had been good with no history of abnormal weight changes.

She had been married for 3 months during which she had her first sexual debut. Coitus was difficult and painful. She reported no postcoital bleeding.

In the year 2000 she sought treatment at a hospital in Nyahururu but could not afford the investigations required. In 2004 she sought treatment at a Marie Stopes clinic in Nairobi

where she was examined and referred to KNH. At KNH GOPC she was informed that she had a problem in her reproductive system and would require further surgical evaluation

Past medical and surgical history

She had had no surgery nor had she been on any medications for any chronic illness. She had not used any form of contraception. She had no food or drug allergies

Family and social history

She was a housewife and did not smoke nor take alcohol. She had one elder sister who was married and had children. The only other sister was younger than her and had no menstrual problems.

General Examination

She was in good general condition not pale, jaundiced or cyanosed. She did not have any edema or lymphadenopathy. The Thyroid gland appeared normal. Vital signs were within normal. She had female physical appearance with breast development Tanner stage V. She had female pubic hair distribution and normal axillary hair. Scalp hairline was female type and she did not have abnormal growth of hair on any part of her body.

Abdominal examination

Abdomen had normal fullness and symmetrically moving with respiration. The umbilicus was flat. No areas of tenderness were noted and no organomegaly or other masses felt.

Pelvic examination

The labia majora and minora appeared normal. Mons pubis appeared normal with normal pubic hair distribution. The clitoris appeared to be normal The urethral opening appeared normal. A septum was noted at the vaginal opening obscuring the rest of the vaginal canal Anal opening appeared normal. Speculum examination was not possible due to the vaginal septum. Digital examination of the vagina revealed a short vaginal canal approximately 3cm long with a blind firm end. No cervix was palpable. It was difficult to do a bimanual examination but the adnexae were free.

Rectal examination.

Revealed normal anal opening. A digital examination revealed normal anorectal canal and normal mucosa. A rudimentary uterus was palpated and the Pouch of Douglas was free.

The central nervous, cardiovascular and the respiratory systems were essentially normal

Impression.

An impression of primary amenorrhoea with a vaginal septum and possible mullerian dysgenesis was made.

Investigations.

The following investigations were requested for:

Pelvic **scan- July 2005:** A hypoplastic uterus measuring 3.8cm x 3.6cm x 2.8cm is seen. Both ovaries are demonstrated and are within normal limits but no follicles noted within the ovaries. No pelvic or adnexal masses are seen. Conclusion: Features suggestive of a hypoplastic uterus possibly due to hormonal imbalance. Hormonal profile is recommended.

Haemogram	
WBC	6.7 x 10/L
RBC	4.68 x 10/L
Hb	15.3 g/dl
Pit	174 x 10/L

	Renal Function Tests
Na+	130 mmo/L
K+	4.1 mmol/L
Urea	3.6 mmol/L
Creatinine	64 umol/L

Management

She was counseled and an explanation of her condition given and was informed of the need for diagnostic laparoscopy to further evaluate her condition. She consented for diagnostic laparoscopy and was prepared for theatre.

Examination under anaesthesia (EUA)

In theatre anaesthesia was induced, she was put in Trendelenburg position, cleaned and draped. Examination under anaesthsia revealed a short vaginal canal (3cm) with a blind firm end. No cervix was palpable. Rectal exam revealed a small rudimentary uterus with the POD and adnexae free with normal anorectal canal.

A stab incision (10mm) was made within the umbilicus and the trocar and canula inserted. Entry was successful and a Verres needle was connected and pneumoperitoneum achieved. The light source and the laparoscope were then connected and the pelvis and abdomen visualized. 5mm incisons were made on the right and left iliac fosa and trocar and canula inserted under direct vision.

Diagnostic laparoscopy findings.

- The uterus was hypoplastic
- There was no cervix
- Both ovaries were normal with active follicles
- > The fallopian tubes were not demonstrated only a rudimentary fimbrial end on the left.
- The round ligament were seen but appeared to have fused with the tubes.
- Pelvic and abdominal endometriotic adhesions were seen
- The Pouch of Douglas was demonstrated and was free
- The rectum was normal.

- Peristaltic movements of the ureters were noticeable.

Post-operative.

She was uneventfully reversed from general anaesthesia and eventually wheeled to the ward On the first post-operative day at 9.00 am she was found to be in good condition and fully awake. Bowels were active and the wound sites were clean with mild abdominal

tenderness. The findings at laparoscopy were explained to her and the fact that she would not be able to conceive normally and carry a baby in her uterus was made clear Options of IVF with a surrogate mother and adoption were explained to her, though the former option was clearly not feasible for her due to financial and social circumstances She was informed that surgery could be done to open up the vaginal canal but this was only in an to attempt to improve her sexual life. She was counseled and discharged to be seen in the GOPC and the patient support centre in two weeks to continue discussions on the options as to the way forward and also to receive psychological support. At the time of writing the report her appointment was not yet due.

was in our patient. The uterus may be absent or hypoplastic as was in this case and the fallopian tubes and round ligaments also show variable degrees of abnormalities. The ovaries are usually normal and active. Various degrees of endometriosis can be encountered possibly due to retrograde menstruation and can cause pelvic pain and dyspareunia (3). This was demonstrated in our patient.

MKRHs should be differentiated from androgen insensitivity syndrome (AIS or testicular feminization syndrome) and conditions such as Turner's syndrome. In AIS there are elevated levels of LH and FSH and also the karyotype will be 46 XY whereas in MRKHs hormones will be normal and karyotype will be 46 XX. Abdominal or inguinal testicles may be present in AIS (4). Apart from hormones and karyotyping imaging techniques can be used to aid in diagnosis of MRKHs. Sonography, magnetic resonance and CT scan can all demonstrate abnormal pelvic organogensis and can be used for evaluation of subperitoneal structures and anatomical relationships of various pelvic structures (5). Intravenous urogram can be used to assess the renal system. Plain radiography can be used to exclude vertebral anomalies. The patient presented had a pelvic scan which showed a hypoplastic uterus.

Laparoscopy provides direct assessment of the uterus, ovaries and fallopian tubes and will demonstrate the extent of organ malformation (6). At laparoscopy uterine remnants or endometriosis causing chronic pelvic pain can also be removed (6). Our patient underwent laparoscopic evaluation and the findings were consistent with MRKHs.

Treatment of MRKHs is multidisciplinary with involvement of an endocrinologist, geneticist, psychiatrist, plastic surgeon and occasionally urologist and orthopaedic surgeon (7). The psychological implications of MRKHs are profound but the physiologic consequences are amenable to surgical and medical treatment. Surgical methods used to create a vagina include, Williams and Mc Indoe vaginoplasties, rotational flap methods and laparoscopic techniques such as the modified Vechietti technique (8). Though available these techniques are associated with morbidities such as excessive skin scarring at donor sites, postoperative rectovaginal, urethrovaginal and vesicovaginal fistulae, vaginal discharge, dyspareunia, vaginal stenosis, inadequate vaginal lubrication and poor

DISCUSSION

Mayer-Rokitansky-Kuster-Hauser syndrome (MRKHs) describes the condition of vaginal atresia with other variable mullerian (paramesonephric ducts) anomalies in the presence of other normal female developmental characteristics (1). Usually it remains undetected until the patient presents with primary amenorrhoea with normal female sexual development in adolescence or early adulthood.

The incidence of congenital absence of the vagina in the United States is 1 case per 4000-5000 births (1). Local data is not available. The cause of MRKHs is unknown and no gene has been linked to this condition (1). MRKHs is sporadic and female relatives of the affected persons have no increased risk for the condition. It is postulated that MRKHs results from failure of development of the mullerian system during the fifth week of gestation (1). The uterus , cervix and upper 2/3 of the vagina develops from the fused lower end of the paramesonephric ducts while the tubes develop from the upper unfused part (2) The lower 1/3 of the vagina develops from the urogenital sinus hence there may be a vagina of variable depth in MRKHs. The ovaries develop from mesoderm while the follicles are ectodermal in origin so their development is not affected (2). The renal system develops simultaneously from the mesonephros and a variety of renal malformations can result as well. Patients can also present with recurrent urinary tract infections or voiding difficulties. Vertebral anomalies though of minimal significance can also be associated with MRKHs. Our patient did not have any renal or vertebral anomalies

MRKHs is usually diagnosed in adolescence when all the secondary sexual characteristics develop but menses fail to appear (primary amenorrhoea). Occasionally as in our patient, it can be diagnosed late due to dyspareunia or infertility. Patients can have cyclic abdominal pains such as was found in our patient

MRKHs patients are phenotypically and genotypically female (3). The height is usually normal with well developed female secondary sexual characteristics. The labia majora, minora and the clitoris are usually well developed. The vagina may be totally absent, a small dimple, or of variable length but with a blind firm end. The cervix is usually absent. Speculum and digital vaginal examinations are usually difficult or impossible as it

compliance with post surgical dilatation schedule (8,9). Moreover, specialists in this type of surgery are scarce in our region.

Patients with MRKHs will not be able to conceive by natural means but can be able through invitro-ferlilization techniques using a surrogate mother (10). The other option is adoption which may be the only option in our set-up where assisted reproduction technology (ART) is still in its infancy.

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ACUTE PELVIC INFLAMMATORY DISEASE (PID)

ANTIMICROBUL THERAPY

Name	**L.K.**	**DOA**	15.06.2004
IP NO	0915881	**DOD**	17.06.2004
Age	23 years		
Parity	1+0		
Diagnosis :	Acute PID		

Presenting **Complaints:**

The patient had lower abdominal pains and vomiting over a period of 3 days.

History of presenting illness:

She had insidious onset of lower abdominal pains which were persistent in nature, radiated to the back and had no relieving factors. She had episodes of vomiting precipitated by feedings. She had constipation with tenesmus and subsequently passed hard pellets of stool.

The patient denied having had any abnormal per vaginal discharge, dyspareunia or bleeding and she had normal micturition habits.

Obstetric and Gynaecologic History:

She was a para 1+0 whose last delivery was in 1999, with the baby being alive and well. The last menstrual period was sometime in August, 2003. She initially had regular menses, having had her menarche at the age of 14 years.

Past Medical History

She had completed the initial phase of treatment for Mycobacterium tuberculosis with 800mgs of ethambutol and 5 tablets of rifater and was on the continuation phase of 5 tablets of rifater alone. She reported having had marked improvement on this treatment

Family and Social History

She was married, unemployed and never drank alcohol. She also did not smoke cigarettes. The husband had a small scale business of selling second hand clothes.
Drug: She had no known food or drug allergy.

Systemic Enquiry:

There was no abnormality.

General Physical Examination:

The patient was sick looking and seemingly in much pain. She was not pale, was febrile and had no jaundice. The temperature was **38°C** and the pulse 141 per minute and regular The blood pressure was 110.70 mmHg, while the respiratory rate was 22 per minute

Abdominal Examination:

The abdomen appeared flat with marked tenderness and guarding at the hypogastrium. There were no abnormal masses.

Vaginal Examination:

The external genitalia appeared normal. There was mucoid, yellowish foul smelling vaginal discharge Cervical excitation test was positive with marked adnexal tenderness. Bimanual palpation was not possible due to the tenderness. Endocervical swab was taken for microscopy, culture and sensitivity.

Other Systems:

These were essentially normal

Diagnosis:

Acute pelvic inflammatory disease

Management

The following investigations were ordered for;

1 Haemogram:

WBC -9.6 X 10/L

Hb - 10.2g/dl

Platelets - 225 x 10/L

2. Urea, Electrolytes and creatinine

Na+ 140 mmol/L

K + 4.6 mmol/L

BUN 4.0 mmol/L

Creatinine 85 umol/L

3. ' Pregnancy test - Negative.

4. Pelvic ultrasound scan - This was not done because it was booked in five days time and the patient was discharged after two days.

5 Endocervical swab - No growth obtained.

The patient was started on 2 mega units of intravenous crystalline penicillin 6 hourly, 80mgs of intravenous getamicicn 8 hourly and 500mg of intravenous metronidazole 8 hourly. She was also given oral doxycycline a dose of lOOmg twice a day.

For pain relief the patient was given oral diclofenac. After 48 hours, she was afebrile and the abdominal pains had reduced markedly. She was counseled to take a HIV test but she declined citing unpreparedness for the test. She was discharged home on oral metronidazole, diclofenac and ciprofloxacin with instruction that she report back for review at the gynaecology outpatient clinic after one week. She had recovered fully when she was seen at clinic.

Discussion

L.K was a 23 -years -old para 1+0 who was admitted with severe lower abdominal pains incidentally found to have foul smelling per vaginal discharge. The diagnosis of acute pelvic inflammatory disease was most likely appropriate, although the results of the microbiological tests done on the endocervical swab were never obtained

Pelvic inflammatory disease (PID) is a general term commonly used to describe an infective process of the upper gential tract. The infections of the upper genital tract most commonly involve the fallopian tubes (salpingitis), but the endomentrium and the ovaries are generally involved as well (endometritis, parametritis, salpingitis and oophoritis). The disease process cab be divided into acute and chronic forms [1] Our patient had acute PID

Acute PID is usually a consequence of infection with gonococcus and chlamydia. Other microorgarnisms that can cause it are aerobic Streptococcus, Staphylococcus Pyogenes, Eschrichia coli. Mycoplasma hominis, Ureapiasma urealyticum and even tubercle bacilli. Most cases of PID are the results of a polymicrobial infection caused by microorganisms ascending from the vagina and cervix to infect the mucosa of the endometrium and fallopian tubes. It is at times difficult to determine which of the organisms isolated from the endocervix is responsible for the ongoing episode of acute PID"

Fomulu found a polymicrobial pattern at the Kenyatta nnational Hospital, with Escherichia coli ocuring in 30% of cases of pelvic infection, but Cartley (1972) found at the same hospital that gonoccus was found in 75% of patients with PID, 4% of these having had pelvic abscess [3 J]. Chow and Manif postulated that gonococcus initiates acute PID and produces tissue damage that changes the local environment to allow aerobic and anaerobic organisms from the vaginal and cervical flora to enter the upper genital tract.

Esheribach and Sweet have, however, suggested that not all PID follows gonococcal infection and that acute PID may initially have a polymicrobial aetiology [1]. Overally, acute PED occurs in about 1% to 2% of young, sexually active women each year.

Predisposing factors to the occurrence of acute PED include multiple sexual partners, previous PID that was not well treated or untreated, nulliparity and HIV infection. Age is also a factor and the incidence of PID decreases with age, 70%, of the patients being younger than 25 years.

Our patient was 23 years old. Surgical procedures that break the cervical mucus barrier like placement of intra-uterine centraceptive device, endometrial biopsy and curretage, hysteroscopy and hysterosalpingography have also been implicated. Abortion is also known to be a risk factor[6] At the Kenyatta Hospital, PID has been found to be commonest in those below 20 years and follows abortion in 18.2% of the patients [1]. Given that tuberculosis is a disease commonly associated with immunosupression, chances that our patient was HIV-positive could be ruled out, but she was not tested for HIV

Oral contraceptives have been thought to reduce the risk of PID and it sis probable that the progestin component makes the cervical mucus thicker inhibiting sperms and bacteria from penetrating into the upper genital tract [2].

The diagnosis of acute PID is usually clinical. The patient presents with lower abdominal pains, cervical motion tenderness and adnexal tenderness . There may also be fever, cervical or vaginal discharge and leukocytosis. Jacobson and Westrom have reported that lower abdominal pain, pelvic pain, fever and leukocytosis are present in only 15-30% of actual PID cases[1]. Pain in the lower abdomen and pelvis is present in more that 90% of patients at initial presentation.

The pain is usually described as dull and accentuated by motion or sexual activity and is usually of recent onset, most likely one week or less. Upto 75% of acute PID are associated with endorcervical infection and coexistent purulent vaginal discharge, but nausea and vomiting are relatively late symptoms. Abnormal vaginal bleeding, especially menorrhagia or spotting may occur in upto 40% of the patients. Perihepatic inflammation and adhesions, more commonly known Fitz - High- Curtis syndrome, develop in 1 -10% of the patient with acute PID.

The patient may have right upper quadrant pain, pleuritic pain, and tenderness in the right upper quadrant when the liver is palpated. It is believed to develop from vascular or transperitoneal dissemination of either N. gonorrhoea or chlamydia trachomatis to produce the perihepatic inflammation. Other organisms may be involved. Despite the short coming of diagnosis, laparoscopic visualization of the pelvis is still the most accurate method of confirming the diagnosis of acute PID

It is even more important in the exclusion of other diagnoses and surgical emergencies. The appearance of the pelvic organs can vary from erythematous, indurated, edematous oviducts, pockets of purulent material, to a large pyosalpinx or tubo ovarian abscess [2,0] Other less invasive methods of diagnosis exist. For instance, Pavonen and associates reported 90% correlation between histologic endometritis and laparoscopically confirmed salpingitis.

A delay of 2 -3 days may result in limited clinical application of this disease such as acute appendicitis, adnexal masses and ectopic pregnancy.
Culdocentesis may be perfomed and a WBC count of >30 x 10^3/ml would suggest acute PID The normal level is <1000 cells.

The sequelae of acute PID can be devastating and include infertility, ectopic pregnancy, chronic pelvic pain, residual infection and , rarely, mortality which could be as a result of adult respiratory distress syndrome (ARDS) due to severe infection. This calls for prompt and effective treatment whose goals should be to eliminate the acute infection and symptoms in addition to preventing the long term sequelae

Based on the consequences that PID is polymicrobial in cause, empirical protocols should cover a wide range of mocroorganisms, including N-gonorrhoea, Chlamydia trachomatis, anaerobic rods and cocci, gram negative aerobic rods, gram- positive aerobes, and Mycoplasma species. Controversy exists over the issue of outpatient treatment with oral antibiotics versus inpatient treatment with parenteral antibiotics. Our patient was initially treated on an inpatient basis and allowed home on oral medication.
The treatment of acute PID should include that of the male partner and education for the prevention of infection.

The patients whose PID are complicated by abscess formation may need surgical intervention which may be laparoscopic or by laparatomy. Percutenous drainage under sonographic or CT - Scan guidance may also be helpful [12]

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GYNAECOLOGY CASE No. 3

INCOMPLETE ABORTION - MANUAL VACUUM ASPIRATION

Name	C K	DOA	08.06.2004
Age	25 Years	DOD	09.06.2004
Parity	0+0	WD	1 D
Ip No	0986754		

PRESENTING COMPLAIN

She presented with complaint of vaginal bleeding for one day. She also had associated backache.

HISTORY OF PRESENTING COMPLAINT

She was well prior to the onset of vaginal bleeding which was spontaneous, dark in colour and in clots. There was associated backache and lower abdominal pains. She also had dizziness, but no dysuria.

OBSTETRICS AND DYNAECOLOGICAL HISTORY

She was a para 0+ 0 gravida 1. Her last normal monthly period was on 09 / 03/ 04 and EDD was on 16 /12 / 04. She had an amenorrhoea of 14 weeks. She had started attending antenatal clinic. Her menarche was at 15 years Menses were regular every 28 days lasting 4 - 5 days. She had not used any contraception.

PAST MEDICAL HISTORY

This was unremarkable

FAMILY AND SOCIAL HISTORY

She was a housewife. Her husband was a butcher. They neither smoked cigarettes nor drank alcohol. Her mother is on treatment for pulmonary tuberculosis.

PHYSICAL EXAMINATION

She was a young woman in fair general condition, clinically afebrile. She had mild pallor, no jaundice nor lymphadenopathy. Respiratory rate was 20 / minute, pulse rate 100 beats per minute, blood pressure 110/ 70mmHg.

ABDOMINAL EXAMINATION

The abdomen was soft, not distended. There was suprapubic tenderness and the uterus was 12 weeks gestation.

VAGINAL EXAMINATION

Normal external genitalia, cervix was posterior 2cm dilated and products of conception were felt. Examining finger was blood stained.

DLAGNOSIS

An impression of incomplete abortion was made

MANAGEMENT

She was for fluid replacement and manual vacuum aspiration.

She was started on IV fluids (normal saline and 5% dextrose). Blood was taken for grouping and cross-matching and she was started on IV gentamicin and crystalline penicillin.

The patient was then taken to the procedure room and put in lithotomy position, cleansed and draped. Vaginal examination confirmed earlier findings. Speculum was inserted and cervix cleaned. There were no cervical tears or lacerations. The anterior lip of the cervix was held at 12 o'clock position. Canula size 10 was inserted into the cavity and connected to the syringe and vacuum created.

Contents of the uterus were evacuated by rotating the syringe through 360°C and pushing it back and forth. 60mls of products of conception were aspirated and the cavity was confirmed empty when no more products were evacuated and there was resistance to movement of canula and a gritty feeling was felt.

There was minimal bleeding after the procedure. She taken to the ward to recover and was discharged home later in the day on antibiotics (doxycycline and flagyl), and analgesic (brufen).

She was also to pass through the family planning clinic for preconception care and early antenatal care in future pregnancies.

DISCUSSION

C.K. presented with incomplete abortion and manual vacuum aspiration was done.

Abortion is the termination of a pregnancy less than 20 weeks or fetal weight less than 500 grams (1).

Abortion can either be spontaneous or induced. Abortion is one of the greatest pubic health problems because of its repercussion to maternal morbidity and mortality and also because of its ethical, political, social, religious, moral and legal implications (2).

Abortion has been and is still used as a method of fertility control or back up to contraceptive failure. It is also related to inadequate family planning knowledge and services (3).

The incidence of abortion worldwide varies from 32 abortions per 1000 women to 46 per 1000 women in women aged 15-44 years (4).

It is estimated that 50 million abortions are performed each year of which 20 million are unsafe and take place in the developing countries where risk of death is estimated at 1 out of every 280 procedures (3).

Up to 60% of total gynecological emergency admissions to Kenyatta National Hospital are due to abortions (6) and 62% of those admissions are likely to be induced (7).

There are several etiological factors of spontaneous abortions, which are broadly classified, as fetal or maternal (1). Fetal causes are mainly genetic and include chromosomal abnormalities, trisomy, polyploidy and other abnormalities. Maternal causes are classified into infections, hormonal, immunological, anatomical or systemic diseases. Abortions are clinically divided into threatened, inevitable, incomplete.

complete, septic and missed Threatened abortion-in this case there is bleeding but the cervix remains closed. Management is bed rest and mild sedation. Inevitable abortion-in this case there is bleeding, pain and cervical dilation. Management is as incomplete abortion.

Incomplete abortion-in this case some products of conception have passed through the cervix. Management is dilation and curettage using either sharp or suction curettage under syntocinon infusion. Complete abortion-in this case the conceptus is expelled completely and management is observation. Missed abortion -in this case there is fetal death but the pregnancy is retained. Management is dilatation and curettage after DIC is ruled out especially if the pregnancy has been retained for more than 4 - 5 weeks after the fetal death. Septic abortion -in this case there is sepsis and management involves broad spectrum antibiotics followed by a D & C.

Complications of abortion include haemorrhage, sepsis and its sequelae, perforation, choriocarcinoma and injury to the bowel and / or bladder (1)

In Kenya, abortion is legally restricted leading to unsafe procedures by untrained providers and hence, high mortality and morbidity. Legalization reduces maternal mortality due to reduction of unsafe abortions (Abortion should be treated as an issue of health and welfare as opposed to one of crime and punishment). Because abortion is illegal, women suffering complications delay seeking medical help. Unsafe abortions have broad and long-term health and social implications. It is associated with long-term effects such as infertility, special and psychological effects and even loss of the mother **(8).**

Treating abortion complications consumes plenty of scarce resources in terms of time, hospital beds, medical personnel and medical supplies. It is estimated that direct costs range from USS 15 to US$ 67 in Kenya (9).

C. K presented with incomplete abortion and luckily came before life-threatening complications set it and vacuum aspiration was done. Manual vacuum aspiration is safe simple and effective in treating incomplete abortion (10). It was introduced in Kenya in 1987 (9).

Maternal mortality arising from unsafe abortions can be tackled by preventing unwanted pregnancies through sex education and contraception, legalizing abortion and improving treatment and post abortal care.

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GYNAECOLOGY CASE NO. 4

RUPTURED TUBAL ECTOPIC PREGNANCY-SALPINGECTOMY

NAME:	P.W	DOA:	20.07.2004
AGE:	28 YEARS	DOD:	24.07.2004
PARITY:	1+0	LMP:	02.062004
		GBD:	7 WEEKS.

PRESENTING COMPLAINT

P.W was admitted in the acute gynaecology ward with amenorrhoea of 7 weeks and lower abdominal pains for 3 days.

HISTORY OF PRESENTING COMPLAINTS

She was well until 3 days prior to admission when she developed sudden lower abdominal pains which were more pronounced on the right side. The pain was aggravated by movements. She had no urinary symptoms, no per vaginal bleeding or abnormal vaginal discharge. There was no history of trauma. She did not have fever, vomiting or diarrhoea. She also noted that her periods had delayed for 3 weeks from the expected date. She was not on any contraception.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was a para 1+0 with her last delivery being in hospital in 1998 with no complications. Prior to the above complaints her periods were regular lasting 2 to 3 days with a cycle of 28 days. There was no associated dysmenorrhoea. Her menarche was at 14 years and she gave no history of contraceptive use.

PAST MEDICAL AND SURGICAL HISTORY

This was non contributory.

FAMILY AND SOCIAL HISTORY

She was a single lady in good relationship with the boyfriend. She was self employed. She neither smoked cigarettes nor drank alcohol. She did not have any chronic illness nor was there a family history of the same.

GENERAL EXAMINATION

She was a young lady in fair general condition. She was moderately pale but with no jaundice, cyanosis or edema The blood pressure was 90/50mmHg, pulse rate 115 beats per minute of reduced volume but regular, respiratory rate of 20 per minute and a temperature of 36 6 C.

ABDOMINAL EXAMINATION

The abdomen was slightly distended and moved with respiration. It was soft with tenderness over the suprapubic region and right iliac fossa. No masses were palpable and there was no hepatosplenomegaly. Paracentesis done was positive for no-clotting blood

PELV IC EXAMINATION

The external genitalia was normal. Digital examination revealed a cervix which was closed and posterior There was tenderness elicited on cervical motion bilaterally. The uterus was bulky. The pouch of Douglas was full. A tender mass was elicited in the right adnexa on bimanual examination. The left adnexae was free and there was a mucoid discharge on the examining finger.

The respiratory, and the central nervous systems were essentially normal.

DIAGNOSIS

A diagnosis of ruptured ectopic pregnancy was made

INVESTIGATIONS

PCV: 24%

Blood for Group and Cross match

MANAGEMENT

P W was informed of the diagnosis and explained to the need for emergency operation An intravenous infusion of normal saline was set up. .An informed consent was obtained. Blood was taken for group and cross match and 2 units of blood was made available. She was pre-medicated with intramuscular atropine, 0.6mg and then wheeled to theatre.

INTRA-OPERATIVE FINDINGS

She was put in supine position and general anaesthesia induced. She was re-positioned in semi-lithotomy and vulvo-vaginal toilet done. A urethral catheter was introduced aseptically and concentrated urine approximately 60mls was drained The abdomen was opened via a pfannestiel incision. Inspection revealed; Haemoperitoneum of approximately 2 litres and a right ruptured ampullary ectopic pregnancy. The right ovary and the left tube and ovary were grossly normal. Right salpingectomy was done. The excised portion of the tube and its contents were taken for histopathology. Haemoperitoneum was drained and peritoneal toilet done using normal saline. The appendix, liver and spleen were inspected and found to be normal Instruments swabs and needles were counted and found to be correct. The abdomen was closed in anatomical layers. The patient was reversed from anaesthesia uneventfully.

POST-OPERATIVE CARE

She was wheeled to the recovery room where vital signs were monitored continuously until she was fully awake. She was put on crystalline penicillin 2 mega units 6 hourly, gentamicin 80mg 8 hourly and flagyl 500mg 8 hourly. She was put on intramuscular pethidine lOOmg 8 hourly.

On the 1st post-operative day she was up and about. Bowel sounds were heard and she was started on oral sips. On the 2nd post-operative day she was started on light diet and put on oral Amoxil 500mg 8 hourly, flagyl 400mg 8 hourly and ponstan 500mg 8 hourly. On the 3rd post-operative day the wound was opened and found to be clean. She was on normal diet and she was discharged on the 4th post-operative day to be seen in the Gynecology out-patient clinic in two weeks.

FOLLOW UP.

She was seen in the gynecology clinic in two weeks and she had no major complaints. The wound had healed well. The histopathology results showed features consistent with ectopic pregnancy. Family planning was discussed and she opted for Norplant implant. She was advised to attend the family planning clinic.

DISCUSSION

P.W was a 28 year old para 1+0 who had a right ruptured ampullary ectopic pregnancy. Laparotomy was done with removal of the ectopic pregnancy and excision of the affected tube She recovered uneventfully.

The term ectopic pregnancy refers to gestation in which the blastocyst implants in a site other than the endometrial cavity (1,2).

The true incidence of ectopic pregnancy is difficult to determine accurately due to difference in population groups studied with different risks factors. The incidence varies with race and socioeconomic factors. In the United States the incidence is reported to vary between 0.25 % and 1.4 % of all pregnancies with an average of 1 %. The incidence for non-white women was higher in every age category than for whites with an overall 1.4 times increased risk for non-white women compared to white women (1,2). In Jamaica, the incidence is reported in 1 in 28 deliveries (3), while in Kenyatta National Hospital, Webala reported an incidence of 1 ectopic pregnancy for every 15 full term pregnancies (4). Mwathe found that 4-5 patients are admitted with ectopic pregnancy every week in Kenyatta National Hospital (5).

The incidence is increasing worldwide, for example in the USA, between 1970 and 1989, the rate has increased five-fold (6). This has been attributed to the increased incidence of sexually transmitted diseases and pelvic inflammatory disease, the efficacy of modem therapy for pelvic inflammatory disease which in the past would have resulted in complete tubal occlusion and sterility, the widespread use of intrauterine contraceptive devices, an improved method of diagnosis and reporting and an increase in assisted reproductive technology (1,3,7,8). After an ectopic pregnancy, there is a 7-13 fold increase in the risk of a subsequent ectopic pregnancy. The chance that the subsequent pregnancy will be tubal is 10-25 % (9).

Despite the significant rise in incidence, the mortality rate due to ectopic pregnancy has decreased due to early diagnosis and intervention (7,8). However, it remains one of the main causes of maternal mortality. In the USA, 15 % of all maternal deaths are attributed to complications of ectopic pregnancy (9). In Kenya Makokha found that ectopic

pregnancy caused 5.1 % of all maternal deaths at Kenyatta National Hospital between 1978 and 1987 (10).

Ectopic pregnancy is commonly found in the oviduct in 95 % of cases, and over 75 % are diagnosed before the 12^{th} week of gestation (1,2). The ampulla is the commonest site with approximately 55 % of tubal pregnancies, isthmus-25 %, infundibulum and fimbriae-17 % and interstitial segment 2-4 % (1,2). Our patient had ampullary ectopic pregnancy. Other sites include; the ovary, abdomen, broad ligament, rudimentary horn of a bicornuate uterus, the cervix, vagina and the myometrium all accounting for the remaining 5 %. Webala at Kenyatta National Hospital found that 61-64 % of the ectopic pregnancies occurred in the distal 2/3 of the tube (4). Ectopic pregnancy occurs more to the right side than the left side probably due to local influence of appendicitis (4). Our patient had a right ectopic pregnancy.

Other rare forms of ectopic pregnancies include heteretropic pregnancy which occurs when there is co-existing intrauterine pregnancy and ectopic pregnancy. The incidence is 1 in 15,000-40,000 spontaneous pregnancies and up to 1 % of patients undergoing in vitro fertilization (9). Multiple ectopic pregnancies have also been reported though they occur less than heterotopic gestation. Pregnancy after subtotal hysterectomy may occur because the patient has a cervical canal that may provide intraperitoneal access. It could also occur during the perioperative period after hysterectomy secondary to a vaginal mucosal defect that allows sperms into the abdominal cavity (9).

The primary causes of ectopic pregnancy include conditions that prevent or impede passage of a fertilized ovum through the uterine tube. These include; chronic salpingitis, adherent folds of tubal lumen due to salpingitis, isthimica nodosa, congenital anomalies of the tube, abnormal tube anatomy due to DES exposure in utero, previous tubal or pelvic surgery, tubal ligation, conservative management of unruptured tubal pregnancy, extrinsic adhesions, pelvic tumours, endometriosis, excessive tubal length or tortuosity, tubal spasms or inadequate peristalsis (1,2,3,7). Webala found evidence of choric salpingitis in 69 % of cases at KNH (4).Ovarian factors predisposing to ectopic pregnancy include; Fertilization of unextruded ovum, ovum transmigration post-midcycle ovulation and fertilization and treatment with ovulation induction drugs such as

clomiphene citrate (1,3). Zygote anomalies such as chromosomal anomalies and neural tube defects are also risk factors for ectopic pregnancy (1,3). Exogenous hormone administration such as progesterone only pill and use of IUCD'S containing progesterone have been shown to increase the risk of ectopic pregnancy Progesterone is thought to act by decreasing ciliation and cell height in the tubes (2,3). Other risk factors include; maternal age, (highest rate occurring in women aged 35-44 years), tubal abortion and subsequent implantation, any form of intraperitoneal bleeding and assisted conception techniques such as in vitro fertilization and embryo transfer (1,3,7, 8, 9). Currently cigarette smoking has been associated with more than two fold risk of tubal pregnancy (9).

In the pathophysiology of tubal pregnancy, the fertilized ovum promptly burrows into the epithelium of the tube with limited resistance for the trophoblast and at the same time, maternal blood vessels are opened (7). The embryo or fetus is often stunted. The uterus undergoes some element of early pregnancy changes. These changes include enlarged epithelial cells, with hypertrophic hyperchromatic, lobular and irregularly shaped nuclei. The cytoplasm is vacuolated, roomy with occasional mitosis. These changes in the endometrium-'Aria Stella' reaction are not specific for ectopic pregnancy and may occur in normal pregnancy (2).

50 % of all ectopic pregnancies may abort, get absorbed, mummify or become chronic (7). Rupture is usually spontaneous. Isthmic pregnancies tend to rupture at 6 to 8 weeks gestation, due to the small diameter of this portion of the tube. Ampullary pregnancies rupture later generally 8-12 weeks. Interstitial pregnancies are the last to rupture, usually at 12-16 weeks, as myometrium allows more room to grow than the tubal wall (1).

No specific symptoms or signs are pathognomonic for ectopic pregnancy. The patient may or may not have symptoms pointing to pregnancy and she may not have a period of amenorrhoea (1,3,7). Thus a high index of suspicion is very important for the diagnosis of ectopic pregnancy Lower abdominal pain or pelvic pain as was seen in our patient is the commonest symptom and occurs in 99 % of cases. It is often present even before rupture. The pain may be caused by distension of the tube and separation of the layers of muscle by the blood but more severe pain is due to the presence of blood in the peritoneal cavity.

The pain may be generalized, unilateral, sub diaphragmatic or sharp shoulder pain from irritation of the diaphragm (1,3). Abnormal uterine bleeding occurs in 75 % of the women irrespective of the site and may be mistaken for late onset menses. The bleeding is scanty and results from sloughing of the decidua. This was not there in our patient. The classical picture of ectopic pregnancy is a triad of abdominal pain, amenorrhoea and irregular vaginal bleeding (1,3). A rough working rule is that, if a patient who is few weeks pregnant complains of a little pain and heavy vaginal bleeding, the pregnancy is probably intrauterine, whereas if she has much pain and little bleeding, it is more likely to be ectopic (1). Syncope may occur in 37 % of cases (1).

The commonest physical sign is abdominal tenderness often with rebound tenderness. Cervical excitation may also be present. About 50 % of patients will have an adnexal mass, and in the majority the uterus is normal in size. Blood pressure and pulse correlate with the amount of hemoperitoneum. Most patients are afebrile unless they have a concomitant infection. This aids in the differential diagnosis from acute PID in which fever is present (1). Our patient had hypotension with tachycardia, abdominal tenderness, cervical excitation was positive and an adnexal mass was appreciated. The major gynecological conditions that mimic ectopic pregnancy are, ruptured or twisted ovarian cyst, acute PID and tuboovarian abscess. Others include uterine abortion, urinary tract infections, degenerating fibroids and normal intrauterine pregnancy (1,2,3).

Investigations that may aid in the diagnosis of ectopic pregnancy include;
Biochemical tests which include pregnancy test, to detect Beta-hcg. Most routine urine pregnancy tests have a sensitivity equivalent to serum Beta-hcg of 500-750Miu/ml and are positive on 50-80 % of cases. A negative test however, does not rule out ectopic pregnancy.

Serum progesterone levels may also be used to rule out ectopic pregnancy with those with levels greater than 24ng/ml seen in less than 2 % of ectopic pregnancies and in less than 4 % of abnormal pregnancies. A progesterone level less than 15ng/ml is seen in 80% of ectopic and 90 % of abnormal intrauterine pregnancies and 11 % of normal pregnancies.

Alfa-fetoprotein and serum amylase may be elevated.

Transvaginal sonography can detect an intrauterine gestational sac by 33 days gestation and 6 weeks gestation by trans-abdominal sonography. Vaginal sonography in diagnosing tubal ectopic has a sensitivity of 96 % and specificity of 99 %. A combination of pregnancy test and sonography gives a positive predictive value in 95 % and a negative predictive value in 100 %.

Laparoscopy is now the gold standard in diagnosis of early unruptured pregnancy. It is advantageous in that it can also be used as the definitive management of early ectopic pregnancy.

A haemogram may show a leucocytosis. Culdocentensis and paracentensis are useful in cases of intraperitoneal bleeding. Culdocentensis reveals non-clotting blood in 95 % of cases. In our patient paracentensis was done and revealed non-clotting blood. Culdoscopy and posterior colpotomy have been used but are associated with other risks. Dilatation and curettage shows curettings without chorionic villi.'Arias Stella ' reaction may be demonstrated.

The definitive management of tubal pregnancy is either surgical or medical Shock should be urgently treated in cases of ruptured ectopic pregnancy as was done in our patient. Intravenous fluid infusion should be commenced immediately and blood taken for group and cross match. In life-threatening situations immediate control of further bleeding by laparotomy is called for. Auto-infusion of blood from the peritoneal cavity can also be done.

Surgical treatment may be conservative or radical. Conservative surgical treatment may be either salpingostomy, segment resection and anastomosis or fimbria expression(l 1). Conservative management should be attempted for all suitable cases, normally unruptured isthmic or ampullary tubal pregnancy in a patient desiring future fertility The tubes can be accessed by laparoscopy or laparotomy Laparoscopic reconstructive surgery is now the gold standard for the management of ectopic pregnancy in a hemodynamically stable patient. It should be used for unruptured tubal pregnancy less than 5cm in diameter

and not in the cornua (11). Uncontrolled bleeding calls for immediate laparotomy. Radical treatment includes salpingectomy as in the case of our patient, or salpingoopherectomy The rate of repeat ectopic pregnancy is similar for both radical and conservative surgery but the rate of intrauterine pregnancy is higher following conservative surgery.

Non-surgical treatment involves the use of systemic drugs such as methotrexate and actinomycin D or local administration of drugs such methotrexate, potassium chloride, hyperosmolar glucose, mifepristone, PGF2a or PGE2 (12). Systemic methotrexate has been the most used drug with a single dose of 50mg/m2 given intramuscularly. Selection criteria for methotrexate treatment are:

a. A hemodynamically stable patient
b. No evidence of tubal rupture or significant intraabdominal hemorrhage
c. Tube less than 3-4 cm in diameter
d. No contraindication to methotrexate
e. Patient's availability for follow-up.

With this criteria, one expanded clinical trial showed that 92.4% had complete resolution with treatment and 5.8% required surgical management of the ectopic pregnancy while 3.3% required a 2nd course of methotrexate (13). Methotrexate lOmg injected directly into the ectopic gestational sac under trans vaginal ultrasound guidance has also been successful in 83% of cases (14).

Antin D immunoglobulin should be given to rhesus negative mothers (2). Post-operatively, serum Beta-hcg should be measured weekly following conservative surgery Conception rate following ectopic pregnancy is about 60% with only half of them resulting in live births. In vitro fertilization offers hope for some patients who have been rendered infertile after ectopic pregnancy (8).

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GYNAECOLOGY CASE No. 5

UNILATERAL BARTHOLIONS ABSCESS - MARSUPIALIZATION DONE

Name	N.K	IP. No	0998666
Age	60 years	D.O.A	07/07/04
Parity	8 + 0	D.O.D	10/07/04

PRESENTING COMPLAINT

The patient presented with a swelling of the vulva for 2 months.

HISTORY OF PRESENTING COMPLAINT

She was well till 2 months prior to admission when she developed right labial swelling. The swelling was of gradual onset and progressively increased. There was no pain. She had backache She had no vaginal discharge or bleeding. There was no dysuria, urinary frequency or urgency.

OBSTETRIC AND GYNAECOLOGIC HISTORY

The patient was a para 8+0. Her last delivery was in 1980. All her deliveries were by spontaneous vaginal and all her children were alive and well. She was post-menopausal for 10 years.

PAST MEDICAL AND SURGICAL HISTORY

She had been on radiotherapy treatment for cancer of the cervix stage **IIIB** until January 2004. She had no history of sexually transmitted diseases.

FAMILY AND SOCIAL HISTORY

She was married, a peasant farmer in Thika. She did not smoke cigarettes or take alcohol. There was no family history of chronic illness.

PHYSICAL EXAMINATION

General Examination

She was in good general condition. She was not in obvious pain. She had mild palour, no jaundice, no oral thrush and no lymphadenopathy. Her blood pressure was 110/90 mmHg, pulse rate 88 beats per minute, respiratory rate 18 breaths per minute and temperature 36.5° C.

Respiratory, cardiovascular and central nervous systems

These were essentially normal.

^Abdominal Examination

"The abdomen was scaphoid and moved with respiration. There were no areas of tenderness and there were no palpable masses.

Pelvic Examination

There was a small, non-tender, cystic swelling on the right labia majora and minora. Speculum examination revealed normal vaginal walls and there was no vaginal discharge The cervix had a small lesion and the uterus was normal in size and mobility.

DIAGNOSIS

A diagnosis of unilateral Bartholin's abscess recurrent cancer of the cervix was made

MANAGEMENT

The patient was informed of the diagnosis and mode of management. She gave an informed consent. She was booked for theatre the next morning. For analgesia she was given intramuscular pethidine lOOmg. She was premedicated with intramuscualr atropine 0.6 mg half an hour before theatre.

Investigations done

1. Haemogram

Haemoglobin		11.7 g/dl
WBC	-	4 3 X 10^9/dL
RBC	-	4.18 X 10^9dL
Platelets	-	158x10^9/dL

2 Urea and electrolytes

Na+	-	137 mmol/1
K+	-	4.3 mmol/l
BUN	-	5.9 mmol/1
Great.	-	98 umol/1

In theatre she was put in supine position and general anaesthesia induced. She was then repositioned in lithotomy position, cleaned and draped. Examination revealed right bartholin's cyst. A linear incision was made at the junction of the mucus epithelium and the keratinized epithelium.

Approximately 50mls of pus was drained The edges of the incision were marsupialized using 2/0 chromic catgut. No active bleeding was seen. General anaesthesia was reversed uneventfully and she was wheeled from theatre. Vital signs were observed half hourly until she was fully stable and then 4 hourly. She was discharged home on amoxicillin, flagyl and ibuprofen and booked for radiotherapy treatment. She was to come for a review in the clinic in two weeks time.

FOLLOW UP

She was seen in the clinic after 2 weeks where she was found to have no major complaints. The cyst had not recurred and the site of incision had healed. She was discharged from the clinic.

The pus swab reported growth of coagulase negative Staphylococcus spp sensitive to suprapen, augmentin, ceftazidime and amoxil.

DISCUSSION

The patient presented was a 60 years old para 8+0 postmenopausal for 10 years admitted with unilateral Bartholin's abscess. Marsupialization was done under general anaesthesia.

The Bartholin's glands are a pair of small compound structures situated beneath the vestibule just outside the lateral margins of the vaginal orifice. These glands produce a colourless mucoid material in response to sexual excitement.

Obstruction of the main duct of the Bartholin's gland results in retention of secretions and cystic dilation. The glands may harbour micro-organisms that cause inflammation within the duct and gland lining which may eventually result in suppuration and abscess formation. Bartholin's abscess may follow primary infection of the gland or follow duct obstruction. The cause of duct obstruction include: infections and trauma e.g. during mediolateral episiotomy or posterior colporrhaphy. Other possible causes are inspissated mucus or congenital narrowing of the duct " .

The incidence of Bartholin's abscess at KNH has been reported to be 1.9 % and most infections occur between 20 and 50 years of age with a mean of 22.5 years[4] Mumia in his study at KNH found that 1.7% of total admissions to the emergency gynaecological

'ward were Bartholin's abscess. He found that 50% of the patients were aged between 18 and 23 years[5].

The patient presented was 60 years of age though.

The main organisms that cause infection in the Bartholin's gland are Neisseria gonorrhoea, E. Coli, staphylococcus and Trichomonas vaginalis[4 6] Our patient had staphylococcus species infection.

Most Bartholin's cysts are asymptomatic. Two types of cysts are identifiable by microscopyductal cysts and gland cysts as seen by the lining epithelium. Bartholin's cysts usually are small and symptomatic. Diagnosis is usually made during routine pelvic examination. Symptoms may occur if the gland gets secondary infection or if the gland grows rapidly. They include pain while sitting or walking and dyspareunia. In the acute phase of the infection, the surrounding tissue are oedematous and iflamed. On

examination, a tender fluctuant swelling in the labia minora is usually palpable [7].

Mumia at KNH found that almost all patients presented with vulval swelling and difficulty in walking or sitting and all had tenderness and fluctuance of the swelling [5]

Our patient did not have pain but had a rapidly swelling labial mass.

Like all abscesses, management of Barholin's abscess is mainly surgical drainage. To avoid recurrence of abscess, marsupialization is done. Marsupialization involves making a wedge- shaped incision in the vaginal mucosa over the center of the abscess outside the hymnal ring. The abscess wall is then opened and the contents drained. The lining of the abscess is then everted and approximated to vaginal mucosa with interrupted 2/0 delayed absorbable sutures. This ensures that the stoma remains patent postoperatively. The procedure can be performed under local, regional or general anaesthesia. The patient should be given appropriate antibiotics and analgesics for pain. Sitz baths should be started on the third or fourth postoperatively day.

Our patient was done marsupialization under anaesthesia and put on antibiotics and analgesics.

The other method of treatment of Bartholin's abscess or cyst is by insertion of a Word catheter This accomplishes the same effect as surgery with minimal or no trauma. In this, a small incision is made with a No. 11 pointed blade at approximately the area of the normal orifice under local anaesthesia. The word catheter is inserted through this opening

^nd the bulb distended with approximately 2mls to 3mls saline. The nipple of the catheter is inserted into the vagina. The catheter is then removed in 3 to 4 weeks by which time epitheliazation of the orifice has taken place so that the reclosure of the duct is unlikely. Coitus may be resumed soon after insertion of the catheter[2,3].

Recurrence of Bartholin's abscess is common especially if simple incision and drainage is done [2,3,7] Marsupialization is associated with recurrence rate of 10 -15% and this occurs secondary to fibrosis of the orifice[3]. In our set up, Mumia found a recurrence rate of 15% with 50% of these occurring in the first year.

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IMPERFORATE HYMEN-HAEMATOCOLPOS AND HAEMATOMETRA: CRUCIATE INCISION.

Name : **M.W** DOA: 08.12.2004

IP No.: 0998035 DOD: 13.12.2004

Age: **14 years**

Ward ID

Presenting complaints

M. W presented with abdominal pain for 3 days, inability to pass urine for one day and constipation for three days.

History of presenting complains

She was well until 3 prior to admission when she developed insidious abdominal pains initially intermittent but later constant and increasing in intensity. She also developed constipation for 3 days. One day prior to admission she developed urinary retention and was admitted in casualty with acute urinary retention.

Obstetric and gynaecological history

She was a para 0+0 and she had not received her menses.

Past medical **history**

This was not significant

Family and social history

She was a standard 5 pupil and lived in Baba Dogo with her parents. There is no family history of chronic illness

General examination

She was a young girl in fair general condition but in pain. She was not pale or jaundiced. Her temperature was 37.0 C, blood pressure 100/65mmHg, pulse rate of 77 beats per minute Breasts were tanners stage III with no discharge from the nipples.

Abdominal **examination**

There was mild infraumbilical distension of the abdomen and was moving with aspiration There was infraumbilical tenderness with guarding. A mass was felt corresponding to 12 weeks gestation arising from the pelvis. There were no any other masses felt. She had pubic hair stage III.

Pelvic examination

There was normal external genitalia. There as an imperforate hymen that was bulging with brownish discoloration. She had a urethral catheter which was draining clear urine. The other systems were essentially normal.

Diagnosis

A diagnosis of haematocolpos secondary to imperforate hymen was made.

Management

She was admitted into ward ID and informed of the diagnosis. The parents were also informed and a written consent was obtained for cruciate incision under general anaesthesia.

Investigations done

PCV 36%

Na~- 130 mmol/1

KT - 3.8 mmol/1

Creatinine - 65 umol/l

Pelvic Scan

The uterus was enlarged with fluid suggestive of blood. The right ovary was normal and the left ovary was not visualized. Vaginal canal was distended with fluid. Conclusion: Haematometra with haematocolpos

Management.

She was premedicated with atropine 0.6mg half hour before being taken to theatre. She was put in supine position and induced with general anaesthesia. She was re-positioned in litothomy position cleaned and draped. Urethral catheterization obtainedlOOmls of clear urine. Vaginal examination revealed an intact hymen which was opened via a cruciate incision.

Approximately 800mls of dark brown chocolate altered blood was drained. The hymenal ring was felt with digital examination using the small finger. The uterus was well contracted. General anaesthesia was reversed uneventfully. She did well post-operatively and was discharged on the 1st postoperative day on Amoxil, flagyl, brufen and betadine vaginal douches twice daily.

Discussion

M.W presented with haematocolpos and haematometra secondary to imperforate hymen Cruciate incision was done with favorable outcome.

Imperforate hymen is the most frequent of the obstructive anomaly of the genital tract with frequency varying from 0.01 to 0.1%. Ma Cann et al examined 93 girls aged between 10 months and 10 years and found 1 child (1.2%) with imperforate hymen and 2 children (2.5%) with hymenal septa (1).

During embryology of the urogenital tract, the cloaca is divided by the urogenital septum into the urorectal canal and the urogenital sinus. The anorectal canal is the predecessor of the rectum and anus.

The urogenital canal forms the uterus and the vagina. The upper portion of the urogenital sinus forms the upper vagina, uterus, fallopian tubes and the inferior portion form the vestibule. These two are normally separated by a membrane (hymen). Which is a proliferation of the sinovaginal bulb. This normally becomes perforate before birth failure of which imperforate hymen occurs (2).

There is a familial occurrence of imperforate hymen hence the need for screening of siblings. Dominant transmission or a recessive mode have been described (3). Diagnosis of imperforate hymen usually occurs at menarche. Prior to menarche imperforate hymen can be diagnosed by the presence of a mucocolpos presenting as a yellow -grey mass at the introitus with an associated abdominal mass. Urinary symptoms may be present.

Occasionally diagnosis has been made in utero using obstetric ultrasonography (1). Diagnosis is usually made at menarche with the typical findings being haematometra (blood within the uterus) haematocolpos (Blood within the vagina). Occasional there may be haematosalpinges with signs of retrograde menses occasionally to the point of intra-abdominal endometriosis and severe adhesions (4).

M.W had intermittent abdominal pains and an abdominal mass.

Differential diagnosis of imperforate hymen include labial adhesions, vaginal septa.vaginal agenesis and androgen insensitivity. Diagnosis is usually by examination during which the hymen is visible as a thin, membrane that bulges with valsalva

maneuver. If there is haematocolpos a brownish color is seen. Occasionally a pelvic mass may be found. Diagnosis may be confirmed by pelvic ultrasound which will rule out the other abnormalities (4).

Occasionally MRI may be used if the diagnosis is not clear (5). Treatment of imperforate hymen is surgical. A diagnostic technique of needle aspiration should not be used as it may introduce infection leading to change of sterile haematocolpos or haematomentra to mucocolpos or pvometra (4).

Two modes of surgical therapy can be used. These are cruciate incisions in diagonal diameters to avoid urethral injury with removal of excess hymenal tissue and the vaginal mucosa is sutured to the hymenal ring to prevent adhesions and recurrence of the obstruction. The second is hymenectomy. Post operative treatment includes NSAID's or local anaesthetic jelly.

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GYNECOLOGY CASE No. 7

ENDOMETRIAL HYPERPLASIA -TOTAL ABDOMINAL HYSTERECTOMY AND BILATERAL SALPINGO-OPHORECTOMY

NAME	**G.N**	**DOA: 23.09.2004**
SEX	**FEMALE**	**DOD: 04.10.04**
AGE	**65 YEARS**	**PARA: 8+0**
IP NO	**0984295**	

PRESENTING COMPLAINTS

G.N Was admitted with complains of postmenopausal bleeding for one month and lower abdominal pains for 6 months

HISTORY OF PRESENTING COMPLAINTS

G.N Was well until 6 months prior to admission when she developed lower abdominal pains on and off. The pain was dull not associated with any vaginal discharge or urinary symptoms . A month before admission she developed per vaginal bleeding which was initially in form of spotting but later was copious and in clots

OBSTERIC AND GYNAECOLOGICAL HISTORY

She was postmenopausal for 8 years She was para 8+0 with the last delivery being in 1970 All her deliveries were SVD and the children were all alive and well

Her menarche was at 15 years she had not used any form of contraception

PAST MEDICAL AND SURGICAL HISTORY

She had appendicectomy done in 1995

She was also a known hypertensive for the previous 9 years prior to admission and had been on capozide 50mg and Atenolol 50mg once a day and the blood pressure was well controlled

FAMILY AND SOCIAL HISTORY

She was a housewife and her husband was a farmer. She did not drink alcohol nor smoke cigarettes .There was no maternal family history of diabetes or hypertension.

GENERAL EXAMINATION

She was an elderly lady in fair general condition She was not pale or jaundiced with no edema Her blood pressure was 135 /85 mmHg, pulse rate 78 beats per minute, temperature 36.8°C and a respiratory rate of 16 per minute Her weight was 82 Kilogram's and a height of 5 feet 4 inches .

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moving with respiration.There was a right paramedian lower abdominal scar due to previous appendicectomy. The abdomen was soft. No masses were felt

PELVIC EXAMINATION

There was normal external genitalia. The cervix was posterior, short and atrophic and the os was closed The uterus was bulky and mobile The pouch of Douglas and adnexa were free.

The other systems were essentially normally.

DIAGNOSIS

Postmenopausal bleeding to rule out Uterine fibroids, endometrial hyperplasia, adenomyosis or endometrial cancer.

INVESTIGATIONS DONE

Pap smear :	Normal squamous and endocervical cells seen
Endometrial:	
Biopsy	Sections revealed endometrial tissues cystically dilated and sparse glands with compact stroma. There were no features of atypia or endometritis. Conclusion: Low grade cystic endometrial hyperplasia.

Pelvic Ultrasound: Uterus is anteverted (83mm x 48mm). Prominent endometrium (27mm). No fibroids seen. Conclusion: Marked endometrial hyperplasia.

Haemogram :- Hb -13.Og /dl , W.B.C- 8.8 x 10/L , R.B.C - 4.2 x 10 fL
Platelets -150 x 10/L, PCV- 40.2%
MCV- 95.2 fl.
Lymphocytes- 43
Monocytes-1
Eosinophils-3
Basophils-1

Urea & electrolytes : Na+ 138 mmol /L
K+ 4.4 mml/L
CL 97 mmol/L L
Urea 6.27 mmol/L

She was informed of the diagnosis and was counseled to undergo total abdominal hysterectomy and bilateral salpingo- ophorectomy and she signed a written consent and was pre-medicated with atropine 0.6 mg I.M and pre-medicaeted with pethidine 50 mg I VI Vi hour before being taken to theatre She was shaved and starved from midnight and 10 mg dulcolax given the night before operation

OPERATIVE MANAGEMENT

In theatre anaesthesia was induced, she was put in semi-litothomy position and VVT done Catheterization yielded 300 mis of clear urine. The vaginal canal and cervix was painted with Betadine paint

She was put in supine position, the abdomen was cleaned and draped then opended via a pfannestiel incision .

The uterus was found to be bulky and uniformly enlarged

Both ovaries were identified and they appeared grossly normal Total abdominal hystrectomy was done as described in the introduction pages of this book. The uterus and ovaries were taken for histology Instrument and swab counts were confirmed to be

correct and the abdomen was closed. Estimated blood loss was 400mls, anaesthesia was reversed successfully.

POSTOPERATIVE

She was put on IV Fuids normal saline to alternate with 5% dextrose, I V X-pen, gentamicin and flagyl with I.M pethidine.

On the 1st post- operative day she was started on oral sips.On the 2nd post-operative day she was started on light diet and oral medications. On the 4Ih post operative day the wound was exposed and was found to be clean. She was discharged home to be reviewed in the out- patient clinic in three weeks.

FOLLOW-UP

After 3 weeks she was reviewed in the gynaecology out patient clinic and she was in good general condition. The wound had healed well.

Histology report

Uterus and cervix and ovarian tissues are seen and both ovaries are normal.

Uterus shows cystic endometrial hyperplasia

She was discharged from the clinic for follow - up after six months.

DISCUSSION

Presented is G.N a 65 year old lady with post-menopausal bleeding who was diagnosed to have cystic endometrial hyperplasia. Total abdominal hysterectomy with bilateral salpingo-ophorectomy was performed from which she recovered successfully.

Endometrial hyperplasia is the abnormal proliferation of endometrial glands and stroma (1). Incidence of endometrial hyperplasia varies; being commoner in whites 2.4% as compared to 1.3% in black women. It's peak incidence is in the 6Ih and 7th decades. The

cause is unknown but elevated exogenous or endogenous estrogen stimulation of the endometrium is usually found (2).

Patients with constitutional status such as diabetes mellitus, hypertension, polycystic ovarian syndrome and obesity are associated with increased estrogen and have higher incidence of endometrial hyperplasia.

Patients receiving estrogens such as in Turners syndrome or in gonadal agenesis or hormone replacement have higher incidences of endometrial hyperplasia (3). Pathologically endometrial hyperplasia varies from slight exaggeration of the proliferative phase to marked over growth resembling endometrial carcinoma. There has been controversy over the classification of endometrial hyperplasia (3).

Previous classification has been into cystic, adenomatous and atypical hyperplasia (3). Cystic hyperplasia is microscopically an overcrowding of the glands giving the characteristic 'swiss cheese' histology appearance. This condition normally results from excess estrogen secretion (2).

In adenomatous hyperplasia there is complex crowding of the glands little stroma and there is epithelial stratification and mitotic activity. Risk of progression to cancer is 15-30% and it is normally found in the 40 - 50 year age group.

Atypical endometrial hyperplasia is the severe form of the hyperplasia and characterized by larae irregular glands and pronounced reaction of the intervening stromal cells which have increased nuclear material. It occurs in the women of the 50 year age group Progression to invasive carcinoma is 50% if left untreated (2).

This classification was found to be complex and not standardized. The International Society of Gynecologic Pathologists under the World Health Organization has devised a uniform classifications into:

I. Simple hyperplasia - There is no cellular atypia but there is endometrial hyperplasia with un- crowded glands.

II Complex hyperplasia without atypia- The glands are complex and crowded.

III. Atypical hyperplasia- In these cases there is cellular atypia regardless of the type of hyperplasia. Incidence of this hyperplasia is 0.9 - 1.3% of endometrial hyperplasias (4).

Abnormal uterine bleeding is seen in 80% of patients. This may be menorrhagia intermenstrual or postmenopausal bleeding.

G.N presented with postmenopausal bleeding.

Simple hyperplasia may be asymptomatic or diagnosed after hysterectomy for other reasons. 10% of patients may present with dysmenorrhoea. Other risk factors may be seen during examination. This may include obesity, advanced age, polycystic ovarian disease, estrogen secreting tumors, hormone replacement or infertility.

Diagnosis of endometrial hyperplasia is usually via histology.Fractional curretage is the gold standard for diagnosis. Other methods like suction curettage, endometrial lavage, hvsteroscopy and endocervical aspiration may be used (2).

Treatment of endometrial hyperplasia depends on age, type of hyperplasia and reproduction desire of the patient.

Using the WHO classification, treatment is classified into hormonal or surgical.

Hormonal therapy consisting of progestin is used in women below 40 years with simple complex hyperplasia, those who cannot withstand surgery or still desire to conceive. Those with hyperplasia secondary to excess estrogen can benefit by simply removing the source of estrogen. Those with anovulation may benefit from progestin or ovulation induction (5).

Women above 50 years should have total abdominal hysterectomy and bilateral salpingo-ophorectomy unless there are medical reasons for not withstanding surgery then progesterone treatment with intracavitary radium may be used (2).

Those with atypical endometrial hyperplasia irrespective of age, hysterectomy is the preferred treatment. Other methods of treatment include Danazol 200mg daily or Tamoxifen 200mg daily.

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GYNAECOLOGY CASE No. 8

VESICO-VAGINAL FISTULA-SUCCESSFUL REPAIR

NAME:	G.W	DOA:	10.07.2004
AGE:	18 YEARS	DOD:	28.07.2004
IP NO:	0876596	PARA:	1+0

PRESENTING COMPLAINT

G.W presented with a I year history of effortless leakage of urine.

HISTORY OF PRESENTING COMPLAINT

G.W was a referral from Kijabe Mission Hospital with a diagnosis of vesico-vaginal fistula following difficult delivery. A traditional birth attendant (TBA) had been asked to conduct her labor and delivery. Unfortunately she was unable to deliver the baby while under the care of the TBA. Due to unavailability of immediate transport, there was approximately another 12 hours of delay. She delivered within 30 minutes of arrival to hospital to a macerated still birth via vaginal delivery. The weight of the baby could not be determined. She began to leak urine immediately after the delivery. She was put on an indwelling catheter for 3 months. When it was removed she was still leaking urine. The urine was leaking continuously and she did not have the urge to urinate. She was to undergo surgery at Kijabe Mission Hospital after removal of catheter but she opted to come to KNH. She had no history of fecal incontinence or weakness of the lower limbs

OBSTETRIC AND GYNECOLOGIC HISTORY

She was a para 1+0. Her last delivery was on 03.07.2002 as recounted above. Menarche was at 14 years. Her menses were regular every 28 days and lasting 3-4 days. Her LMP was on 25.06.2004. She did not give history of contraceptive use.

FAMILY AND SOCIAL HISTORY

She was single and lived with her sister in Narok. She had no formal education. She was unemployed. She had no history of alcohol use or cigarette smoking. There was no chronic illness in the family.

PAST MEDICAL AND SURGICAL HISTORY

This was non-contributory.

GENERAL EXAMINATION

She was in fair general condition and in good nutritional status. She had no palor. edema, jaundice or cyanosis. Her blood pressure was 110/70 mmHg, pulse rate 73 beats per minute, respiratory rate of 18 per minute and a temperature of 36.8 C.

ABDOMINAL EXAMINATION.

The abdomen was not distended and moved with respiration. It was soft and non-tender. There were no palpable masses.

PELVIC EXAMINATION

There was moderate excoriation of the vulva and perineum. It was obviously wet. Continuous dribbling of urine was noted and there was a pungent smell. Using the Sims speculum a vesico-vaginal fistula was seen in the anterior vaginal wall. It was about 2.5cm in diameter, 1.5cm from the external urethral orifice and 5cm from the cervix. There was no vaginal discharge. There was no vaginal stenosis or stricture
The respiratory, cardiovascular and the central nervous systems were essentially normal.

DIAGNOSIS

A diagnosis of vesico-vaginal fistula was made.

MANAGEMENT.

The patient was informed of the diagnosis and an explanation of the condition and mode of management given. She signed an informed consent for examination under anaesthesia and repair at the same sitting. Investigations done included;

Haemogram:	WBC	5.9 x 10/L
	Hb	11.9 g/dL
Urea & electrolytes	Na+	135 mmol/L
	K+	4.4 mmol/L
	Urea	2.7 mmol/L
	Creatinine	68 umol/L

On the pre-operative day, she was starved from midnight and started on intravenous fluids. She had enema at 6.00pm and 6.00am. on the morning of the operation, half an hour before theatre, she was pre-medicated with intramuscular atropine sulphate 0.6mg, pethidine 50mg intramuscular and intravenous gentamicin 80mg stat. She was then wheeled to theatre.

VESICO VAGINAL FISTULA REPAIR

In theatre spinal anaesthesia was given as intrathecal bupivacine solution 0.5 %. After the effect of anaesthesia was achieved, she was placed in litothomy position. Vulvo-vaginal toilet was done and she was draped with sterile towels. A piece of sterile gauze was placed over the anus. The drapes were clipped to the gluteal muscles and the labia majora everted and stitched to the thighs with a stay suture. An Auvard speculum was inserted into the vagina. Examination under anaesthesia was done and confirmed the earlier findings. Jungle juice (adrenaline, lignocaine and normal saline solution) was infiltrated around the fistula. A transverse incision was made around the fistula and the bladder freed from the vaginal wall. The fistula was then repaired in two layers using vicryl 3/0 on an atraumatic needle A urethral catheter size 16 was then inserted and inflated with 5mls of normal saline. Using the catheter the distance between the external urethral orifice and the catheter balloon was determined as 3cm, the approximate length of the urethra. Dye test was done by instilling 60ml of methylene blue into the bladder and checking for leaks at the suture line. The dye test was negative. The vaginal wall was then repaired. A gauze soaked in betadine was placed over the repair site to be removed in 24 hours. The labial sutures were removed. The Foley's catheter was sutured to the patient's inguinal region to avoid traction and mobility. It was fixed to a urine bag. The patient was then wheeled to the recovery area.

POSTOPERATIVE CARE

Vital signs were observed continuously for half an hour then quarter hourly for another hour then 4 hourly in the wards. An intravenous drip of normal saline alternating with 5% dextrose 500mls 4 hourly for 24 hours was started in theatre. The patient was also encouraged to take oral fluid freely. She was advised to have bed rest and take at least 6 litres of fluid per day. .After 24 hours intravenous fluids were stopped. The vaginal pack

was removed. The catheter was inspected regularly for drainage and clarity of urine. A fluid input-output chart was kept. This was satisfactory. On the first postoperative day she had no complaints and ambulation of the lower limbs was encouraged. On the second day she was up and about. She was also asked to maintain good perineal hygiene. On the third day she was advised on perineal exercises. Urine for microscopy culture and sensitivity was taken weekly and were found to be normal. She was not leaking urine in bed

After 14 days, she was reviewed and she had no problem. The catheter was draining clear urine. A Sim's speculum was used to expose the site of the repair and found not to be leaking any urine. Dye test was done and found to be negative. The catheter was removed. She was to continue with perineal exercises at home several times a day. She was to empty her bladder every 1-2 hours during the day and every 3-4 hours at night. She was to avoid sex for 6 months and to seek medical advise in case of dysuria. She was advised to attend antenatal clinic should she conceive and to deliver by caesarean section all future pregnancies She was asked to come to the VVF clinic after 6 weeks.

FOLLOW LP AT 6 WEEKS

She was seen as scheduled and had no complaints. The repair site was inspected and found to have healed well. An appointment to come after 6 months was given but she was lost to follow up.

DISCUSSION

Presented is G.W, an 18 year old para 1+0 who developed a vesico-vaginal fistula following a prolonged difficult delivery. She underwent a successful repair.

A fistula is a communication between two epithelial surfaces. Vesico-vaginal fistula (VVF) is a communication between the epithelial surfaces of the urinary bladder and the vagina allowing urine to find its way from the bladder into the vagina. The constant dribbling of urine, wet clothes and the accompanying smell are a social embarrassment to the affected woman (1,2). Malnutrition and neglect may also supervene (3).

The true incidence of VVF is unknown. In Africa it is estimated to be 1-2 per 1000 deliveries where the mother survives (1). At Kenyatta National Hospital (KNH) 166 cases were treated between 1979 and 1982 (4).

The etiology of VVF varies enormously all over the world. In the developed countries, it is usually a consequence of surgery, radiation and malignancy. In the United States, 85 % of VVF's follow surgery, 10 %radiotherapy and only 5 % obstetric cause, usually operative vaginal delivery (5). In developing countries, the predominant cause of VVF is prolonged obstructed labor. It accounts for over 85 % of the cases (1). In Nigeria, Tahzib found that 83% of VVF resulted from obstructed labor and only 1 % were from surgical injury (6). In KNH, Orwenyo found that 92% resulted from obstetric causes(4). Gunarantine and Mati, found that 40-80% of VVF occurred in the primigravida of whom 70% had obstructed labor with cephalo-pelvic disproportion (7). Some factors which may contribute to prolonged obstructed labor leading to VVF formation in developing countries include; Marriage and conception at a young age before full pelvic growth has been achieved, chronic malnutrition and lack of access to qualified health care professionals or medical facilities during child birth (2). Our patient conceived at a young age of 16 years. She laboured at home for two days while being monitored by a TBA. It took an extra 12 hours to arrive to a medical facility. Other causes of VVF are trauma, infection and congenital malformations (8).

The pathophysiology of VVF as a result of prolonged obstructed labor is either as a result of pressure necrosis (in majority of cases) or as a result of uterine rupture (3). During

labor the bladder is displaced upwards into the abdomen and the bladder base and urethra are compressed between the presenting part and the posterior surface of the pubis. When this pressure is unrelieved as in obstructed labor, the intervening soft tissues become devitalized by ischaemia.This results into tissue necrosis and the devitalized areas sloughs off resulting in urinary incontinence. Infection then increases the amount of tissue that sloughs off further increasing the size of the fistula (1). The use of instruments such as the vacuum extractor and obstetric forceps or destructive procedures may cause injury to the maternal tissues leading to fistula formation (1).

During caesarean section, the posterior bladder wall may be incised accidentally during lower segment incision or torn during downward reflection of the bladder again resulting in fistula formation Accidental passage of a suture through the posterior wall during the repair of the uterine incision may lead to the bladder wall tissue being held by the suture and sloughing off. In the patient presented, pressure necrosis from prolonged labor was the most probable cause of the VVF.

Diagnosis of VVF is made from clinical history and physical examination. In the history, large fistulas will present with all the urine draining through the vagina, while in smaller fistulas there is dribbling of urine per vagina intermittently with normal voiding. In the case of our patient the urine was leaking continuously without the feeling of the urge to urinate The time of initial insult to clinical presentation depends on the etiology of the VVF.

Fistulas associated with pelvic surgery are symptomatic within 7-30 days post delivery. An anterior vaginal wall laceration associated with obstetric fistulas typically presents in the first 24 hours of delivery Radiation induced fistulas may present 30 days to 3 months later (2). Our patient began to leak urine immediately after delivery of the baby.

A large fistula can be identified visually on speculum examination or by digital palpation When the fistula is small and not visualized, a dye test can be used. This involves swabbing the vagina dry and instilling a solution of methylene blue into the

bladder by means of a catheter. If the blue solution can be seen to trickle into the vagina, the diagnosis of a vesico-vaginal fistula is established. If on the other hand, urine trickles into the vagina but is not stained blue the fistula may be uretero-vaginal. In the case of our patient, the vesico-vaginal fistula was easily seen in the anterior vaginal wall. There was no need for a dye test.

Other important physical signs to look out for in patients with VVF, include general examination for features of cachexia and palor. Other lesions that may also occur in these patients include loss of labia minora, urine induced dermatitis, perineal paralysis, pressure ulcers over prominent bones and foot drop. Apart from mild excoriation of the vulva and perineum, our patient did not have any of these complications.

Once the diagnosis of WF has been established, a full vaginal inspection is essential and should include; assessment of tissue mobility, accessibility of the fistula to vaginal repair, determination of the degree of tissue inflammation, edema, infection and if possible association of a recto-vaginal fistula. Examination under anaesthesia is no longer a prerequisite before surgical intervention. This can be done at the same time of definitive surgery. Urine should be collected for culture and sensitivity and patients with positive results should be treated before surgery (2).

Other investigations that may be done include; intravenous urogram, cystoscopy and hvsterosalpingogram. These may be more important in ureteric fistulae. Haematuria during menses suggests utero-vesical fistula. This is more commonly seen secondary to pelvic surgery A full blood count and renal function tests should be done as part of pre-operative assessment of the patient. These were done for the patient presented.

There are different ways of classifying WF's. More recently, Kees Waaldjik has subdivided fistulas into three major groups depending on the anatomic and physiologic location (1).

Type I	Fistulas not involving the closing mechanism
Type II	Fistulas involving the closing mechanism
A	Without total involvement of the urethra
	a. Without a circumferential defect
	b. With a circumferential defect
B	With total urethral involvement
	a. Without a circumferential defect.
	b. With a circumferential defect
Type III	Miscellaneous e.g. uretero-vaginal and other exceptional fistulas

In our patient the fistula involved the closing mechanism, part of the urethra was involved with no circumferential defect. The fistula was classified as Type IIAa. This classification has prognostic significance with a worsening prognosis with a higher class.

Fistulas can be classified according to size;

Small:	< 2cm
Medium:	2-3 cm
Large:	4-5 cm
Extensive:	> 6 cm

Based on this our patient had a medium sized fistula as it was approximately 2.5 cm in diameter Management of VVF can be conservative or surgical Conservative management involves placement of a transurethral catheter and maintaining it for 4-6 weeks Up to 40-60 % of fistulas less than 4 cm have been shown to heal by resting the bladder this way (2). Previously surgery was only done after 3 months (as was planned for our patient at Kijabe Mission Hospital) but this has been shown to be unnecessary. Once the slough has cleared off, repair is possible with good results. However in the case of radiation induced fistula, one year interval is recommended to ensure full resolution of

tissue necrosis (1,2). In the case of pelvic surgery 6 months is preferred. Routine antibiotic cover is not advised as most of the urine specimens have been shown to be sterile Antibiotics should be used only on confirmed infection. Cachectic patients are put on a high protein diet Anemia is corrected with use of haematinics. Blood transfusion should only be used in emergency situations. Our patient did not have anemia and was in good nutritional status. Estrogen replacement therapy in patients with prolonged amennorrhoea or those who are post-menopausal may assist in optimizing tissue vascularization and healing. Sitz baths and barrier ointments such as zinc-oxide preparation can provide relief from local dermatitis. Occasionally acidification of the urine to diminish the risk of cystitis, mucus production and formation of bladder calculi may be necessary before surgery.

VVF's which do not heal spontaneously, must be treated surgically. The route of repair for VVF can be abdominal or vaginal. This depends on the surgeon's experience and type of fistula. The approach route of choice for type I and II fistula is the vaginal route. For type III fistula, the abdominal approach may be necessary (2). The vaginal route is characterized by minimal blood loss, low post-operative morbidity, shorter operative time and shorter post-operative recovery time. This was the approach of surgery used in our patient. Litothomy position was favored for the repair of this particular lesion. In some cases the knee- chest position can be used. The choice of anaesthesia is dependent on the facilities available. Spinal anaesthesia has the advantage of reduced cost, easy accessibility and the fact that the surgeon can give the anaesthesia. The repair of our patient was done under spinal anaesthesia.

The main objectives of any VVF is to close the fistula, make the patient continent and preserve and restore sexual function. During surgery the size, location and amount of fibrosis around the fistula are determined. A circumferential incision is made at the fistula edge with bilateral transverse incision in smaller fistulas. The anterior vaginal wall is widely dissected sharply and bluntly from the bladder. The bladder is mobilized sufficiently to allow tension free suturing and closure of fistula. Closure is done from the lateral margins to the middle. The fistula may be longitudinal or transverse and is closed without tension. Bladder mucosa is avoided from suture bites and the first layer is inverted with interrupted stitches. The first dye test is done and if water tight, then the

vaginal mucosa is closed with interrupted averting mattress sutures (8). These procedures were done for the patient presented.

A Foley's catheter is left in situ and stitched above the urethra for 14 days post-operatively when the patient is re-examined for leakage. Continuous bladder drainage is vital for successful repair of VVF. A large caliber catheter minimizes the potential for catheter blockage by blood clots, mucous and calcific deposits (1,2) To achieve continuous drainage of urine, the patient must drink at least 6 litres of fluid daily to maintain bladder washout. If the catheter blocks, it must either be flushed out or changed. Any delay will result in tension of the sutures and the repair may break down (1).

Post-operative analgesia is recommended, but use of antibiotics post-operatively is controversial. Many physicians administer oral antibiotic prophylaxis until the Foley's catheter is discontinued while others check closely for the development of urinary tract infection and administer antibiotic therapy when urine cultures are positive for bacterial growth (2). Our patient was put on antibiotics post-operatively as it is the common practice in our set-up.

Once the catheter is removed, the patient is instructed to refrain from sexual intercourse for 6 months following the operation. A pregnancy following successful repair mandates elective caesarean section (5). Our patient was advised accordingly. Complications of VVF repair include; haemorrhage, ureteral obstruction, breakdown of repair, vaginal stenosis and urinary incontinence (8). Our patient did not have any of these complications after the two subsequent reviews. About 80-90% of VVF should heal at the first attempt like in our patient while 10% heal after second attempt. Further attempts are usually less successful as more scar tissue is involved at each repair The overall cure rate at KNH was found to be 60%(7). Gunaratine and Mati found the highest cure rate to be with mid-vaginal fistula (85.7%) (7). Orwenyo found this to be with juxta-cervical fistula (80.6%) (4).

Mortalities associated with development of obstetric fistula as reported by Orwenyo include still birth rate of 70% with a perinatal mortality of 80%(4). Gunaratine and Mati

reported a still birth rate of 63.7% and a neonatal mortality of 60% (7). The patient presented had a still birth.

Prevention of WF in our set-up should be aimed at improvement of maternity services Health facilities offering caesarean section should be made more accessible and affordable to pregnant women. TBA's should be well trained in identifying the high risk clients and refer them promptly.

Improvement of the general socio-economic status of the population and economic empowerment of women and education to the girl child is paramount to prevention of W F . Teenage girls should be discouraged from engaging in early sexual activity and the government should enforce laws against early marriage. Our patient a teenage girl conceived at 16 years. Both her parents were peasant farmers. She had no formal education and she was unemployed. Her labor was being monitored by a TBA and the nearest health facility offering maternity services was not easily accessible. All these factors contributed to the obstetric vesico-vaginal fistula.

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GYNAECOLOGY CASE. No. 9

SYMPTOMATIC UTERINE FIBROIDS- TOTAL ABDOMINAL HYSTERECTOMY

Name:	CA	DOA. 05.01.2005
Age	47 years	DOD: 17.01.2005
IP No:	0990667	PARITY. 3+0

Presenting complaints

C. A presented with history of progressive abdominal swelling heavy prolonged menses and lower abdominal pains for the previous 10 years.

History of presenting illness

She was well until 10 years prior to when she started having heavy prolonged menses. The bleeding was in clots and was associated with dysmenorrhea. She had also noticed a progressive lower abdominal swelling over the same period but she did not complain of any urinary symptoms or constipation.

Obstetrical and gynecological history

She was a para 3+0, with the last delivery being 13 years earlier. All deliveries were S\T) but one child passed away a few days after birth due to neonatal pneumonia.
Her menarche was at 16 years. The cycles were initially regular every 30 days lasting for 3 - 4 days but changed to every 20 days lasting 4 - 7 days. There was no associated dysmenorrhea. She had not used any form of contraception.

Past medical and surgical history

This was non-contributory

Family and social history

She was widowed for the previous 10 years . She was a house help in Yaya estate. There was no history of chronic illness in the family . She neither smoked cigarettes nor drank alcohol.

General examination.

She was in fair general condition, not pale, jaundiced nor cyanosed with no lymphadenopathy. She was clinically afebrile. Pulse rate was 90 beats per minute, respiratory rate 14 breaths per minute and blood pressure was 130/70 mmHg.

Abdominal examination

The lower abdomen was distended by a mass of pelvic origin corresponding to 32 weeks fundal height. It was firm, slightly mobile, with an irregular nodular surface. There was no tenderness and there was no other organomegaly.

Pelvic **examination**

There was normal external genitalia. The cervix felt smooth and healthy with the os closed. A pelvic mass of uterine origin was felt, and was slightly mobile. The pouch of Douglas and both adnexae were free. There was a mucoid bloody discharge on examination finger

The other systems were essentially normal.

Impression

Symptomatic uterine fibroids

Investigations

1 Pap smear: normal

2 Pelvic scan: multiple uterine fibroids some with calcified rims largest 104mm x 120mm. Adnexa and pouch of Douglas are free.

3 Intravenous urogram:

(IVU) Features suggestive of right hydroureter and hydronephrosis.

4. Haemogram:

W B C	6.37 x 10/L
Platelets	364 x 10/L
Hb	13.7 g/dl

5. Urea and electrolytes

Na+	139 mmol/l
K+	4.4 mmol/l
Bun	3.7 mmol/l
Creatinine	117 mmol/l

Management

She was scheduled for total abdominal hysterectomy. She was counseled and filled a written consent form. 2 units of blood was grouped and cross matched She was shaved the night before theatre and starved from midnight. Atropine 0.6mg was given hour before being wheeled to theatre. Enemax was also given the morning before going to theatre.

Operation

She was put in supine position and general anesthesia induced. She was put in semi-lithotomy position and vulvovaginal toilet done Catheterization was done and 150mls of clear urine was obtained and the catheter was left insitu. The vagina and cervix were then painted with betadine solution. She was put in supine position and abdomen cleaned and draped The abdomen was then opened via midline subumbilical incision.

The findings were: Huge uterus with multiple fibroids some pedunculated and appeared to have undergone sarcomatous changes. The ovaries and tubes appeared to be grossly normal. Total abdominal hysterectomy was done as described in the introductory pages of this book. The specimens were taken for histopathology

The abdomen was cleaned with saline and closed in layers. She was reversed uneventfully.

Post Operative Management

Vital signs were monitored continuously postoperatively until she was fully awake then '4 hourly until she was taken to the wards then Vi hourly for the first 3 hours and then 4 hourly thereafter.

Normal saline alternating with 5% dextrose was given 1 litre every 8 hours. She was put on I V antibiotics and analgesics.

On the first post-operative day the bowel sounds were present and she was started on oral sips and encouraged to ambulate. On the second post-operative day she was started on light diet. On the third post-operative day the wound was opened and found to be clean, and was dressed again with betadine. She was started on normal diet and oral medications

On the fourth day post-operative she was discharged home to come to the gynecology out-patient clinic in 3 weeks and also have the stitches removed in the nearest health facility on the 7^{th} post-operative day.

Follow Up

She came to the clinic after 3 weeks and she was found to be stable. The wound had healed well.

Histology report.

Sections showed features consistent with uterine leiomyoma some with calcific and sarcomatous changes. No evidence of malignant changes. The cervix was normal with no evidence of malignant cells.

Discussion

C A presented with symptomatic uterine fibroids. Total abdominal hysterectomy was done Uterine fibroids are benign tumours of the smooth muscles of the uterus. They are also referred to as leiomyoma or myoma (4).

They are the commonest pelvic tumours in women (2). They are estimated to be in about 25° o of women in reproductive age (1). This incidence may be higher as most are asymptomatic.

At Kenyatta National Hospital they account for 66.7% of hysterectomies carried out (3). Uterine fibroids develop between the age of 20 - 50 years. They are rarely seen before 20 years. They have a peak at 30 - 40 years and are 3 - 9 times more commoner in blacks than whites (1).

They are commoner in nulliparous and relatively infertile females (1). It is not clear if the fibroids cause subfertility or subfertility causes fibroids or both have a common cause (1). However fibroids may cause infertility by mechanical means by causing obstruction or interfering with implantation (2).

At Kenyatta National Hospital 70% of the patients had less than 2 children (3). The patient presented was a 49 year old para 3+0 black woman whose last delivery was 13 years earlier.

The cause of uterine fibroids is unknown. Estrogens have been implicated as evidenced by increased estrogen receptors in fibroids as compared to the surroundings myometrium and the fact that they grow after puberty and regress after menopause

They also enlarge with estrogen replacement (1,4). Reduction of fibroid size has been seen with administration of lutenizing hormone releasing hormone agonists (LHRH-GnRH) which render the women hypoestrogenic (5). Fibroids also have familial tendencies suggesting genetic factors (1).

Fibroids are classified according to the anatomical location into submucuos, intramural, interstitial and subserous (1). Majority of the fibroids are in the corpus of the uterus although 1-2% are found in the cervix (1).

Microscopically they are compared to non-striated muscle fibres arranged in a whorl pattern. Individual cells are spindle shaped with an elongated nucleus. They are dermarcated by a pseudocapsule from the sorrounding tissues (4).

Clinical presentation depends on the number, size, location, and presence or absence of complications. 30 - 50% of the fibroids are symptomatic of which 30% present with abnormal uterine bleeding (4).

Menorrhagia may be due to abnormalities of ovarian function leading to endometrial hvperplasia, large surface area due to submucous fibroids or the presence of abnormally dilated venous plexuses due to fibroid obstruction (4)

Others include abnormalities in prostaglandin production and uterine contractions which control blood flow through the uterine wall (4,5). Other symptoms include, pelvic pressure and pain. They may also cause urinary and bowel symptoms, infertility, miscarriages and vaginal discharge (1). In pregnancy they may cause complications which, include increase in uterine size, high caesarean section rate, malpresentation, premature labour and post partum hemorrhage (6).

Systemic manifestations include anemia due to menorrhagia occasionally polycythemia may be seen due to production of erythropoeitin by the tumor or compression of the ureters by the tumor leading to erythropoetin production by the kidney (4.5) Pain is another systemic manifestation which occurs following infarction or torsion of a pedunculated fibroid Uterine contraction to expecl a submucous fibroid or fenestration of the fibroid can lead to pain.

The fibroid can undergo several types of degeneration which include hyaline, cystic, calcific, septic, red, sarcomatous and fatty degeneration. 0.1 - 0.5% develop malignant transformation to leiomyosarcoma (5). Diagnosis of uterine fibroids is mainly clinical but many tests are valuable.

Ultrasonography may be able to tell the size and location of the fibroid and may differeniate between adenomyosis and ovarian masses. This may be enhanced by filling the uterus with saline (sonohysterogram), other tests are plain abdominal X-ray, hysterosalpingography and hysteroscopy. Laparoscopy and magnetic resonance imaging can be of use (MRI).

Hemogram may show anaemia or polycythemia. It may show a leucocytosis and elevated ervthrocyte sedimentation if there is septic degeneration (5). MRI may provide an excellent picture but usually the cost is not justified as all the information needed to plan management can be obtained by other methods (7).

Management of women with uterine fibroids depends on the patients age, parity, pregnancy status, desire for future pregnancies, general health and symptoms as well as the size and location of the fibroids (4).

Emergency treatment includes correction of anemia in those who have lost blood This includes blood transfusion and haematinics. Surgery may be indicated in those who have infected fibroids, acute torsion or intestinal obstruction (4). No treatment is required for asymptomatic uterine fibroids but judicious patient observation and follow-up is required.

Asymptomatic women who want to have children and are not infertile are best left alone as post operative adhesions formation can lead to tubal occlusion (8).

Perimenopausal women are sometimes not treated if symptoms are minor or are given gonadotropin releasing hormone (GnRH) agonist to reduce symptomatology (8,9). This is because they tend to shrink after menopause with the loss of estrogen (8). This should however, be reconsidered with current recommendation of hormone replacement (8).

Surgical treatment is recommended for symptomatic uterine fibroids and some of the indications are; abnormal uterine bleeding with resultant anemia, unresponsive to hormone treatment, chronic pain with dysmenorrhoea, dyspareunia, lower abdominal pain, acute pain secondary to degenerative change or torsion, urinary symptoms, rapidly growing fibroid or infertility (9).

When future fertility is desired or when there is a small submucous or subserous fibroid or the woman wishes to retain her uterus myomectomy is the method of choice. The patient has to consent for hysterectomy. The main risk of myomectomy is hemorrhage which may necessitate hysterectomy.

Hysterectomy is the definitive surgical option. For small fibroids (less than 12 weeks), vaginal hysterectomy can be done. Laparascopic hysterectomy is also possible for small fibroids. This can be total by morcellation or by laparoscopic assisted vaginal hysterectomy.

The normal total abdominal hysterectomy by laparotomy is the preferred mode as the cervix is removed reducing the risk of cervical cancer. However subtotal hysterectomy is getting new emphasis as it has demonstrated better bladder function after the procedure (8)-

Bonney's clamp and Rubin torniquet have been used to reduce bleeding during hysterectomy but now diluted vasopressin (1 ml/20 IV vasopressin with 19mls normal saline) either intramurally or perivascular has been shown to be better than the torniquet.

Other complications include adhesion formation which can cause infertility and uterine perforation in hysteroscopic myomectomy

Submucous fibroids can be removed by hysterosopic myomecctomy and subserous by laparoscopic myomectomy (8).

Medical treatment is used if the fibroids are large and required to be reduced in size and to correct anemia prior to surgery or if there is medical contraindication to surgery or if the patients is perimenopausal (3). Medical treatments include progestrones like depo-provera which reduced the bleeding. GnRH agonists cause temporary menopause leading to readuction in fibroid size. They are not used beyond 3 months due to osteoporosis and hot flushes (11).

They are normally used prior to surgery or in perimenopausal women. Other drugs are Danazol and progesterone antagonist RU 486 (Mifepristone) which result in significant reduction of uterine size (7).

Recently uterine artery embolization involving arterial catherization and embolization is being studied Radiotherapy can be used in conservative management especially in patients who are poor surgical risk.

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GYNAECOLOGY CASE No. 10

CHORIOCARCINOMA (LOW RISK)

CHEMOTHERPAY

Name:	H.K.	IP NO.	0978858
Age:	24 years	DOA.	22.09.04
Parity:	1 + 1	DOD	28 0904

PRESENTING COMPLAINT

The patient was readmitted for the second course of chemotherapy for treatment of low-risk choriocarcinoma.

HISTORY OF PRESENTING COMPLAINT

The diagnosis had been made in early September 2004 after she presented with a one-month history of per vaginal bleeding following suction curettage and sharp curettage for hydatiform mole 2 months prior to that. She also had rising beta-Hcg levels being 124 IU/L and 1871U/L. There was no history of cough, chest pain or vaginal bleeding.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was a para 1 + 1. Her last normal menstrual period was on 10.01.04. Her 1^{s1} delivery was in the year 2000 - spontaneous vertex delivery. She had abortion at 2 months in 2002 and evacuation was done Her menarche occurred at the age of 15 years. Her menses occurred every 28 days with a flow of 3 days. She had no dymenorrhoea. She used Depoprovera for contraception and her last injection was on 2nd July 04.

PAST MEDICAL AND SURGICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY

She was a housewife who stayed with her husband in Embakasi. She neither smoked cigarettes nor drank alcohol. There was no family history of diabetes mellitus. hypertension or asthma.

PHYSICAL EXAMINATION

General Examination

She was in good general condition. She had no pallor, no jaundice, no lymphadenopathy. She was clinically afebrile. The vital signs were: blood pressure 120/60 mmHg, pulse rate 92 beats per minute, respiratory rate 24 breaths per minute and temperature 37.2°C.

Abdominal Examination

The abdomen was scaphoid and moved with respiration. There were no areas of tenderness and there were no palpable masses.

Pelvic Examination

The external genitalia was normal The uterus was of normal size and there were no palpable masses in the adnexae or pouch of Douglas

Other systems.

The respiratory, cardiovascular and central nervous systems were normal.

DIAGNOSIS

1. Haemogram

   Hb - 14.5 g/dL

   WBC - 9.2 x 10^9/L

   RBC - 4.94 x 10^{12}/L

   Plat - 345 x 10^9/L

2 Urea/electrolytes

   Na+- - 136mmol/L

   K+ - 3.8 mmol/1

   Urea - 3.2 mmol/1

   Creat - 78 pmol/1

3 Beta-HCG - 4.21 U/L

4 Before treatment;

   (a) Pelvic scan - bilateral ovarian cysts the largest measuring 5 8 cm

   (b) Abdominal scan - normal

5 Histology;

   (a) After sharp curettage - degenerating products of conception

   (b) After suction curettage - features consistent with hydatiform mole.

6 Chest X-ray - Normal

MANAGEMENT

She received her second course of chemotherapy which she tolerated well and was discharged to be readmitted after 2 weeks with beta HCG results. She was to continue with two other courses of chemotherapy if the beta-HCG levels remained within normal limits. She was to be followed up for a total of two years.

DISCUSSION

The patient presented was a 24 year old para 1 + 1 who developed choriocarcinoma following a molar pregnancy. Choriocarcinoma is one of the neoplasms classified under gestational trophoblastic diseases. These are diseases characterized by expression of human chorionic gonadotropin in the absence of fetal heart tones and fetal structures. They are highly curable by chemotherapy. The other forms are hydatidiform mole and invasive mole [12] Choriocarcinoma may develop following a pregnancy (gestational choriocarcinoma) or in the absence of a pregnancy (non-gestational choriocarcinoma)[1] " Non-gestational choriocarcinoma is rare and is often associated with some teratoma or seminoma and other germ cell tumours [u]

Choriocarcinoma does not contain chorionic villous structures but is composed of sheets of both anaplsatic cytotrophoblast and syncytiotrophoblast. Placental site trophoblastic tumour (PSTT) is an uncommon variant of choriocarcinoma composed entirely of mononuclear intermediate trophoblast and does not contain chorionic villi. Choriocarcinoma has a propensity for early vascular invasion with widespread dissemination. The most common metastatic sites are the lung, vagina, brain and liver [1,2A] In this case, there were no metastatic lesions.

Choriocarcinoma occurs in about 4% of patients after the evacuation of a molar pregnancy and infrequently following other pregnancies [14]. Available data shows that 50% of cases of choriocarcinoma follow evacuation of a hydatidiform mole, 25% follow an abortion, 20% follow term pregnancy and 5% follow extrauterine pregnancy[5] Our patient developed choriocarcinoma following evacuation of a hydatidiform mole. The incidence of choriocarcinoma in the U.K. and the U.S. A is of the order of 1.50,000 to 1:70.000 pregnancies and it is ten times more common in South East Asia (5). The incidence at KNH was reported as 1:1,118 deliveries (6). The antecedent pregnancy was

hydatidiform mole in 57% of cases, normal pregnancy is about 26% and an abortion in about 17% of cases. The risk of choriocarcinoma after a hydatidiform mole is about 2 to 4% while that of a partial mole is 4 to 8%.

The diagnosis of choriocarcinoma requires a high index of suspicion. It is mainly clinical. History of persistent or irregular uterine haemorrhage following an abortion, a molar pregnancy or a normal delivery, should always raise the suscipicion of choriocarcinoma. The bleeding is usually profuse, but sometimes there may be only blood stains When amenorrhea occurs it is due to a very high level of HCG secreted by the metastatic growth outside the uterus. The rupture of the uterus with intraperitoneal haemorrhage simulates an ectopic pregnancy. The disease may also present by way of its metastasis. Dyspnea and haemoptysis are noticed with lung metastasis. Neurological symptoms like hemiplegia, convulsions, headache and visual disturbances may occur with brain metastases. Vaginal metastastasis appears as a bluish red vascular tumour which bleeds easily on touch. The uterus may be enlarged and granulose lutein cysts may be palpable Elevated BhCG together with any of the above symptoms makes the diagnosis of choriocarcinoma more likely.

All patients with suspected choriocarcinoma should undergo a careful pretreatment evaluation which includes:

1 Complete history and physical examination

2 Measurement of the serum BhCG value

3 Hepatic, thyroid and renal functions tests.

4 Determination of the baseline peripheral white blood cell and platelet counts.

Metastatic workup should include: a chest X-ray or computed tomography, ultrasonography of the abdomen and pelvis, CT scan of the head and in some cases selective angiography of abdominal and pelvic organs. Head CT scans helps in the early diagnosis of asymptomatic cerebral lesions

Choriocarcinoma is staged according to the International Federation of Gynaecologist and obstetricians. The staging is as follows:

Stage I: Persistently elevated BhCG levels and tumour confined to the uterus.

Stage H: Tumour outside the uterus but limited to the genital structures (Adnexa, vagina, broad ligament).

Stage III: Pulmonary metastases with or without uterine vaginal or pelvic involvement.

Stage IV: All other sites of metastases

At each stage patients are further classified into A, B, C depending on the risk factors. "A* is when there is no risk factor, 'B' only one risk factor and 'C' two risk factors. The risk factors are>

1 Human chorionic gonadotropin levels more than 100,000 mlu/mL.

2 Duration of diseases of more than 6 months from termination of antecedent pregnancy.

Other factors taken into consideration include prior chemotherapy and placental site tumours.

Apart from staging, selection of chemotherapy and the prediction of drug resistance depends on a prognostic scoring system. The prognostic scoring system in use currently is the WHO prognostic scoring system.

The scoring system is as follows:

PROGNOSTIC FACTOR	SCORE			
	0	1	2	4
Age (years)	<39	>39		
Antecedent pregnancy	H. Mole	Abortion	Term	
Interval between end of antecedent pregnancy and start of chemotherapy (months)	<4	4-6	7-12	>12
HCG (IU/L)	10^{J}	$10M0^{4}$	10^{4}-10^{5}	$>10^{3}$
ABO groups (female x male)		O x A A x O	B AB	
Largest tumour including uterine tumour (cm)		3-5	5	
Sites of Metastases		Spleen Kidney	G.I. Tract Liver	Brain

The total score for a patient is obtained by adding the individual scores for each prognostic factor. When the prognostic score is 8 or greater the patient is considered high risk and requires intensive combination chemotherapy to achieve remission. A score of

less than 4 is considered low risk and a score of 5-7 middle risk. Generally patients with stage 1 disease have a low-risk score and patients with stage IV have a high-risk score[1,4] Our patient was classified as low risk.

Choriocarcinoma is highly curable even with widespread metastases. Chemotherapy is the main stay of treatment of choriocarcinoma. Low risk patients are treated with single agent drug therapy. Methotrexate with or without folic acid rescue is used [1,4,7] Actinomycin D, 5-Flourouracil or Bleomycin may also be used 7. In our set up methotrexate is mainly used. Medium and high-risk disease are usually treated with combination chemotherapy. Several combination regimens have been advocated but currently the most commonly used are the M.A.C. and EMACO regimens The M A C protocol is a combination of Methotrexate given in doses of 0.4 mg/kg/d intravenously for 5 days, Actinomycin D given in doses of 10-12ug/kg/d intravenously for 5 days and cyclophosphamide in a dose of 3-5 mg/kg/d intravenously for 5 days. The cycle is repeated after 14 days or as toxicity allows. The EMACO regimen encompases Etoposide, Methotrexate, Actinomycin-D and Leucovorin calcium alternating with vincristine and cyclophosphamide 1,4,7. The EMACO regimen results in response rates of about 90% and survival rates of 80 to 100 percent (3,8). In our set up the MAC regimen is commonly used. In the event of MAC therapy failure EMACO regimen is used. Our patient had good remission with the MAC regimen. Patients with brain metastases should be treated with whole head irradiation with 3000 rads [1"] In the follow-up, all patient with stages I, II and III should have weekly BhCG measurement until normal 3 weeks and then monthly hCG until levels are normal for 12 consecutive months. Patients are encouraged to use oral contraceptive pills during the entire period of followup and our patient was put on the combined pill. Patients with stage IV disease are followed by weekly beta-HCG values until normal for 3 weeks and then monthly beta-HCG values until normal for 24 months.

Hysterectomy may be required in choriocarcinoma to control uterine hemorrhage or sepsis In patients with extensive uterine tumour hysterectomy may substantially reduce the trophoblastic tumour burden and thereby limit the need for multiple courses of chemotherapy. Hysterectomy is also indicated for placental site tumour because it is

quite resistant to chemotherapy. Hysterectomy is preceded and followed by chemotherapy to prevent the risk of dissemination and development of distal metastasis.

Patients with choriocarcinoma and other gestational trophoblastic diseases who are successfully treated with chemotherapy can expect normal reproduction in the future. The frequency of congenital anomalies is not increased, although chemotherapeutic agents are known to have teratogenic and mutagenic potential. However, women who have had trophoblastic disease are at an increased risk for developing trophoblastic disease in a subsequent pregnancy [1-3,4].

Women with low risk choriocarcinoma who are treated aggressively with single or multiagent chemotherapy have remission rate of almost 100%. In high risk choriocarcinoma remission rates have been reported to vary from about 45 to 65%. Among the prognostic factors are the site of metastases and the number of metastases[3,5]

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GYNAECOLOGY CASE No. 11

POLYCYSTIC OVARIAN SYNDROME - LAPAROSCOPIC OVARIAN DRILLING

Name	:	F. N	DOA:	03.04.04
Age	:	38 YEARS	DOD:	07.04.04
Parity	:	0+0		
[P.No.	:	0794220		

PRESENTING COMPLAINTS

She came with inability to conceive for 10 years was admitted through the gynecology clinic for ovarian drilling.

HISTORY OR PRESENTING COMPLAINTS

She was a para 0+0 who had been married for the previous 10 years. She had had sexual intercourse regularly. Menses were irregular and she experienced amenorrheic episodes of upto 4 months. Menarche was at 13 years. She had been attending several clinics for 6 years and ovulation induction drugs had been given for the previous three years 3 years.

PAST MEDICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY

She is was housewife who did not smoke nor drink alcohol. Her husband was a businessman. There was no family history of chronic illness.

PHYSICAL EXAMINATION

She was in fair general condition, not pale or jaundiced. Her blood pressure was 120/80mmhg, pulse rate 84/min, temperature 37° C and she weighed 96kg.

BREAST EXAMINATION

Breasts were soft and there was no galactorhea.

TNECK EXAMINATION

Thyroid gland was not enlarged

ABDOMINAL EXAMINATION

It was soft and uniformly distended. No masses were palpable

PELVIC EXAMINATION

There was normal external genitalia. The cervix was posterior, and the uterus normal and ante-verted. There were bilateral adenexal masses.

INVESTIGATIONS

Pelvic ultrasound - This showed a normal uterus with bilateral enlarged cystic ovaries consistent with polycystic ovaries

Laboratory tests

LH - 30.7 min (n - 1.2 - 12. 5)

FSH - 279 miu/1 (n-3.2-10)

PRL - 204 MIU/L (N - 268 - 490)

MANAGEMENT

She was put on liquid diet from the day before the operation, given senekot the night before the operation and enema in the morning. Pre-medication with 0.6mg atropine and pethidine 50mg intramuscularly was given half hour before theatre. In theatre general anesthesia was induced she was placed in Trendelenburg position and the abdomen was cleaned and draped. VVT was also done and the necessary drapes put The uterine elevator was placed in position. A umbilical stab incision (10mm) was made with a knife followed by insertion of the 10mm trocar and canula. Entry was successful and a Verres needle was inserted and carbondioxide introduced to make a pneumoperitoneum. Once pneumoperitoneum was achieved the light source was connected and a 30° angled laparoscope was introduced. Two more stab incisions (5mm) were made on the left and right iliac fossa and laparoscopic endocoagulators introduced through the 5mm canulas. Both ovaries were identified and found to be polycystic - with about 20 cysts in each

vary. Using unipolar diathermy drilling of each ovary was done. The uterus was noted to t ? e normal. Both fallopian tubes were patent and a dye test with Methylene blue showed trilateral peritoneal spill. The abdomen was irrigated using normal saline and heparin 3000 IU and hydrocortisone 200mg was left insitu. The stab wounds were closed using "Vicryl No. 2/0.

POST-OPERATIVE CARE

She was observed V_2 hourly till fully awake then 4 hourly. She was started on analgesics. On the second post operative day she was put on light diet and discharged home on brufen and amoxil for review after two weeks.

FOLLOW - UP

She was seen in the clinic after two weeks, she had no complaints. The wounds had healed well. She was advised to loose weight and was put on metformin 500mg once a day. She was seen after three months when she was confirmed to be pregnant and she was booked for antenatal clinic.

DISCUSSION

FN presented with 1° infertility secondary to Polycystic ovarian syndrome and ovarian drilling was done.

Polycystic ovarian syndrome was originally described in 1905 by Stein and Leventhal as a syndrome consisting of amenorrhea, hirsutism and obesity in association with enlarged polycystic ovaries. It is now realized that this syndrome is in fact a hormonal disorder and most clinicians prefer referring to it as a 'syndrome of hyperandrogenic chronic anovulation'.

The incidence of polycystic ovarian syndrome (PCOS) is about 3% in both adolescent and adults, it is the most common cause of hyperandrogenism of pre-pubertal onset (2). In PCOS the main hormonal disturbance is elevated LH (Leutenizing hormone) which results in increased androgen secretion from the ovary resulting in wasting of the ovarian

Dllicles by interfering with production of the dorminant follicle. This results in absence »f mid LH surge, which results in unovulation (3).

t is also known that these patients have insulin resistance, which results in excess androgen production thus follicular wasting.

Symptoms and signs of PCOs include irregular or absent periods, unovulation, weight «ain. hirsutism, acne, multiple ovarian cysts and ancthosis nigrans. In addition there is unoppesed estrogen stimulation of the endometrium leading to endometrial hyperplasia and rarely adenocarcinoma (4).

Diagnosis is based on the signs and symptoms mentioned above though not all may be present. In addition, infertility is major symptom. Ultrasound may show ovaries with multiple cysts.

Laboratory tests may show elevated LH, with normal or low FSH. Progesterone levels in luteal phase may show unovulation levels. Androgens are usually elevated both testrosterone and andrestenedione and in 20% of patients with PCOS there is elevated prolactin (3).

Treatment of PCOS is aimed at menstrual irregularity, anovulation or infertility and hirsutism. Weight loss causes decreased insulin levels and causes ovulation and improves hirsutism. Insulin sensitizing drug like metformin may lower sugar levels hence leading to reduced insulin requirement and in return improving insulin resistance.

Treatment of hirsutism and hyperandrogenism can be done using oral contraceptives in those who do not want pregnancy, in addition, antiandrogens can be used. Progestins may be used to control irregular cycles by mimicking the action of progesterone. Irregular menses may also be controlled by GnRH (Gonadotrophin releasing hormones analogues) and again this lead to suppression of LH and FSH.

Ovulation induction may be achieved by use of clomiphine citrate with ovulation 3-chieved in 80% of women and a 6 month successful pregnancy rate of 45 - 50%. The patient presented had been given clomiphine without success. If this fails ovarian stimulation using GnRH agonist with IVF may increase rate of pregnancy to 54 % after 6 months and 62% after 12 cycles. This is not practiced in our center (4).

Laparascopic ovarian diathermy or drilling is an alternative to ovarian stimulation. Exact mechanism of its action is unknown but it results in pregnancy rate of 60 - 80% after 12 month's (5).

The patient presented underwent successful ovarian drilling and at the time of writing the case she had already conceived.

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GYNAECOLOGY CASE No. 12

CARCINOMA OF THE OVARY STAGE IIIB- DEBULKING AND CHEMOTHERAPY.

NAME	Z.W	DOA:	09.05.2004	DOD	4.06.2004
AGE:	62 YEARS	DOA:	30.06.2004	DOD:	5.07.2004
IP.NO:	0960097	DOA:	21.07.2004	DOD:	26.07.2004
PARITY:	5+0	DOA:	14.08.2004	DOD:	20.08.2004
LMP:	10 YEARS	DOA:	06.09.2004	DOD:	12.09.2004
	POSTMENOPAUSAL	DOA:	04.10.2004	DOD:	09.10.2004

PRESENTING COMPLAINTS

Z.W was admitted with complaints of progressive abdominal swelling for three months and abdominal pains for two weeks.

HISTORY OF PRESENTING COMPLAINTS

Z.W was a referral from Kijabe AIC hospital where she had presented with a history of progressive abdominal swelling for three months and abdominal pains for two weeks. She had no constipation or altered bowel habits or vomiting. Her appetite was slightly reduced and she was also growing weak. She did not have any urinary symptoms. She did not have abnormal vaginal bleeding, no abdominal hair or deepening of her voice.

OBSTETRIC AND GYNECOLOGIC HISTORY

She was a para 5+0, her last delivery being in 1984. All her deliveries were in hospital and were normal. All her children were alive and well. In her reproductive period her menstrual cycle was normal with a cycle of 30 days lasting 3-4 days. She had no menorrhagia or dysmenorhoea. She did not give any history of contraceptive use.

FAMILY AND SOCIAL HISTORY

She was widowed, she lived in Kijabe where she was a farmer. She did not smoke or take alcohol. There was no chronic illness in the family.

FAST MEDICAL AND SURGICAL HISTORY

"This was non-contributory.

GENERAL EXAMINATION

She was in fair general condition and nutritional status. She was mildly wasted and mildly pale. She did not have jaundice, cyanosis or edema. She had no lymphadenopathy Her blood pressure was 110/75mmHg, pulse rate of 80 beats per minute, temperature of 36.9 C and a respiratory rate of 20 per minute.

ABDOMLNAL EXAMINATION

The abdomen was uniformly distended and moving with respiration. There were no visible vessels. There was a pelvic mass corresponding to 28 weeks fundal height. It was firm, nodular with restricted mobility and mildly tender. One could not go below it. Fluid thrill and shifting dullness were positive. There was no hepatosplenomegaly.

PELV IC EXAMINATION.

The external genitalia looked atrophic. On speculum examination, the vaginal walls were atrophic with loss of ruggae. The cervix looked atrophic. No evidence of metastatic implants were noted. On digital examination, the vaginal mucosa was smooth with loss of elasticity and the cervix was firm and smooth. There was a pelvic mass corresponding to 28 weeks, felt more to the right adnexae, was of restricted mobility and it was difficult to delineate the size of the uterus. The mass could be felt through the pouch of Douglas.

The central nervous system, respiratory and cardiovascular systems were essentially normal.

IMPRESSION

A diagnosis of an ovarian tumor was made.

INVESTIGATIONS

She had come with investigation results which were done at the Kijabe Mission Hospital.

1. Chest X-ray: Lung fields were clear, heart size and shape were normal. Pulmonary vessels and bony cage were normal the costophrenic margins were normal

2 Abdominopelvic scan: showed complex multiseptate right ovarian mass 65cm x 57cm. The liver and spleen normal. Moderate ascites noted. Left ovarian cyst 5cm x 5cm noted.

3 Haemogram: WBC:6.8x1O/L
Hb: 9.8 g/dl
Platelets: 390 x 10/L

4. Liver Function Tests: Alb: 46
ALT: 9
AST: 22
ALP 93
GGT: 13
Total Prot: 64 g/dl.
Bilirubin. T-5.7mmol/L. D-0.5mmol/L.

5 Renal Function Tests: Na+ 136 mmol/L
K+ 4.5 mmol/L
Urea 4.1 mmol/L
Creatinine 84 umol/L

6 PAP Smear C[N O.

MANAGEMENT

She was informed of the diagnosis and need for surgery and chemotherapy. She was transfused one unit of blood and 2 units were grouped and cross matched in readiness for surgery. She gave an informed consent and she was prepared for laparotomy. She was starved overnight and enema given at 6.00 AM the day of the surgery. She was pre-medicated with 0.6 mg of atropine and wheeled to theatre.

1NTRA-OPERATIVE FINDINGS

In theatre, the patient in supine position was put under general anaesthesia. She was repositioned to semi-litothomy and vulvovaginal toilet done. Under aseptic technique, catheterization was done and 150mls of clear urine was obtained. Examination under anaesthesia revealed a short closed cervix in posterior position. There was an ill defined right adnexal mass which was mildly mobile and nodular Pouch of Douglas was full. The vagina and cervix were painted with methylene blue dye. She was put in supine position and the abdomen was cleaned and draped.

The abdomen was opened through a midline sub-umbilical incision. There was a right ovarian mass approximately 70cm x 50cm with a twisted stalk attached to the meso-ovarium .it was firm and nodular and appeared hemorrhagic, the capsule was broken. The left ovary had a cycstic mass 5cm x 5cm. The uterus was small and atrophic. There were tiny tumor seedlings on the omentum and peritoneal surfaces. Approximately 1200 mis of amber colored ascitic fluid was sucked out of the peritoneal cavity. The liver and the spleen were normal. The mass was removed and total hysterectomy and left salpingoopherectomy was done and all specimens taken for histopathology Infracolic omentectomy was done. Tumor seedlings were also taken for histopathology. 10 mis of ascitc fluid was also taken for cytology. Haemostasis was achieved and peritoneal lavage done with warm saline and rifocin. The abdomen was closed in layers after the swabs and instruments were counted and ascertained to be correct. Anaesthesia was reversed uneventfully.

POST-OPERATIVE CARE

She was transferred to the recovery room, where vital signs were observed continuously until she was fully awake. Normal saline to alternate with 5 % dextrose was continued at least 3 litres in 24 hours. She was started on crystalline penicillin 2MU 6 hourly, gentamicin 80mg 8 hourly and flagyl 500mg 8 hourly all given intravenously. She was also given intramuscular pethidine lOOmg 8 hourly.

On the first post-operative day, bowel sounds were heard and she was started on oral sips. On the second day she was started on light diet and oral medication, ampiclox 500mg 6

friourly, flagyl 400mg 8 hourly and ponstan 500mg 8 hourly. On the third day the wound was inspected and found to be clean. The stitches were removed on the post-operative day. On the 10th postoperative day the histology results were obtained.

HISTOLOGY REPORT

Histology of the specimen showed well differentiated serous cystadenocarcinoma of the ovary with spread to the omentum and peritoneal surfaces. Uterus had atrophic endometrium.

CHEMOTHERAPY

She had repeat hemogram renal function and liver function tests which were normal and on the 15th postoperative day she was started on cyclophosphamide 500mg once a day for five days, Adriamycin 50mg stat and Cisplatin 50mg stat. She was discharged on the 21st post-operative day to come again in two weeks time for the second course of chemotherapy. She was seen on the days as indicated and she tolerated the cytotoxics well. Her laboratory parameters remained within normal and she completed 6 courses of chemotherapy. She was then to be followed up in the gynecology out-patient clinic but she was lost to follow-up.

DISCUSSION

^ . W was a referral from AIC mission hospital with complaints of progressive abdominal swelling and abdominal pains. A diagnosis of ovarian cancer was made and staged as stage IIIB. She received chemotherapy but was lost to follow-up

Masses of the ovary may be divided into neoplastic and non-neoplastic lesions. The non-neoplastic lesions are predominantly of inflammatory origin (1,2). The neoplastic lesions may be divided into physiological enlargement and those that are pathological (1,2). Physiological neoplasms develop exclusively during the years between menarche and menopause. They are usually transient, unilateral and related to aberrations of ovulation. If cysts persist beyond 60-90 days, despite normal menstrual cycles, the enlargement is considered a pathological neoplasm. If the tumour disappears during this time, it is most likely a functional cyst. Pathologic neoplastic masses may be benign, malignant or of borderline malignant potential. Of all gynecological neoplasm's, ovarian malignancies represent the greatest clinical challenges. Ovarian cancers also represent major surgical challenges. It has the highest fatality to case ratio of all gynecological tumors(3).

Cancer of the ovary accounts for about 25 % of all malignant neoplasm's of the female genital tract. In North America, ovarian cancer claims the lives of more women each year than all other gynecologic malignancies combined. Over 50 % of deaths ascribable to gynecologic cancer are due to cancer of the ovary. It is the 5^{Ul} leading cause of cancer related morbidity among American women accounting for 50 % of all such deaths (3,4). In India, ovarian cancer accounts for about 5 % of all gynecological cancers (5). Njuki (1979), at Kenyatta National Hospital found that cancer of the ovary comprised 8 % of all female genital tract malignancies and ranked third as a cause of gynecologic malignant disease after cancer of the cervix and choriocarcinoma (6) The worldwide incidence of ovarian cancer is higher in developed countries compared to developing countnes.

Ovarian cancer is a disease of the postmenopausal woman and the prepubertal girl although it is documented to occur in females of all ages (3). With the exception of

teratomas and special sex cord tumours which have their own age incidence, ovarian neoplasm's are commonly found in women aged 40-60 years (3). Njuki in his study found an age range of 9-63 years, while Ojwang and colleagues at the same hospital found an age range of 40-60 years with a mean age of 47.7 years (6,7). The patient presented was 62 years old and was 10 years postmenopausal.

The cause of cancer of the ovary is unknown. However a number of risk factors have been identified. Women of low parity, infertility and delayed child bearing are at an increased risk (1,2,3). Stimulation of the ovary by ovulation induction drugs such as clomiphene during infertility treatment slightly increases the risk of ovarian cancer. Exposure to industrial agents such as asbestos and talc and high fat diet also increases the risk. Genetic factors also play an important role as seen in site specific familial ovarian cancer breast/ovarian familial cancer syndrome and Lynch II syndrome. A familial history of ovarian cancer has been observed in 7 % of the cases. The most common pedigree's are sister/sister and mother/daughter patterns (2,4). Patients with turner svndrome are at increased risk of dysgerminoma and gonadoblastoma. Chronic anovulation, multiparity and breastfeeding are protective Pregnancy decreases the risk of ovarian cancer by 30-60 %. Oral contraceptive use also decreased the risk by 30-60 % depending on the duration of use (2). Patients done tubal ligation are at decreased risk though the mechanism is unknown (1). Our patient did not have increased risk factors however she had few of the reduced risk factors.

Ovarian neoplasm's are classified into four major types based on the cell type of origin. These are:

1. Epithelial tumours which account for 70-80 % of all ovarian neoplasm's
2. Germ cell tumours account for 15-20 % of all ovarian neoplasm's
3. Sex cord and stromal tumours which account for 5-10 % of all ovarian neoplasm's
4. Neoplasms metastatic to the ovary (2,3).

Epithelial tumours account for more than 90 % of all malignant ovarian tumours. They include serous, mucinous endometrioid, clear cell, transitional cell and undifferentiated.

fYi<ey are commonly found in adults (2,3). Our patient had an epithelial tumour of the ^^rous adenocarcinoma type. Germ cell tumours tend to occur between the second and .•inird decades. They include; dysgerminoma, endodermal sinus tumour, embroyonal cell c3-*"cinoma, teratoma, non-gestational choriocarcinoma and gonadoblastoma. Most of -riese neoplasm's produce biological markers which can be monitored to assess response zo therapy (2,3). Sex cord and stromal cell neoplasm's include; granulose cell tumors, fibroma. thecoma and sertoli-leydig cell tumors Granulosa cell tumors are associated with hyperestrogenism and may cause precocious puberty in young girls and adenomatous hyperplasia and vaginal bleeding in postmenopausal women (8). Androgen producing tumours (sertoli-leydig) may cause virilization. Our patient did not have the mentioned clinical features.

"Neoplasms metastatic to the ovary account for approximately 25 % of all ovarian malignancies. They mimic primary ovarian cancer and are unilateral in as many as 25 % of patients. Most common cancers that metastasize to the ovary are those of the breast, stomach, colon and endometrium. Carcinoma of the stomach metastatic to the ovary is commonly referred to as Krukenburg tumour. Carcinoma of the ovary is staged according to the International Federation of Gynecology and Obstetrics (FIGO) classification of ovarian neoplasm's (2,5).

Stage I: Growth limited to the ovaries

- la: one ovary involved, capsule intact
- lb: both ovaries involved, capsule intact
- Ic: one or both ovaries involved with malignant cells on the surface, or ruptured capsule or ascites with malignant cells or positive peritoneal washings.

Stage II: Growth involving one or both ovaries with pelvic extension

- Ila: Extension or metastasis to the uterus or tubes
- lib: Extension to other pelvic tissues
- He: Stage Ila or lib with tumor on surface of the ovary or ruptured capsule or ascites with malignant cells or positive peritoneal washings.

Stage III: Tumour extension to the abdomen

Ilia Abdominal peritoneal surfaces with microscopic metastases

Illb: Tumour metastasis < 2cm in size

IIIc: Tumour metastasis > 2cm in size or metastatic disease in the pelvic para-aortic or inguinal nodes.

Stage IV Distant metastasis

Pleural effusion, lung metastasis, liver or splenic parenchymal metastasis and to the supraclavicular nodes or skin.

The patient presented had stage IIIB disease because she had tumor on the peritoneal surfaces but were less than 2cm diameter Ovarian cancers are rarely symptomatic except for those tumours that have endocrine function Ovarian cancers are usually diagnosed late as seen in our patient. Patient's ignorance due to lack of education and non-availability of health care in the remote areas of the country also contribute to late diagnosis (7). Symptoms include abdominal swelling from large tumour or associated ascites, dyspepsia, cachexia, urinary retention, bowel obstruction and aching pain in the abdomen (2,3). Menstrual abnormalities and abnormal vaginal bleeding may develop in 15 % of the patients (8). Our patient presented with abdominal swelling and pain. On physical examination presence of ascites plus a pelvic mass that is fixed, solid, nodular or bilateral is suggestive of malignancy. Presence of Sister Mary Joseph's nodes suggests metastasis to the umbilicus (2,5). Our patient had a pelvic mass that was firm nodular and relatively fixed.

Evaluation of a patient with suspected ovarian neoplasm include; a complete blood count, liver function tests, urea and electrolytes, coagulation profile, cervical cytology and intravenous urography to define the ureters and exclude a pelvic kidney. Barium enema is done to rule out colonic involvement or colonic cancer. Chest X-ray is done to detect metastasis to the lungs. Pelvic ultrasound is done to evaluate the mass. Ultrasound features of malignant tumours include. Solid, cystic and solid, multiple septations more than 3 mm in size bilaterally and ascites. CT scan provides information about the retroperitoneal structures in addition to the pelvic organs. The ultimate diagnosis however

depends on surgical exploration (2,3,5,7).Our patient had all investigations done except IVU, CT scan and barium enema.

Tumour markers are useful in diagnosis and management of various ovarian malignancies CA-125 is an antigen produced by most primary ovarian malignancies though raised levels may also be found in other malignancies and benign conditions (9). An elevated CA-125 in the postmenopausal patient is particularly suggestive of the presence of an ovarian malignancy. Other cancers that may be associated with elevated CA-125 levels include cancers of the colon, breast, pancreas, stomach, uterus and fallopian tubes (2). Other markers include alpha-fetoprotein, human chorionic gonadotrophin. Markers for specific tumours include;

Epithelial tumours	-	CA-125
Dysgerminomas	-	LDH
Teratoma	-	alfa-feto protein
Choriocarcinoma	-	beta-hcg
Sertoli-leydig	-	Testosterone
Granulosa cell	-	Estradiol

Our patient was not investigated for any tumour marker

There are two main modes of treatment of ovarian neoplasms, namely surgery and chemotherapy. Surgery is usually the first line of treatment. It is usually performed to establish the stage, type and histologic grading of the tumour (2,5). In early disease surgery may be all that is required. However, in some cases when the surgeon feels that the cancer may be difficult to remove, a biopsy is taken, a few courses of chemotherapy ire given first and surgery thereafter. During surgery the incision should provide naximum exposure of the pelvis and allow thorough examination of the abdomen. If iscites is absent peritoneal washings are obtained from the pelvis, right and left paracolic gutters and supra-hepatic space by instillation of lOOmls normal saline into each area 2,4,5). The operation aims at resecting as much tumour as is safely possible. Total ibdominal hysterectomy is the preferred treatment for stage I and II disease. Stage III and Vdebulking of the tumour plus omentectomy should be attempted as much as possible 2,4,5).

Chemotherapy is often given after surgery in stage **III** and **IV** and if the surgeon feels there is a high risk that microscopic cancer cells may be present. Sometimes a few courses of chemotherapy may be given to shrink the tumor before surgery is carried out. Most of the epithelial tumours and germ cell tumours respond well to chemotherapy. Generally a pulse therapy regime is used. This involves five days of chemotherapy every three weeks for 6-8 cycles For epithelial cancer, drugs used include cyclophosphamide, cisplatin and adriamycin up to twelve cycles In germ cell tumours regimes containing cisplatin have been used. These regimes include, bleomycin, etoposide, and cisplatin (5). Multi-drug chemotherapy has been reported to result in good response Other new drugs carboplatin and paclitaxel (taxol) are now the drugs of choice (3). In our set-up cisplatin, cyclophosphamide and adriamycin are commonly used. **During** therapy the patients are seen monthly. A full blood count, liver function tests and renal function tests are performed before each cause of chemotherapy. A -pelvic examination to assess disease status is also done on monthly basis. Second look laparotomy is done to determine whether chemotherapy has eradicated the tumour, to determine cessation of chemotherapy or to debulk the residual tumour (4,5,10). It is usually performed after 6 to 8 courses of chemotherapy. Patients who have completed chemotherapy and who are disease free are evaluated every 2-3 months for two years. Thereafter they are evaluated every 6 months. Our patient was lost to follow-up and no second look laparotomy was done.

Radiotherapy is not often used to treat ovarian cancer but may be occasionally used to treat individual areas of the cancer if it recurs after surgery and chemotherapy. However granulosa cell tumours and dysgerminomas are highly radiosensitive (2,5).

The prognosis of ovarian cancer is related to stage of the disease. The 5-year survival rate tor patients with stage I epithelial ovarian cancer is approximately 80 %, stage II is 40-50 % stage III is 30 % and stage IV less than 10 %. Germ cell tumours are associated with better 5-year survival rates than epithelial neoplasms (2,5).

Screening methods for cancer of the ovary include, routine pelvic examination, pelvic ultrasound and CA-125. The disadvantage of the tests is lack of sensitivity and

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Screening methods for cancer of the ovary include, routine pelvic examination, pelvic ultrasound and CA-125. The disadvantage of the tests is lack of sensitivity and

voecificity There has been no evidence on whether screening improves outcome for **omen in any risk group (2,4, 5,9,12).**

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GYNAECOLOGY CASE NO. 13

CARCINOMA OF THE VULVA WITH GENITAL WARTS: MODIFIED VULVECTOMY AND RADIOTHERAPY

Name:	**P.M.**	**I P. NO.:**	**0903894**
	29 years	D**.O.A:**	**18.08.04**
pjrity	0+2	D.O.D:	**02.09.04**

PRESENTING COMPLAINT

She was admitted through casualty with a one-month history of a genital swelling.

HISTORY OF PRESENTING COMPLAINT

She was well till one month prior to admission when she developed a swelling on her aenitals. The swelling initially was small with a smooth surface but progressively increased in size. There was no associated pain or pruritus vulvae and no vaginal discharge. She complained that the lesion would be painful especially at night

OBSTETRICS AND GYNAECOLOGY HISTORY

She was para 0 + 2. Her first abortion was in 1993 and the second one in 1995. Both abortions occured at a gestations of 4 and 5 months respectively. Her menarche occurred at the age 17 years. Her last menstrual period was on 8th September 2004. She had regular menses with a cycle of 28 days and a duration of flow of 3 to 4 days. She had no dysmenorrhoea. She was not on any contraceptives.

PAST **MEDICAL HISTORY**

She was admitted in IDH in 1998 due to tuberculosis and put on 6 month treatment regime which she completed. She had also had an apendicectomy and cervical node excision biopsy.

FAMILY AND SOCIAL HISTORY

She was single and lived with her parents. She was unemployed at that time although previously, had been engaged in business. She neither smoked cigarettes nor drank alcohol. There was no family history of chronic illnesses.

PHYSICAL EXAMINATION

General Examination

She was a young lady in fair general condition. She was not pale, had no jaundice or pitting pedal edema. She was clinically afebrile. She had a surgical scar on the right

cavical region of the neck bilateral inguinal lymphadenopathy. The nodes were non-tender and mobile.

Xbdominal Examination

The abdomen was scaphoid and moved with respiration. There were no surgical scars or therapeutic marks. There were no palpable masses

Vaginal Examination

There was a large fungating mass on the lower half of the left labia majora that was obliterating the introitus. Its size was about 6cm in diameter. The mass was friable and bleeding easily on touch. It was no possible to do a digital examination.

Other systems

The respiratory system, cardiovascular system and the central nervous system were essentially normal.

DIAGNOSIS

A diagnosis of cancer of the vulva stage IV was made.

MANAGEMENT

The diagnosis and management was explained to her and an informed consent obtained. The patient was prepared for examination under anaesthesia for staging and biopsy. She was also done pre-and post test counselling for HIV and VDRL Blood was taken for urea and electrolytes and haemogram.

Investigations done

1	Haemogram	-	12.3.g/dL
	WBC	-	8.1 x 10^9/d/L
	RBC	-	4.22 x 10^{12}/d/L
	Platelets	-	246 x 10^9/D1
2	Urea and electrolytes		
	Na+	-	129 mmol/l
	K+		3.5 mmol/l
	Urea	-	2.9 mmol/l
3	VDRL		Negative
4	Elisa test for HIV		Positive

20.08.04 Examination under anaesthesia (EUA).

She was premedicated with intramuscular atropine 0.6mg 30 minutes before theatre. In -.-eatre. general anaesthesia was given and she was placed in lithotomy position. Vulvo-a n a l toilet was done then draped. Aseptic catheterization was then done.

She was found to have a large fungating mass involving the left labia majora and minora. It had a necrotic centre. It was very friable and bleeding easily on touch. The right labia majus was not involved. There were multiple, small warts on the right labia with signs of infection Small warts involving the vagina and cervix were also found. There were no adnexal masses or masses in the pouch of Douglas. The urethra and rectum were not involved but the warts extended up to the skin around the anus. The uterosacral ligaments were not involved. There was bilateral inguinal lymphadenopathy that was mobile A wedge biopsy from the mass was taken for histology. General anaesthesia was reversed uneventfully and the patient returned to the ward in a stable condition. She was started on antibiotics-Amoxicillin 500mg 8 hourly and metronidazole 400mg 8 hourly.

Histology results

Showed an infiltrating moderately differentiated squamous cell carcinoma of the vulva.

The patient was then planned for modified vulvectomy. The nature of the operation was explained to her and an informed consent obtained. Haemogram and urea and electrolytes were repeated and the results were normal.

She was scheduled for operation on 28.08.04. Blood was taken for grouping and cross-match and three units reserved for the operation. She was started on light diet (low fibre diet) on the previous day She had enema done at 6.00 p.m. the previous day and at 6.00 a m on the day of the operation. She also had dulcolax given at 6.00 p.m. on the previous day. She was starved from midnight. On the morning of the operation, she was premedicated with atropine 0.6mg intramuscularly half-hour before theatre.

Modified Vulvectomy

In theatre, the patient was put under general anaethesia in supine position. She was then rlaced in lithotomy position. Vulval toilet was done then she was draped. Aseptic catheterization was done with a Foley's catheter and clear urine obtained A repeat EUA confirmed earlier findings.

F:ndings were a wide fungating mass on the left lower third of the vulva which had a wide stalk and was mobile Wide excision beyond the mass (>5cm) was done isolating and removing it. Bleeders were clamped and cauterised as need arose and good haemostasis was eventually achieved. The edges of the subsequent gaping would were undermined to minimize tension then closed up in two layers. The perianal and vulval warts present were cauterised. A pfannestiel incision was made, bleeders cauterised and both superficial and deep inguinal lymph nodes (approx. size 3 x 5 cm) were removed bilaterally The incision was closed by interrupted vertical mattress stitch using nylon 2/0.

Three specimens were collected:

(i) Vulval mass

(ii) Right inguinal node

(iii) Left inguinal node

The patient was successfully reversed from anaesthesia. Total blood loss was 400mls. The specimen was taken for histology.

POST OPERATIVE CARE

She was transferred to the recovery ward and observed 1/2 hourly until fully awake and stable. She was then transferred back to the ward to be observed 4hourly. She was put on intravenous fluids normal saline alternating with 10% dextrose 3 litres in 24 hours. She was started on intravenous crystalline penicillin 2 mega units 6-hourly, Gentamicin 80mg 8-hourIy and Amoxicillin 500mg 8-hourIy. She was given pethidine lOOmg 8-hourly for 48 hours. She was to have daily saline sitz baths. The intravenous antibiotics were changed to oral antibiotics after 3 days. She was transfused one unit of blood. She recovered well post-operatively.

Sutures **were removed** on **the** 14th postoperative day and the wound had apposed well except for **a raw area** in the perineum. **She** continued on daily warm saline sitz baths jitil the wound healed. The wound healed well and she was discharged to be seen in the clinic after two weeks.

Follow-up

She was seen in the GOPC after two weeks and she had no complaints. She was in good general condition and the incision site had healed well. She was to book radiotherapy after healing of the wound.

DISCUSSION

This was a 29 year old woman who presented with carcinoma of the vulva with vulval warts and was HIV positive. She had modified vulvectomy done and was referred for radiotherapy.

Carcinoma of the vulva is an uncommon malignancy accounting for 0.3% of all female cancers in the United States of America and 3 to 5% of all female genital malignancy[1] It accounts for 1% of all malignancies in women and is the fourth most common gynaecologic cancer after endometrial, ovarian and cervical cancer (2). It is usually a disease of older women with the median age being 67 years (1,3). In Kenya 3 .3% of all genital malignancies are due to vulva carcinoma. It is the fourth commonest genital malignancy after cervical carcinoma, ovarian cancer and choriocarcinoma. An average of four cases are seen annually at the KNH. Carcinoma of the vulva accounted for 3-5% of all female cancer deaths (4). This patient was a 29 year old woman

Vulval carcinoma may arise from the skin, subcutaneous tissues, urethra, glandular elements of the vulva or the mucosa of the lower 1/3 of the vagina. The predominant histological type is squamous cell carcinoma. 90% of lesions are of squamous origin with 3.5% being melanoma, 2% basal cell carcinoma and 1% originating in Bartholin's gland. Sarcomas are rare accounting for less than 1% of vulva malignant tumours.

The aetiology of **vulva** carcinoma is unknown. Epidemiologic factors that have been iisociated with the development of vulva cancer include granulomatous infection, herpes simplex and human papillomavirus. Our patient had vulval warts hence was at a high risk of developing **vulval** cancer. Associated disorders found mostly with vulva carcinoma are obesity, hypertension and chronic vulval irritation secondary to diabetes mellitus. Chronic immunosuppression has also been associated with the development of vulva cancer.

Vulva carcinoma in-situ, like carcinoma in-situ of the cervix is considered a precursor to invasive diseases though the risk of progression is lower occurring in about 3% of all patients 1,6 Vulva carcinoma in situ tends to be multifocal with a low risk of invasive disease in younger women but tends to be unifocal with higher risk of invasive disease in older women 7. For this reason, all patients with vulva carcinoma in-situ should be treated and long term follow-up is mandatory. Patients with carcinoma of the cervix are at increased risk of developing vulva carcinoma and vice versa. Vulva cancer has 3 distinct methods of spread lymphatic, haematogenous routes and direct extension to adjacent structures. The most common method of spread is the lymphatic route. The lympathic spread is by way of the superficial inguinal, deep femoral and external iliac lymph nodes. Contralateral spread may occur as a result of the rich intercommunicating lymphatic system of the vulva skin. Direct extension to the deep pelvic lymph nodes primarily, the obturator nodes occurs in 3% of patients and seems to be related to midline involvement around the clitoris, urethra or rectum or to cancer of a vestibular (Bartholin's) gland. The superficial and deep nodes intercommunicate and all drain into the para aortic nodes, finally reaching the thoracic duct. Haematogenous spread is rare and occurs late to the lungs, liver and bones (1,2,3,8). In our patient the inguinal lymph nodes were involved at admission.

The most common symptoms of vulva cancer are pruritus, a visible or palpable mass, pain, bleeding, ulceration dysuria and vaginal discharge (9). The patient often becomes aware of a lesion on her vulva, but despite the superficial nature of the lesion, delay in seeking medical help is common. There can also be delay by the physician by treating other conditions without taking biopsy for histology 1,2,10. This patient had a one-

month history of a vulva swelling with no associated vaginal discharge, bleeding or jvsuria.

Staging is done using the modified International Federation of Gynaecologists and Obstetricians (FIGO) staging which is based on prognostic variables and takes into account the depth of invasion in early stage disease. The staging is as follows:

Stage I Lesions 2 cm or less confined to the vulva or perineum or both with no lymph node metastases.

IA: Stromal invasion less than 1mm

IB Stromal invasion greater than 1 mm.

Stage II: Tumour confined to the vulva or perineum or both - more than 2cme in greater dimension. No lymph node metastases.

Stage III: Tumour of any size with one or both of the following:

(a) Adjacent spread to the lower urethral and/or vagina and/or anus.

(b) Unilateral regional lymph node metastasis.

Stage IVA: Tumour invades any of the following:

Lpper urethra, bladder mucosa, rectal mucosa, or pelvic and / or bilateral lymph node metastases

Stage IV B: Any distant metastases

The patient presented had a tumor of more than 2cm, involving lower 1/3 of the vaginal and bilateral lymph node metastases and was staged as IVA.

Diagnosis of vulva carcinoma depends on biopsy and histological examination of the tumor. Small tumours treated by excisional biopsy. Colposcopy may be used to identify abnormal areas (after application of 3% acetic acid or 1% toludine blue) and biopsy of these areas done.

Once diagnosis has been confirmed, primary treatment for cancer of the vulva is surgical excision. Radical vulvectomy and bilateral inguinal lymphadenectomy involves a wide excision of the entire vulva and mons pubis as well as block dissection and removal of the inguinal and femoral nodes on both sides. This is essential even though the lesion is

unilateral because the lymphatic of the vulva communicate freely from one side to the other Limited surgery such as simple vulvectomy leads to poor results. Our patient was done modified vulvectomy (wide vulval excision) with bilateral lymphadenectomy.

Radical vulvectomy leaves a large surgical defect that is associated with marked disfigurement of the genital areas. Other complications include increased risk of venous thromboembolism, chronic leg edema (lymphedema), high rate of wound complications, psychosexual effects, urinary and stool incontinence, and pelvic relaxation which can lead to rectocoele and cystocele. In this patient, the wound healed well. She did not get any of above complications.

To decrease the associated morbidities and improve patient quality of life, surgical modifications have been described. Such modifications include selective groin lymphadenectomy and more conservative vulva resection in certain patient populations. Modifications in lymph node management include deletion of lymphadenectomy in selective patients, separate groin incision, unilateral lymphadenectomy unilateral lesions, dissection of sentinel lymph nodes, and the management of pelvic lymph nodes.

Use of radiotherapy for carcinoma of the vulva is indicated in cases of unresectable disease and when the inguinal nodes contain metastatic disease. Studies have been done, which confirm that radiotherapy can be used to cause tumour regression to a point where a more limited resection can be undertaken with sparing of organ function and improved quality of life.

Chemotherapy may be used in combination with radiotherapy in the treatment of carcinoma of vulva. This approach though under study, available results suggest a high rate of local control for locally advanced or recurrent disease. The most commonly used chemotherapeutic agents are 5-fluorouracil, mitomycin - c, and cisplatin. The most common morbidity associated with this type of treatment is mucositis in the vulvovaginal areas.

An important prognostic factor for vulval cancer is the stage of disease at the time of diagnosis. Survival is directly proportional to the extent of disease. According to the

Gynaecological oncology group study for vulval staging and survival, 5-year survival rates are as follows: 90% for stage I, 77% for stage II, 51.33% for stage III and only 18% for stage IV The overall 5- year survival of patient with vulva cancer is approximate 70% Another significant prognostic factor is the status of the groin nodes. Patients with negative groin modes have approximately a 90% survival, whereas patients with positive nodes have a survival of approximately 50% . This patient was in stage IV at diagnosis and therefore poor prognosis.

Patients with squamous cell carcinoma of the vulva develop recurrence after treatment in 15 - 40% of cases. The incidence of recurrence depends on original stage of the disease, depth of invasion and regional lymph node status. It is managed by resection and radiotherapy.

Careful follow up of patients should extend over the remaining years of the patient's life in order to detect recurrence early and treating it aggressively. All patients should be examined every three months for two years and every six months thereafter[1]

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GYNAECOLOGY CASE No. 14.

CANCER OF THE CERVIX STAGE IB-WERTHEIM'S HYSTERECTOMY

NAME:	S K.N	LMP:	20.05 2004
AGE:	42 YEARS	DOA:	03.06 2004
IP NO	0985926	DOD:	17 062004
PARITY:	3+1		

PRESENTING COMPLAINTS

S K.N was admitted through the gynaecology out-patient clinic with a 1 year history of abnormal per vaginal discharge and occasional intermenstrual bleeding.

HISTORY **OF PRESENTING COMPLAINTS**

She was well until 1 year prior to admission when she developed an abnormal per vaginal discharge. It was watery, whitish with slight occasional offensive smell. She also occasionally experienced intermenstrual bleeding over the same period. There was no history of dyspareunia or post coital bleeding.

She had her first sexual contact when she was 16 years old. She got married at 20 years of age and lived with the spouse for 5 years. They had two children but later were divorced due to husband infidelity. She remarried and stayed with the second spouse for 8 years, they had one child and she had one miscarriage. Unfortunately the spouse died due to a road traffic accident and she was widowed since then. She had been treated for sexually transmitted diseases severally during her first marriage.

Two weeks prior to admission into the ward she had been seen in the gynaecology out-patient clinic with the above symptoms. A speculum examination revealed small ulcerative lesions on the external os of the cervix with normal vaginal mucosa. A digital examination revealed an indurated cervix with small friable lesions on the external os. The adnexae and Pouch of Douglas were free and non-tender. There was blood on the examining fingers. The patient was then scheduled for examination under anaesthesia for staging and biopsy.

Examination under anaesthesia (EUA).

This was done a week prior to admission into the ward. In theatre anaesthesia was nduced and she was put in litothomy position. Vulvo-vaginal toilet was done and then she was drapped Aseptic catheterization was done and clear urine obtained. The external genitalia was noted to be normal. Using a Sim's speculum on the upper and lower side of the vaginal opening, the vaginal mucosa was inspected and was found to be normal. The cervix was exposed. The cervix was seen to have small ulcerative lesions all around the external os but not obliterating the os. A four quadrant biopsy was taken for histopathology examination. Digital examination revealed an indurated cervix, the fomices and parametria were free of induration. The uterus was normal size and the adnexae and Pouch of Douglas were free. Rectal examination revealed a free and mobile rectum with no masses felt. The space between the cervix and the pelvic wall was free of induration or nodularity. Cystoscopy and proctoscopy were not done. The stage of the tumour corresponded to stage IB. a vaginal pack was left in place to be removed after 6 hours. She was reversed from anaesthesia uneventfully and late in the evening the same day she was discharged home on antibiotics to come to the clinic for histopathology results.

Histology results.

Sections showed features of an invasive well differentiated squamous cell carcinoma of the cervix

She was informed of the results and the intended management. She was asked to do several investigations for the preparation of surgery and she agreed to do them as soon as possible and to be admitted to the cold gynecology ward with the results.

OBSTETRIC AND GYNECOLOGY HISTORY

She was a para 3+1. Her first delivery was in 1982 and her last in 1995 She had an abortion at 3 months in 1994, evacuation was done. Her menarche was at 13 years. LMP '-vason 25.05.2004. Her periods had been irregular with occasional intermenstrual bleeding. Initially she had a light flow of 2-3 days with a cycle of 28 days. She had used oral contraception during the second marriage for 1 year. She had not had a pap smear

PAST MEDICAL AND SURGICAL HISTORY.

This was non-contributory.

FAMILY AND SOCIAL HISTORY

She was widowed since 1998. She lived in Mlango Kubwa in Mathare. She was a casual iabourer with the Nairobi City Council. She neither smoked nor drank alcohol.

PHYSICAL EXAMINATION

She was a middle aged woman in good general condition and good nutritional status. She had no palor, jaundice, edema or lymphadenopathy. Her blood pressure was 119/75 mmHg, pulse rate of 80 beats per minute, respiratory rate of 20 per minute and temperature of 36.6 C.

ABDOMINAL EXAMINATION

The abdomen was not distended and it moved with respiration. It was soft and non-tender. There were no palpable masses.

PELVIC EXAMINATION

The external genitalia was normal

Speculum examination revealed normal vaginal mucosa. The cervix had small multiple ulcerative lesions all around the external os. The cervix easily bled on touch.

Digital examination revealed an indurated cervix around the external os. The uterus was normal in size and the adnexae and Pouch of Douglas were free and non-tender There was blood on the examining finger

The respiratory, cardiovascular and the central nervous systems were essentially normal.

DIAGNOSIS

Cancer of the cervix stage IB

MANAGEMENT

The patient was admitted having done the following investigations:-

Haemogram	WBC:	4.3 x 10/L
	Hb.	13.2 g/di
	Platelets:	190 x 10/L
Renal Function	Na+	135 mmo/L
Test	K+	4.2 mmol/L
	Urea	3.1 mmol/L
	Creatinine	78 umol/L
Liver Function	T. Bil.	6 mmol/L
Test	ALP	178IU/L
	SGPOT	18IU/L
	SGOT	16IU/L
	GGT	20IU/L
	T.Prot.	70 g/L
	Alb.	35 g/L
Intravenous		
Urogram (IVU)-	Normal	
Chest X-ray -	Normal	

She gave a written consent. Three units of compatible blood were made available. She was put on low fibre diet and given an enema on the eve of the surgery and repeated the following morning(operation day). She was pre-medicated with atropine 0.6mg and pethidine 50 mg intramuscularly half an hour before theatre and was then wheeled to **theatre.**

Wertheim's Hysterectomy.

In theatre general anaesthesia was induced and she was then repositioned to litothomy position and vulvo-vaginal toilet done. The urinary bladder was catheterized with a Foley's catheter and 150 mis of clear urine obtained.

Examination under anaesthesia revealed earlier findings. The cervix and upper vaginal were then painted with betadine solution and the patient repositioned to supine position. The abdomen was cleaned and draped. The abdomen was then opened via a midline subumbilical incision. The Pouch of Doulglas was clean. The uterus ovaries and fallopian tubes were found to be normal bilaterally. The liver, intestines, stomach, spleen, kidnevsand the omentum were noted to be normal. The para-aortic nodes felt normal on palpation. The intestines were packed away from the pelvis.

The round ligament on the right side was double-clamped close to the pelvic wall, cut and iigated using vicryl No. 1. The infiindibulo-pelvic ligament on the left was then divided betw een clamps and Iigated close to the pelvic wall. The anterior leaf of the broad ligament was then opened. A similar procedure was done on the right side. The posterior leaf of the broad ligament on the left side was reflected to reveal the pararectal space. The left ureter was identified, mobilized and marked with a ribbon tape. The uterine vessels were identified at their origin from the hypogastric vessels and then resected. The external and internal iliac arteries and the common iliac and obturator arteries were identified. Lymph nodes along the external iliac, common iliac and obturator arteries and para-aortic nodes were palpated and non-found to be enlarged All identifiable lymph nodes were dissected and recovered and sent for histopathology examination. A similar procedure was carried out on the right side.

The utero-sacral ligaments were then divided between the clamps and transfixed. The bladder was further deflected downwards until the upper 1/3 of the vagina was exposed. The vagina was then opened about 3 cm from the vault and cut all round preserving the lower 2/3 of the vagina.The whole of the uterus, ovaries, fallopian tubes and upper 1/3 of the vagina were delivered. The vaginal vault was then apposed with mattress sutures using Vicryl No. 1. Haemostasis was achieved and pelvic reperitonization was done carefully avoiding the ureters. The abdomen was closed in layers after correct count of swabs and instruments. The urethral catheter was checked and found to be draining clear urine and was retained. The estimated blood loss was 800mls and the patient received 1 pint of blood intra-operatively. She was successfully reversed from anaesthesia. The Ivmph nodes, were carefully labeled together with the uterus and ovaries and were taken for histopathology examination.

POST-OPERATIVE CARE.

She was wheeled to the recovery room where vital signs were monitored continuously until she was fully awake She was then wheeled back to the ward . She was started on intravenous crystalline penicillin G 2 MU 6 hourly, gentamicin 80 mg 8 hourly, flagyl 500mg 8 hourly. She was put on intramuscular pethidine lOOmg 8 hourly for 48 hours.

On the first post-operative day, bowel sounds were present and the catheter was draining clear urine She was started on oral sips. On the second day she was started on light diet and the catheter was removed On the third day she was on full diet and was changed to oral Augmentin lg 12 hourly, ponstan 500 mg 8 hourly and flagyl 400mg 8 hourly.The wound was exposed and found to be clean. Betadine solution was applied. She did well thereafter and on the 8th post-operative day she was discharged after removal of stitiches. She was to be seen in the GOPC in two weeks time.

FOLLOW-UP

She was seen in the GOPC as scheduled. She had no major complaints and was in good general condition. The incision site had healed well. The histology report showed no lymph node metastasis and no metastasis to the corpus uteri and ovaries. Sections of the cervix showed well differentiated squamous cell carcinoma of the cervix. The margins were clear of tumour This was discussed with her. She was given a 3 monthly schedule for 2 years for vault smears. She was also informed to come to the acute gynecology ward in case of vaginal bleeding or discharge, discomfort of pelvis, leg swelling, enlarged neck or groin nodes, difficulty in urination or defaecation.

Vault smears had been normal with no malignant cells seen on 2 subsequent visits. She had had no complaints and was still on follow-up at the time of reporting.

DISCISSION

S K.N was a para 3+1 who had presented with a one year history of abnormal per vaginal discharge and intermenstrual bleeding. Examination under anaesthesia staging and biopsy revealed carcinoma of the cervix stage IB Histology indicated invasive well differentiated squamous cell carcinoma of the cervix. She was managed by Wertheim's hysterectomy and recovered well.

Cervical carcinoma is the second most common malignancy in women worldwide with about 500,000 new cases diagnosed each year (1) It represents 18 % of deaths from gynaecological malignancies, and is the leading cause of cancer related deaths for women in developing countries (1). In the United States, cancer of the cervix is the third most common type of cancer in women after cancer of the breast and endometrium (2). The incidence is much lower in Jewish women perhaps because of hereditary immunity, circumcision of the male and better genital hygiene. In Kenya the exact incidence is unknown, but by hospital data, it is the commonest female genital malignancy in this country (3,4). Kaguta showed the malignant tumours of the cervix accounted for 71.5 % of all gynecological malignant tumours in the year 1974-1981 (3).

The average age of diagnosis is 45 years, but may occur even in the second decade of life and occasionally during pregnancy (2). Carcinoma of the cervix is believed to manifest at an earlier age in the tropics and other developing countries (5). In Kenya the peak age was 35-45 years (4). Our patient was 42 years.

The cause of cervical cancer is not known, however, epidemiological data demonstrates a direct relationship with sexual activity. Major risk factors observed include; sex at young age. multiple sexual partners, promiscuous male partners and history of sexually transmitted diseases. Cancer of the cervix is four times more frequent in prostitutes than other women and is exceptional in celibate women (2). Our patient had first sexual contact at 16 years, thereafter she had a promiscuous husband who infected her severally with sexually transmitted diseases and she had also two sexual partners.

Human papilloma virus (HPV) has been implicated as a possible carcinogen. HPV viral D\'A. has been detected in more than 80% of squamous intraepithelial lesions and

invasive cervical cancers (1). In view of the fact that HPV infections clear spontaneously within months to a few years and only a small proportion progress to cancer, this means that other crucial factors must be involved in the process of carcinogenesis Three main factors have been postulated to influence the progression. These include;

1 The type and duration of viral infection, with type 16 and type 18 found in 50-80% of SILS and up to 90% of invasive cancers.
2. Conditions that compromise immunity such as multiparity or poor nutritional status.
3. Enviromental factors such as smoking (1, 2,6,7).

Other sexually transmitted infections associated with cancer of the cervix include herpes simplex and Human Immunodeficiency Virus disease (HIV). The role of HIV infection in the pathogenesis is not fully understood However studies have shown a higher prevalence of HPV infection in HIV-seropositive women than in seronegative women The HPV prevalence was directly proportional to the severity of immunosuppression. Impaired lymphocyte function have been postulated to enhance latent or sub-clinical HPV activity resulting to a high rate or persistent infection (1).

The role of smegma has been controversial. Repeated application of human smegma on cervices of rodents had produced cancer of the cervix. A viral or chemical agent is thought to be transmitted through smegma (7).

Squamous cell carcinoma accounts for 70-80% of cervical carcinomas. Adenocarcinomas accounts for approximately 10-15% and the remainder are composed of sarcomas (0.5%) and undiffentiated carcinomas(6). The tumour is further classified histologically into three grades; Grade I- Well differentiated. Grade II-Moderately differentiated, and Grade Ill-Poorly differentiated (2,6). It has been found that the more undifferentiated the tumour, the higher the incidence of pelvic node metastasis. Our patient had well differentiated squamous cell carcinoma. There was no evidence of node metastasis after the Wertheim's hysterectomy.

Invasive cervical cancer begins as an intraepithelial lesion at the squamo-columnar junction at the transformation zone. Most cervical cancer probably begin as a dysplastic change with gradual progression over a period of several years to a pre-invasive form

i carcinoma insitu) (2). Carcinoma insitu, if left untreated will progress into invasive carcinoma in a period of between 1 and 20 years (2,8).

Carcinoma of cervix spreads principally by direct local invasion to the vagina, uterine cavity and laterally through the cardinal and utero-sacral ligaments. Laterally, extending carcinoma encompass and obstruct the ureters and ultimately cause hydroureter, h\dronephrosis and eventual loss of kidney function. Extension in to the bladder or bowel may result in vesico-vaginal or recto-vaginal fistulas (6). When lymphatics are involved, tumour cells are carried to the regional lymph nodes (Hypogastric, obturator, external iliac and sacral). The more pleomorphic or extensive the local disease, the greater the likelihood of lymph node involvement (2). Squamous cell carcinoma clinically confined to the cervix involves the regional lymph nodes in 15-20% of cases. When cancer involves the parametrium (IEB), tumour cells can be found in pelvic nodes in 30-44% of cases and in the para-aortic nodes in about 10% of cases (2).

It is important to estimate the extent of disease to aid in the plan of management and also in the prognosis. Staging is done according to the International Federation of Gynecologists and Obstetricians (FIGO) 1995 and is as follows;

Stage 0: Carcinoma in situ: Pre-invasive carcinoma

Stage I; Carcinoma confined to the cervix

la: Preclinical cancer. Microscopically diagnosed

Ial Less than 3 mm invasion

Ia2 3-5 mm in depth

lb: Lesion> 5mm invasion

Ibl Less than 4 cm

Ib2 Greater than 4 cm

Stage II: Carcinoma extended beyond the cervix but not to the pelvic wall and involves the vagina but not the lower third.

Ila: no obvious parametria! involvement

lib: obvious parametrial involvement

Stage III: Extension to the pelvic wall, involves lower one third of the vagina, all cases of renal involvement with hydronephrosis or non-functional kidney.

Ilia: no extension to the pelvic wall

Illb: pelvic wall extension and/or hydronephrosis

Stage IV Extension beyond the true pelvis or has involvement of bladder or rectum.

IVa: spread to adjacent organs

IVb: Distal spread

The patient presented had cancer of the cervix stage 1B2. In Kenya, most patients with cervical cancer present late. Ojwang in his series of patients less than 35 years of age with cancer of the cervix, found that only 7.4% presented in stage I and 19% in stage III (5). In another study, he reported that over 60% of patients were seen with stage III or IV of the disease (4). Rogo in his study found that 55% of his study population had stage III disease (9).

The most common symptom of invasive cancer is abnormal vaginal bleeding frequently intermenstrual bleeding. Other forms include; blood-stained leukorrheal discharge, scanty spotting or history of post-coital bleeding on specific questioning(2). Stage 0 and stage 1 are usually symptom-less and are discovered on routine pap smears (1,2,6). Advanced disease usually presents with pelvic pain radiating to the hip or thigh. Symptoms such as haematuria, haematochezia or fistula reflect local organ involvement. Pelvic wall involvement may present with a triad of leg edema, leg pain and hydronephrosis (1,2,6). Our patient presented with a history of abnormal vaginal discharge and inter-menstrual Bleeding. She had no stool or urinary incontinence. She had no leg edema. She actually Presented relatively early hence the good outcome.

On physical examination, patients with early-stage disease may look generally normal as **in** our patient. However in advanced disease, the patient may be in poor nutritional status and anemic. Cervical examination may reveal gross erosion, ulceration or a mass. These abnormalities may extend to the vagina. Digital exam may reveal an irregularity and firm consistency that may extend to the adjacent parametrium. Rectal examination may reveal an external mass or gross blood from tumour erosion depending on rectal involvement

(1.2,6) Depending on the stage of the disease, bimanual examination findings often reveal pelvic metastases. Leg edema suggests lymphatic/vascular obstruction from the tumour If the disease involves the liver some patients develop hepatomegaly (1,2,6). Our patient had cervical erosion with small ulcerative masses. All the other signs mentioned above were not elicited

Diagnosis is usually confirmed histologically by examining tissues taken from suspicious areas of the cervix as was done in our patient. Differential diagnosis include; cervicitis, endometrial carcinoma, pelvic inflammatory disease and vaginitis

Preclincal lesions are diagnosed by cytological examination or colposcopy. Suspect or positive pap smears calls for further investigations. About *6%* of cytological smears are falsely negative. Any suspicious lesion of the cervix is biopsied. Colposcopically directed biopsies with endocervical curettage or conization of the cervix may be necessary (2). Staging of the disease is based on examination under anaesthesia to determine the extent of the tumour involvement of other organs. In the same sitting, cystoscopy and proctoscopy should be performed to help rule out local invasion of the bladder and the colon. This was not done in our patient as the cystoscope and proctoscope were not available in the EUA theatre.

Other tests necessary include; a complete blood count, serum urea, electrolytes and liver tests to look for any abnormalities for possible metastatic disease. These were done in our patient and were normal. Imaging studies that can be performed for staging purposes include; chest X-ray, intravenous urogram, barium enema, ultrasonography and CT scan of the abdomen and pelvis to look for metastasis in the liver, lymph nodes and also help rule out hydroureter and hydronephrosis. Our patient had an intravenous urogram and a chest X-ray which were normal.

Treatment of cervical cancer varies with the stage of the disease. General measures include treatment of vaginal, urinary and pelvic infections, correction of anaemia, encouragement and provision of adequate oral food intake. Pain is controlled with analgesics such as NSAIDS and codeine (1,2,10).

Women with cervical dysplasia up to carcinoma in situ (Stage 0), should be treated with local ablative measures such as cryo-surgery, thermal cauterization, electro-cautery, laser ablation, cone biopsy or loop excision. Hysterectomy should be reserved for patients with other gynecological indications to justify the procedure However, in carcinoma in situ, when reproductive function is no longer desired, the most effective measure is by total hysterectomy. Conization is chosen when reproductive function is to be preserved (2). Follow-up is recommended with several vaginal/cervical smears after either treatment.

In invasive carcinoma, as in our patient, there are two effective methods of treatment, radiotherapy and radical surgery. Radiotherapy is more widely used because is applicable to all primary cervical cancer whereas radical hysterectomy is definitive only in stage I to stage Ila lesions (2,6,10). The overall 5-year cure rates for surgery and for radiotherapy in operable patients are approximately equal (2,10). Surgical approach to carcinoma of the cervix may be preferred in a young woman in whom preservation of the ovaries is important (2,10).

The surgical treatment of invasive cancer of the cervix consists of;

1. Extended hysterectomy without pelvic node dissection for stage Ial.
2. Radical hysterectomy with pelvic lymph node dissection(stage Ila).
3. Pelvic exenteration: anterior, posterior or total.

In our set-up patients with cervical cancer stage I to Ila are managed by radical surgery and if pelvic node metastases are found, this is followed by radiotherapy treatment Patients with stage lib to IV b are treated with radiotherapy. The patient presented was at stage Ib2 and was treated by radical surgery.

Radical hysterectomy requires the removal of the uterine cervix, parametria! tissues and upper vagina in conjunction with a pelvic lymphadenectomy from the bifurcation of the iliac vessels to approximately the level of the inguinal ligament(6, 10). The ovaries may be preserved in the young patients to avoid the need for replacement therapy. Patients with positive pelvic nodes, or positive surgical margins benefit from postoperative

combination of cisplatin-containing chemotherapy and pelvic radiation (1). Our patient had tumour free pelvic nodes.

Successful combination of external beam radiation and brachytherapy has ben used in the treatment of cervical cancer External radiation (teletherapy) is usually used first to diminish the volume of central tumour and increase the chances of later delivering a cancericidal dose with an intra-cavitary device. A standard regime is administration of 1 8-2 Gy (180-200 rads) per day, 5 days per week for 5 weeks, for a total dose of 50 Gy (5000 rads). Intra-cavitary radiation (brachytherapy) is done using caesurium at an optimal dose of 60-80 Gy (6000-8000 rads) (2). Palliative radiation can also be used individually to control bleeding, for pelvic pain or for urinary or partial large bowel obstruction from pelvic disease (1,10).

More recent studies have demonstrated that adjuvant treatment of patients with advanced disease with chemotherapy (cisplatin) in combination with radiotherapy and or surgery significantly improves the survival of these women (11,12.13,14). Other agents used include; 5-fluorouracil and 1-fosfamide with response rate of approximately 20%.

Prevention of the cancer of cervix includes the use of barrier methods of contraception. This is presumably secondary to lessened exposure to HPV (15). Increased intake of micro-nutrients and other dietary factors such as carotenoids are associated with decreased risk. Prevention of morbidity and mortality from cervical cancer also involves early recognition and treatment. Risk factors must be recognized and patients at risk followed up closely. Universal cytologic screening of all post-pubertal women must be continued on a regular basis. They should be performed annually after the onset of sexual activity and after 3 consecutive negative results, the screening interval may be prolonged Women older than 60 years should continue with pap smear screening.

The prognosis of carcinoma of the cervix depends on the stage of the disease In general, the 5-vear survival rate for stage I disease is higher than 90%,stage II 60-80%, stage III 50% and stage IV is less than 30 % (1). Renal failure and uraemia are the leading cause of death and are a result of bilateral ureteric obstruction by tumour.. Less frequent causes

of death are hemorrhage, infection, pulmonary embolism, intestinal obstruction and liver **failure (2).**

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GYNAECOLOGY CASE NO. 15

LONG-TERM REVERSIBLE CONTRACEPTION-JADELLE INSERTION.

Name: S **P**		LMP:	10.05.2005
Age: 33 years		FP Client:	1480/05
Last Delivery: 18.01.2005		Parity:	2+0
		Date of insertion:	13.05.2005

PRESENTING COMPLAINT

S P had come to the family welfare clinic as a new client in search of a family planning method She had used the intrauterine contraceptive device from the year 2000 to 2004 but had it removed due to heavy menses and intermittent pelvic pain. After counseling on various methods, she opted for Jadelle

OBSTETRIC AND GYNAECOLOGY HISTORY

She was a para 2+0 with both deliveries being spontaneous vertex deliveries at a Nairobi hospital The First was in 1999 a male infant and the other in 2005 a female infant both of whom were alive and well. Menarche was at 15 years. Menses were regular every 30 days for a period of 3-4 days.

PAST MEDICAL HISTORY

This was non-contributory

FAMILY AND SOCIAL HISTORY

She was a married secretary and stayed with her husband in Kahawa Sukari. She did not smoke or drink alcohol. Her husband was a security officer. There was no chronic illness in the family

GENERAL EXAMINATION

She was in good general condition. She did not have jaundice, edema, lymphadenopathy or cyanosis. Her blood pressure was 120/80mmHg, pulse rate of 80 beats per minute which was regular and of good volume. Her temperature was 36.7 °C and weight was 71

ABDOMINAL EXAMINATION

The abdomen was not distended and moved with respiration. There were no palpable masses or organomegaly.

PELVIC EXAMINATION

There was normal external genitalia. The cervix was firm, posterior with a parous os. The uterus was anteverted, normal size. Adnexae and pouch of Douglas were free and non-tender.

INVESTIGATION

Pap smear- Showed satisfactory smear with no abnormal cells seen. Endocervical cells present.

MANAGEMENT

She was counseled about insertion, advantages and side effects of Jadelle.

INSERTION

The procedure was explained to her. She was placed in supine position with her left arm outstretched on a table by the side.

The upper arm was cleaned and draped with sterile towels. The insertion site was identified approximately 8 cm above the elbow. 5mls of 1% lignocaine hydrochloride was infiltrated in the insertion site in a 'V-shaped' fashion. The 'V' consisted of two injection positions 2mm apart facing the axilla.

Using the scalpel, a 2mm long incision was made at the apex of the 'V". The trocar was inserted in the incision and pushed subdermally upwards towards the axilla up to the second mark at the position of the first injection site.

The first implant was inserted into the trocar with the thumb and index finger and pushed up with the plunger until there was resistance.

The plunger was steadied and the trocar was removed gently up to the first mark leaving **the rod** in situ but not totally removing the trocar under the skin. The position of the first

rod was fixed using the left forefinger and the trocar again advanced along the side of the finger to form a 'V' pattern. This ensured a suitable distance between the implants (1-2mm) The second implant was inserted and pushed with the plunger until resistance was encountered. .An approximate distance of 5mm from the incision site to the ends of the implants was ensured to prevent expulsion. The trocar was then removed and the edges of the incision were approximated using an elastoplast bandage The site was covered with gauze and wrapped with bandage to ensure hemostasis.

She was advised to keep the site dry and remove the bandage after 48 hours and leave the elastoplast until her appointment after seven days. She was advised to avoid unprotected sex until after 24 hours.

FOLLOW UP.

She was seen as scheduled and the elastoplast was removed. The incision site had healed and the implants could be felt. She was then seen after 1 month and then after 2 months. She had no complaints and was advised on six monthly visits.

DISCISSION

S P was a 33 year old para 2+0 who needed a long term reversible contraception. She had used the IUCD but had it removed due to heavy menses and intermittent pelvic pain and discomfort. A new formulation levonorgestrel, Jadelle, was then inserted.

Jadelle was introduced in Kenya in 1986, with regulatory approval obtained on August 8th 1989 (1). The method had been approved in 27 countries by 1992. Jadelle was approved for marketing as a three-year method in the United States in 1996 and in Finland in 1997 (2). In 2000 Finland approved the extension of use of the method to five years. In 2003, the FDA approved extension of use to five years (2).

Prior to the introduction of Jadelle (also known as Norplant-2), the six capsule Norplant system was the most widely used long term progesterone method in our country. The Norplant system consisted of six match stick size capsules, 34 mm long and 2.4mm wide made of silastic capsules each containing progestin levonorgestrel 36mg in crystalline form. The capsules are inserted under the skin of the woman's upper non-dominant arm in a fan shaped pattern using a simple trocar. The hormone is released at a steady state starting at 85 ug per day and decreasing to 50 ug at 9 months, 35 ug at 18 months and 30 ug thereafter (3).

The Jadelle system consists of two rods, therefore it is easier to insert and remove compared to Norplant. Rods ditfer from capsules. Each Jadelle rod is 43 mm long and 2 5mm in diameter (1cm longer and 0.1mm thicker than Norplant). Each rod contains 75mg of levornogestrel for a total of 150mg, while six Norplant capsules contain a total of 216mg. Both the capsules and rods have outside sheaths composed of silicone rubber but they are made differently. In the Norplant capsule, levornogestrel crystals are packed within the rubber sheath which is then sealed at each end. In the Jadelle rod a core of mixed levonorgestrel and elastomer (a polymer having elastic properties of natural rubber) is enclosed within the rubber sheath, which is then sealed at each end with medical adhesives Norplant and Jadelle are bioequivalent and are used for 5 years (4).

Jadelle is one of the most effective reversible contraceptives available. It causes thickening of cervical mucus, anovulation in up to 50% of users, accelerated ovum erowth and when ovulation occurs degeneration of the corpus luteum The cumulative rregnancy rate in clinical trials was 0.3 % for 3 years and 1.1% for 5 years (5) The pregnancy cumulative rate for Norplant after 5 years is 3 .7% (6)
Jadelle has lower failure rate than the pill and most IUCDs with a comparable efficacy to that of surgical sterilization.

In the clinical studies on which approval was based, cumulative continuation rates were 83 3% after the first year, 60.6% after 3 years and 41.5% at 5 years (7). Women discontinue using Jadelle because of side effects, because they want to become pregnant or for other personal reasons. Studies conducted in western countries indicate that, over a three year period, 14.1 per 100 women stopped using Jadelle because of menstrual irregularities and 14.7 per 100 women discontinued for other medical reasons, 9.7 per 100 women did not continue for the full three years because they were planning a pregnancy (8). Medical occurrences most frequently cited as reasons for removal were headaches, depression, weight gain or hair loss.

Jadelle should not be used by women who are or suspect to be pregnant. Some of the contraindications include; Thrombophlebitis, thromboembolic disorders such as blood clots in legs or lungs or eyes, undiagnosed abnormal genital bleeding, acute liver disease, non-cancerous or cancerous liver tumors, known or suspected breast cancer, history of idiopathic intracranial hypertension, hypersensitivity to levornogestrel or any of the other components of the rods (e.g. silicone elastomer). It is relatively contraindicated in patients on drugs that affect liver enzymes such as phenobarbitone, rifampicin and anticoagulants (9).

Insertion is usually done within seven days of menses, after abortion or 6 weeks post panum (10). Protection from pregnancy is provided within 24 hours of insertion. The woman rapidly returns to her normal fertility when the implants are removed. Despite the effectiveness, safety and patient satisfaction with Jadelle, it is not widely used in the L'nited States and a few western countries for fear of constant litigation. This is based on the silicon content producing "illness".

Insertion site complications include infection, bleeding, expulsion of rods and pain. The cost **is** also slightly higher at the time of insertion. Local studies on Jadelle need to be conducted to determine aspects of its use **in** our country

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OPERATIVE GYNAECOLOGIC LAPAROSCOPIC SURGERY AT THE KENYATTA NATIONAL HOSPITAL: A RETROSPECTIVE CASE ANALYSIS.

SUMMARY

Objective: **To describe the utilization of the laparoscope in operative gynecological surgery at the Kenyatta National Hospital.**

Design: A retrospective case analysis

Subjects: Two hundred and twenty one cases of operative gynecologic laparoscopic surgery which **were** undertaken from January 1998 to December 2004 were analyzed Data was collected through a questionnaire which was filled in from the clients' files from the records department. Both doctors and nurses' cardex notes were used Files that were not retrieved or had no notes or incomplete notes (18.0%) were excluded Analysis was done using statistical package for social sciences.

Results: There were seven hundred and eighteen cases in gynecology where the laparoscope was used between January 1998 and December 2004. Four hundred and seventy five (66 1%) were diagnostic while two hundred and forty three (33.8%) were operative. Operative procedures done via the laparoscope included; Tuboplasty 25.1%, adhesiolysis 45 3%, management of ectopic pregnancy 2.1%, ovarian cystectomy 4.8%, ovarian drilling 14.2%, myomectomy 18%, laparoscopic assisted vaginal hysterectomy (LAVH) 3 .3% total laparoscopic hysterectomy 0.3%, laparoscopic drainage of pelvic abscess 1 ***2%*** and ablation of endometriotic foci 1.2%. Seven point two percent of the patients had conversion to laparotomy intra-operatively. Intra-operative complications included; bowel injury 18%, bladder injury 0.9%, vascular injury 3.6% anaesthetic complications 0.9% and equipment malfunction 0.9%. The mean blood loss was 267±143.02mls. Five patients (2.3%) required blood transfusion. The mean operation time was 115±46.56 minutes. The mean time in the recovery room was 38.99±17.98 minutes There were major post-operative complications which included; bowel injury 18%, respiratory complications 1.4%, ureteric injury 1.8%, bladder injury 1.8%, wound sepsis 0.5% and mortality of 0.5%. The patient died due to complications of bowel injury.

Conclusions:Despite the early installation of laparoscopic equipment at **KNH,** it's optimal utilization is yet to be achieved. Being the largest teaching and referral hospital in the country and in this region the cases done via the laparoscope are still very few. The

laparoscope in gynaecological surgery was mainly used for elective cases. Very few emergency cases especially ectopic pregnancies, ovarian cysts and drainage of pelvic abscesses were done. This contributes to the gross under utilization of the laparoscope. The operative times are still long and complications relatively high compared to other centres worldwide. Though the trend is promising more surgeons need to be trained and re-trained in order for minimal access surgery to take root at the institution.

Recommendations: More emphasis needs to be put to advocate minimal access surgery at the Kenyatta National Hospital More surgeons need to be trained and re-trained in laparoscopic procedures. More emergency gynaecological cases need to be undertaken iaparoscopically. Workshops should be organized to give in-service training to gynecologists who are interested in laparoscopic surgery. Collaboration with other institutions both local and abroad where our professionals can get training and experience needs to be undertaken seriously. Laparoscopic surgery is now incorporated in the curriculum of postgraduate training in gynecology. This is a trend in the right direction but more emphasis needs to be made on minimal access surgery in postgraduate training in gynecology and general surgery.

ABBREVIATIONS

EBL	Estimated blood loss
KNH	Kenyatta National Hospital
LAVH	Laparoscopic Assisted Vaginal Hysterectomy
MAS	Minimal Access Surgery
PONV	Post-Operative Nausea and Vomiting
PPV	Positive Pressure Ventilation
CO2	Carbondioxide
BTL	Bilateral Tubal Ligation
TLH	Total laparoscopic hysterectomy

DEFINITIONS

Laparoscopy: This is the viewing of the peritoneal cavity providing direct vision of the internal female genitalia through an endoscope under illumination.

Operative gynecologic Laparoscopic surgery Laparoscopic procedure on the female reproductive organs and The pelvis where a surgical intervention was done.

Key words: Gynaecology, laparoscopy, operative, surgery

INTRODUCTION

The term laparoscopy is derived from two Greek words, "Lapara" which means "the soft parts of the body between the rib margins and the hips' and "skopein" which means "to see, view or examine".

Though the first description of endoscopy came from the Kos school led by Hippocrates (460-375 BC), it is generally accepted that it was Phillip Bozzini in 1806 who gave the first impetus to modern endoscopy (4). He developed a complex system which conveyed light from a lamp through a tube into the vagina to illuminate it and allow observation of the cervix through a second channel.

Hysteroscopy was the first gynaecological endoscopic procedure to be attempted but most were futile since the cystoscopes lacked lenses and were using light from a candle reflected through a tube. The development of the telescopes with lenses began in the late nineteenth century, and in 1910 Jacobeaus of Sweden introduced a Nietze cystoscope in the peritoneal cavity and coined the term laparoscopy (4).

The development of the cold light source in France and the Hopkins rod lens system in England enabled the safe transmission of light and optimal visualization that is a prerequisite for laparoscopic surgery. Early attempts at laparoscopic surgery were primarily directed at simplified methods of female sterilization, pioneered mainly in continental Europe by Palmer and Fragenheim who are generally regarded as the fathers of modem laparoscopy (4).

Hope, from the USA in 1937, described the use of a laparoscope for the diagnosis of extra-uterine pregnancy. Bosch (1936) in Switzerland and Anderson (1937) in the USA suggested the use of coagulation or figuration to perform tubal sterilization (4). The first requirement for laparoscopy is the safe provision of a pneumoperitoneum. The Verress needle used for insufflation was devised by JanosVeress in 1938 (5).

The control of intraperitoneal bleeding is a prerequisite for any surgery and in the case of laparoscopy can usually be achieved by mono- or bipolar electrocoagulation or by thermal coagulation. Monopolar electrocoagulation had been introduced in the early 1950's for tubal sterilization and this was the method most commonly used for almost 30 \ears. Complications resulting from electrical burns led to the development of the bipolar coagulation by Frangenheim (1972) in Germany and Rioux and Coulier (1974) in Canada and thermocoagulation by Semm in Germany (6)

Laser was introduced to laparoscopic surgery by Bruhat et al (1979) in France, Tadir et al (1981) in Israel and Daniell and Brown (1982) in the USA. This technique allowed for more complicated laparoscopic operations, particularly extensive adhesiolysis and the destruction of endometriotic foci (6).

Major milestones in the development of laparoscopy has involved safe peritoneal insufflation, cold light illumination, laparoscopes with rods lens system, instruments for the safe manipulation of pelvic organs, techniques to ensure haemostasis, laser, video monitoring and more recently double-optic laparoscopy, initially for salpingoscopy and subsequently to perform microlaparoscopy in early peritoneal endometriosis and ovarioscopy and intra-ovarian surgery in cases of ovarian endometriosis.

EQUIPMENT, INSTRUMENTATION AND ENTRY TECHNIQUE.

Recent technical advances in endoscopic equipment has led to the possible replacement of over 75% of gynaecological laparotomies by operative laparoscopic surgery (3). Constant advances in techniques and instrumentation have established an operative system which provides the greatest efficacy for the surgeon and at the same time the greatest safety to the patient. Operative laparoscopy requires the following equipment:

1. Insufflator.

A pneumoperitoneum of constant pressure and volume is mandatory for operative laparoscopy. The use of a verres needle with an electronically controlled insufflator, provides an initial insufflation pressure of 25mmHg and a flow rate of 1 litre per minute, followed by laparoscopic confirmation using a 10mm, 0 or 30 degree laparoscope through a 10mm flap valve Trochar, to indicate adequate intraperitoneal placement. The pressure is then maintained at 15mmHg and the flow rate increased to 20 litres per minute.

2. Laparascope and Light source.

In the standard operative laparoscopy, a 5mm or 10mm, 0 or 30 degree angled laparoscope is used. A straight laparoscope can also be used. An optimal light transmission is provided by a fluid light cable The light source must generate 150W and is adequately provided by the Endo-Illumination Apparatus.

3. Endocoagulator.

The advent of destructive heat at a temperature of only **100 °C** as an effective method of haemostasis has become an integral part of operative laparoscope For this reason the endocoagulator together with its attachments, the crocodile forceps, the point coagulator, and the myoma enucleator are necessary Endocoagulation prevents the risk of uncontrollable burning which can occur with the application of monopolar or bipolar **high**-frequency current.

4. Instruments.

A full range of instruments allowing the surgeon to perform the operative steps are necessary. Included are instruments for perforation, dilatation, grasping, cutting, aspiration, instillation, morcellation, haemostasis, drainage and emergencies.

5. Suture and suture materials.

With the advent of endosuture, endoloop, endoligature and the intra- and extra-corporeal operative knotting techniques, classical methods used at laparotomy were introduced to laparoscopic surgery and have become a mainstay.

6. Aquapurator

In order to maintain a clear view of the operative field, pelvic irrigation and lavage necessary This is adequately provided by the monofilar-bivalent irrigational system, the Aquapurator or the CO2 Aquapurator with its attachments. This system allows for

effortless instillation and aspiration of physiological saline at 37 °C during the operation 2-4 litres of normal saline are kept in a warm water bath set at 37 °C; a second bath set at 50 °C is used for cleaning and prevention of fogging of the pelviscope.

7. Pertubation apparatus.

Endoscopic tubal diagnosis and surgery requires an initial controlled pertubation using either C02gas or a blue dye solution. (methylene blue). This is performed using the pertubation apparatus which is connected to a cervical vacuum adaptor which also allows for easy manipulation of the uterus.

8. Aids for surgeon.

The surgeon must work in a relaxed and comfortable position. An optic holder with a flexible arm attachment, an operative stool and a shoulder rest are obligatory

ENTRY TECHNIQUE.

The patient is placed in the dorsal litothomy position and draped after the induction of anaesthesia and preparation of the abdomen and pelvic areas. The bladder is emptied by catheterization to decrease the risk of injury during subsequent introduction and use of other instruments. .After a careful bimanual examination, a tenaculum is attached to the cervix, and a tubal insufflation cannula is inserted into the cervical canal and finally fixed to the tenaculum so that it can be used as a "handle" to maneuver the uterus. A 1-cm incision is made within or immediately below the umbilicus: A verress needle is inserted through this incision into the peritoneal cavity. Carbon dioxide is then introduced and monitored by the pneumatic insufflator. The amount of gas insufflated will vary with the

patient's size, the laxity of the abdominal wall, and the planned procedure. In most patients, 2-3 L of gas will be needed to obtain adequate visualization. The maximum insufflation pressure should not exceed 20mmHg. The needle is widthrawn and the laparoscopic trocar and cannula inserted. After proper abdominal entry, the trocar may be widthrawn and replaced with the fibre-optic laparoscope. The examiner manipulates the intrauterine cannula so that the pelvic organs can be observed. A second trocar with a cannula may be inserted under direct laparoscopic vision through a 5-mm transverse midline incision at the pubic hairline. Electrical cutting forceps or an aspiration probe may be used through the second cannula. Additional punctures are utilized as necessary for the placement of other instruments. Surgical knots may be tied and sutures placed using specially made equipment. The operation is terminated by evacuating the insufflated gas through the cannula, followed by removal of all instruments and placement of a 3/0 sub-cuticular suture for wound closure. A small dressing is applied to the wound.

LITERATURE REVIEW.

OPERATIVE GYXAECOLG1C LAPAROSCOPIC SURGERY.

ADVANTAGES AND DISADVANTAGES.

The laparascope has become an invaluable tool in both diagnostic and operative gynaecology The advantages of operative laparoscopy are now clear and have been documented in various clinical trials(7). Avoidance of a laparotomy incision results in less operative pain and less need of analgesia. With smaller wounds and less pain, patient recovery is rapid and are able to resume their normal activities in a shorter period(7,8). This leads to shorter hospital stay and hence reduction in hospital costs. Small wounds also means less blood loss during surgery and a better cosmetic result.

.Also noted is a significant reduction in postoperative adhesion formation following operative laparoscopic surgery compared to laparotomy(9). In addition, the brilliant and magnified views that modern equipment provides allows the precise definiton of anatomy and pathology, and with a newer generation of instruments, constantly being developed and re-developed, facilitate accurate surgery(lO).

Development of the video monitoring system has the added advantages of improving the coordination between the surgeons and the assistants, magnifying the operative field, maintaining interest of theatre staff as well as being useful for teaching.

A randomized study done in Finland looked at twenty five women who underwent hysterectomy by laparoscopy and another twenty five by laparotomy. They found less estimated blood loss for laparoscopy (157± 104 mis) compared to laparotomy(268± 137mls). Mean haemoglobin drop was lower in the laparoscopic group (19± 8 g/dl)

compared to the laparotomy group (27± 13 g/dl) (p= 0.006). The mean length of hospital stay was 2.1± 0.3 days in the laparoscopic group and 3 4± 0.7 days in the laparotomy group (p< 0.001). The mean sick leave was 21± 7 days and 39± 6 days respectively (p< 0 001). There was no significant difference in the rates of complications between the two groups (24% vs. 25% respectively) (11).

In a Canadian study looking on cost analysis of tubal anastomosis by laparoscopy versus laparotomy, costs for operating room were similar, pharmacy costs and expenses were less in the laparoscopy group. The mean total cost for laparoscopic and tubal anastomosis was less ($ 861 ± S 137) than tubal anastomosis by laparotomy (S 1348 ± $ 188) (p< 0.001) (12).

A study in Hungary of 335 patients to compare the length of time needed for the patients to feel themselves completely free of complaints, found the estimated average healing time is 29.8 days for laparotomy and 18.9 days for diagnostic and or operative laparoscopy The duration of the healing process was found to be independent of laparoscopic findings and the type of procedure(13).

Operative laparoscopy is not without disadvantages. The time length of operative laparoscopic surgery is longer than the standard open procedures particularly in the early learning curve phase Equipment costs are higher so is the cost of training. Prolonged surgery has led to the problems of operator fatigue and discomfort. But these have largely ?een overcome by repositioning the patient, the use of body platforms for support, and Inally video monitoring instead of through-the-lens-viewing.

THE SCOPE OF OPERATIVE GYNAECOLOGIC LAPAROSCOPIC SURGERY

Therapeutic options open to the laparoscopic surgeon are many and varied, from simple idhesiolysis to the resection of dense endometriotic tissues and the removal of relatively arge benign tumors such as fibroids. Procedures can be classified according to the organ involved or based on the complexity of the procedure.

TABLE 1

PROCEDURES CLASSIFIED BASED ON ORGAN INVOLVED

A. TUBAL	B. OVARIAN	C. UTERLNE
1 Adhesiolysis 2 Tuboplasty 3 Ectopic pregnancy. • Salpingostomy • Salpingectomy • Salpingotomy 4 Tubal ligation 5 BTL Reversal.	1. Ovarian biopsy 2. Ovarian drilling 3. Cystectomy 4. Opherectomy	1. Myomectomy 2. Laparoscopic Assisted Vaginal Hysterectomy j. Total Laparoscopic Hysterectomy 4. Uterine suspension

CLASSIFICATION BASED ON COMPLEXITY OF THE PROCEDURE

Martin and Diamond have in the recent past suggested a useful classification of rrocedures based on their complexity (14).

TABLE 2

A. **Basic operative laparoscopy**	**B. Intermediate operative laparoscopy**	**C. Extensive operative laparoscopy**
1 Tubal sterilization 2 Biopsies 3 Coagulation of mild endometriosis 4 Aspiration of small ovarian cysts	1. Lysis of mild to moderate adhesions 2. Coagulation of moderate endometriosis 3. Exploration of small ovarian cysts 4. Uterine suspension 5. Salpingectomy 6. Salpingectomy for ectopic pregnancy	1. Cuff salpingostomy 2. Salpingotomy for ectopic pregnancy 3. Lysis of extensive adhesions 4. Excision of moderate to severe endometriosis 5. Enucleation of ovarian cysts (endometriosis. dermoids) 6. Oopherectomy 7. Myomectomy 8. Tubal anastomosis 9. Laparoscopic assisted vaginal hysterectomy 10. Total laparoscopic hysterectomy

Yat May **Wong (1999**) noted that an increasing number of gynaecological procedures are now being competently managed laparoscopically rather than by laparotomy(15). The American Association of Gynecologic Laparoscopists has recommended that the majority of patients with pelvic pathology requiring surgery can be managed laparoscopically(16). Whatever the procedure, however, both the surgeon and the patient must be prepared for failure and the need for laparotomy, since in any given unit 10 to 15% of all cases may inadvertently be converted to laparotomy(17).

Parkar conducted a retrospective review of the procedures he had performed between July 1996 and June 2000, at various private and public hospitals in the Coast Province of Kenya. He performed a total of 697 cases but he reviewed a total of 596 cases (85.5%). The caseload increased from 2.4 per month to peak at 27.5 per month in 1999. This was apparent as surgical competence improved, improved equipment was acquired, and the procedures became increasingly acceptable. The surgical procedures undertaken included adhesiolysis (45 9%), salpingectomy for ectopic pregnancies (3.1%), salpingostomy for tubal infertility (13.6%), ovarian drilling (14.6%) for polycystic ovarian disease, myomectomy(9.8%) and ovarian cystectomy(7 8%). The operating time averaged 71-80 minutes in 35.9% of the procedures, the shortest being 42 minutes and the longest being 130 minutes. The time recorded was from scope entry to scope removal. In 20 6% of the clients no previous surgery had been undertaken, 244(40.9%) had previous diagnostic laparoscopy, 172 (28.9%) had undergone a laparotomy for various indications and 78(13.08%) had previous caesarean sections (18).

For any surgery to be successful and safe there must be an unimpeded view of the operative field. For this reason, particularly after previous surgery, adhesiolysis is often

the first and at the same time essential preamble to more extensive procedures. While the lysis of adhesions can be the simplest of maneuvers, it can potentially be the most difficult and is fraught with the danger of trauma to surrounding tissues and organs In 408 cases of laparoscopic surgery undertaken at the Aga Khan hospital in Nairobi, (2) adhesiolysis of pelvic adhesions comprised 34.55% of the procedures done In his review, Parkar performed a total of 274(45.9%) cases of adhesiolysis involving the uterine tubes (18). Pelvic adhesions secondary to infections are a common pathology in this region, and form a majority in the female with compromised fertility.

Lundorff et al (9) documented that postoperative adhesions developed more significantly and more often following laparotomy than after laparoscopic surgery.($p>0.0001$).

The management of ectopic pregnancy has undergone marked changes in the past decade Goldner et al (**10)** reported a four fold increase in the incidence of ectopic pregnancy in the United States between 1970 and 1989, with a fall in the mortality from 35.5 to 3.8 deaths per 10,000 cases. In the United Kingdom the increase of ectopic pregnancies doubled from 4.0 to 9.6 per 1000 pregnancies between 1973 and 1993, whilst the mortality decreased from 16 to 3 per 10,000 cases**(19).**

Sau et al recommended that laparoscopic surgery be the preferred option to laparotomy in the management of haemodynamically stable patients with ectopic pregnancies**(20).** At the Aga Khan Hospital, Nairobi, **(2)** 33 cases of ectopic pregnancies (8.08%) were managed laparoscopically between May 2000 and May 2002. Of these 10 were salpingostomy, 21 salpingectomy and 2 salpingotomy.

In an **earlier review by Parker, (18) a** total **of** 28 (**4.69%**) ectopic pregnancies **were** handled laparoscopically out **of 596** laparoscopic surgical cases performed in the Coast Province **of** Kenya. **19** patients **(3.1%)** underwent salpingectomies while **9** patients (15%) underwent salpingostomy. Yao and Tulandi, **(21)** reported in a randomized controlled trial that there was no doubt that the laparoscopic approach in the management of tubal ectopic gestation is associated with a significantly less blood loss, lower analgesic requirement, shorter hospital stay, lower costs and quicker post operative recovery. The subsequent intrauterine pregnancy rate was **61** % when compared to 52% after laparotomy

The recurrent ectopic pregnancy rate was also lower in the laparoscopic group(8%) than following laparotomy (14 %) (21).

Sylvia et al in a prospective study, compared the reproductive outcomes following laparoscopic salpingectomy and salpingostomy. The intrauterine pregnancy rate were **60%** and 53.3%, and the recurrent ectopic rates were 18.3% and 7.7% respectively(22).

In a series at the Aga Khan Hospital Nairobi, (**2)** 33 (8.08%) cases of ectopic pregnancies were handled laparoscopically out of the 408 cases performed by minimal access surgery Of these, tubal conservation was undertaken in 12 cases, while 21 cases had a salpingectomy done**(15).** Yap Lip Kee reported a swing of suitable surgery undertaken laparoscopically in the management of ectopic pregnancies from less than 5% in the 1990 to over 93% in 2000 **(23).**

There is no doubt that worldwide today, the laparoscopic management of ectopic pregnancies is justifiably regarded as the gold standard. In the Aga Khan series 2 of the 33 cases was ectopic gestation unruptured, while the rest had significant

-aemoperitoneum. The presence of pelvic adhesions can be yet a limiting factor. In view of the above reasons, patients who are haemodynamically compromised, should only undergo laparoscopic intervention if the operating team has acquired adequate experience.

.Although laparoscopic surgery is now the preferred choice in the successful management of ovarian cysts, there is some concern about the risk of cyst rupture in undiagnosed cases of malignancy. Hulka et al (16) noted only 53 (0.4%) cases of unsuspected ovarian cancer amongst 13,739 cases of laparoscopic ovarian cyst surgery, and the risk is therefore small.

In the Aga Khan series **(2)** there were 69 cases (16.91%) of ovarian cystectomy undertaken, and with all specimens subjected to histological evaluation, there were no cases of malignancy reported. However, there were 8 cases of ovarian endometriosis or endometriomas which were confirmed histologically. These cases were adequately handled laparoscopically. There were 3 cases of dermoid cysts(one twisted and gangrenous) which were managed by laparoscopic oopherectomy and delivery by colpotomy. Nineteen cases(4.65%) of polycystic ovaries were encountered and were handled laparoscopically using a monopolar needle electrode. In the review by Parkar, (18) ovarian cystectomy was undertaken in 46 (7.8%) cases while cystostomy was done in 21 cases(3.5%). Although the pelvic ultrasound was the primary investigation to rule out malignancy, a few patients had normal serum CA 125 levels.

The feasibility and safety of laparoscopy in the management of ovarian cysts and masses have been well demonstrated and documented**(24 , 25).**

N'ehzat et al. in 1991 recommended that myomectomy using the laparoscopic approach was justified provided that patients are carefully selected, the surgeon has had appropriate training, the equipment quality is not compromised and the theatre team is experienced(26) The technique of laparoscopic myomectomy was popularized and refined by Jean Bernard Dubuisson in Paris, who used a combination of monopolar and bipolar electosurgery and intracorporeal sutures to achieve adequate haemostatsis and tissue apposition(27).

Miller from Chicago used the ultrasonic (harmonic) scalpel with particular usefulness during myomectomy and made the procedure surprisingly bloodless(28).

Between May 2000 and May 2002, 63 cases of laparoscopic myomectomy were undertaken at the AgaKhan Hospital, Nairobi, (2) the smallest being 2cm and the largest 18cm. Three (4.76%) were pedunculated, 12 (19.04%) intramural and the rest (76.20%) were sub serous. In general anterior wall or fundai fibroids were easier to remove than those on the posterior wall. The maximum number of fibroids removed were 5, although laparoscopic surgery was attempted only when there were 2-3 fibroids on the pelvic ultrasound. Myoma extraction from the peritoneal cavity was undertaken by morcellation in **58** cases(92.06%), posterior colpotomy in 4 cases(6.34%), and by a minilaparotomy in 1 case Haemostasis was adequately achieved in all cases using bipolar, monopolar coagulation and intracorrporheal sutures In his review, Parkar performed a total of 59 myomectomies and 2 myolysis due to uterine myoma.

Laparoscopic hysterectomies of various types have now been performed for over a decade since they were first reported in 1989(29). Fernandez et al. noted that before the introduction of laparoscopic assisted vaginal hysterectomy (LAVH) at the Antoine

Beclere Hospital in France, 42% of all hysterectomies were vaginal and 58% abdominal(30) After the introduction of LAVH, in three years the procedure accounted for 20% of the hysterectomies which resulted in a concomitant decrease in the rate of abdominal hysterectomy(30).

In the Aga Khan series(2), 62(15.19%) cases of laparoscopic assisted vaginal hysterectomy and nine cases(2.20%) of total laparoscopic hysterectomies were undertaken. Chaparon and Dubuisson noted that the fact that most hysterectomies for benign disease are still being carried out by the abdominal route is ample justification for laparoscopy to have a role in hysterectomy(31).

Brechin et al (32), of the Scottish Laparoscopic Surgery Audit Group, evaluated the use of LAVH by a cross section of gynaecologists in Scotland Of the 505 cases of LAVH done from April 1994 to March 1996, 463(91.7%) were successfully undertaken. Although the patient compliance was remarkable, the intraoperative complications encountered included bladder perforation(0.6%), ureteric injury(0.2%) and haemorrhage(2.8%). The laparotomy conversion rate was 1.0%. There were no cases of bowel injury(32) The initial reviews of laparoscopic hysterectomies mentioned complications similar to the standard complications of hysterectomy, including urinary and bladder injuries(33).

Worldwide majority of the hysterectomies for benign uterine disease are still being carried out by the abdominal route (33). Dicker et al. (34) and Wilcox et al.(35) in extensive reviews showed that less than 30% of all hysterectomies were performed vaginally.

COMPLICATIONS OF OPERATIVE GYNAECOLOGIC LAPAROSCOPIC SURGERY.

Laparoscopic surgery presents new potentially life-threatening complications that are -sually not seen with traditional "open" approach It is significant that the benefits of laparoscopic procedures be weighed against potential complications. The known rate of mtra-operative and post-operative complications in adequately trained hands ranges from 1-5% and the mortality between 4-8 deaths per 100,000 cases(36).

The training and experience of the surgeon performing the procedure influences the complication rate. Because of the learning curve associated with laparoscopic procedures, the incidence of complications should decrease after approximately 30 to 50 procedures**(37).** In a large multicenter study in France, **29,996** cases of diagnostic and operative laparoscopic gynecological cases were reviewed in seven centers over a period of **9** years. The mortality rate was 3.33 per 100,000 laparoscopics. The rate of complications requiring laparotomy was 3 per 1000(96 cases). 1 out of 3 complications **(34** 1%:43 cases) occurred while setting up for laparoscopy, and 1 out of 4 complications(28.6%) were not diagnosed during operation**(38).**

Querleu et al.(39) classified complications in laparoscopic surgery as potentially fatal(vascular, intestinal), or non-lethal intra-operative(urinary tract, minor vessel injury) and post-operative complications(infections , thromboembolism).

Severe and even fatal vascular injuries can occur during surgical instrumentation, particularly during insertion of the veress needle or trocar. Incidence of vascular injury in laparoscopic surgery is estimated to be 0.64% with 80% of these injuries occurring in the

mesosalpinx(40) Insertion of veress needle or trocar into major vessels such as the aorta, common iliac vessels or inferior vena cava has been reported(41). Injuries to the vessels in the abdominal wall (e.g. superficial and deep hypo gastric vessels) is common because of use of multiple tocar.

Bowel injuries have been reported to occur in between 0.1% to 4% of cases(39). In a large Japanese study bowel injury was reported to occur in 0.06% to 0.4% of cases with a mortality rate of 5% (40). Electrocautery and laser can cause thermal injury to the viscera and abdominal vessels (42) These injuries are a major cause for morbidity and mortality. Brosens and Gordon(43), in a multinational survey estimated the prevalence of bowel injury to range from 1 in 1652 procedures to 1 in 280 procedures depending on the level of experience attained by the surgeon.

Injuries to the bladder and ureters are rare with a reported incidence of 2 per 10,000 cases(44). Previous abdominal surgery and congenital anomaly of the urinary tract increases the risk of urinary tract injuries. Prevention and early recognition of urinary tract injuries is critical because this reduces the severity of these complications. The presence of adhesions either from previous surgery infections or endometriosis predispose to injury both bowel and vascular.

Post-operative wound and peritoneal infections are usually minimal but can occur especially in the elderly , obese, diabetic and immunocompromised patients. Although uncommon, with an incidence of 0.1%, an incisional hernia can develop at the trocar insertion sites because of inadequate re-approximation of wound, pre-mature suture disruption and infection(45). Injury during access or during the surgical procedure were

statistically more significant when the surgeon performed fewer than 100 laparoscopics a vear<43).

In the Aga Khan Hospital, Nairobi,(2) there were 5 (1.22%) cases of intraoperative conversion to laparotomy. There were 2 cases of herniation(one small bowel and one omental). Both patients had laparotomies after day seven post-operatively. Although there were no cases of class I bowel injuries (entry related injuries by verres needle or trochar), there were 3 (0.73%) cases of class II injuries (Injuries acquired whilst operating, during release of dense adhesions). In the earlier review by Parkar (18), he performed a total of 697 cases and reviewed 594 cases after one week. In this series 99 5% had no complications either during the procedure or at subsequent 1 week review There were 2 cases of intra-operative port bleeds (0.34%) and one case of port abscess 4 patients underwent conversion to laparotomy (0.67%), and in one obese patient the procedure of Trochar/ Verres needle insertion failed. Of the 596 cases reviewed, 312 (52.3%) spent one night in hospital, while 4 patients (0.67%) spent more than three nights in the wards. These were the cases that underwent intra-operative conversions to laparotomies. All in all, 234(39.3%) women conceived following laparoscopic adhesiolysis, tuboplasty, ovarian drilling and / or myomectomy.

Laparoscopic surgery presents unique anaesthetic challenges. Gas insufflation to produce pneumoperitoneum can compromise respiratory and cardiac function and contribute to rain The Trendelenberg (head-down position) can further compromise cardiac and respiratory capacity, increase the risk ofregurgitation and nerve injuries. The incidence of anaesthesia-related complications during laparoscopy is relatively low.

An earlier study in the United States reported that anaesthesia related complications accounts for approximately one third ofthe reported deaths associated with laparoscopic tubal ligation(46). The most common anaesthesia-related causes of death were attributed to hypoventilation and cardiopulmonary arrest. Misplacement of the veress needle can lead to subcutaneous emphysema with an increased surface area for carbondioxide diffusion leading to hypercapnia and respiratory acidosis(47). Extensive subcutaneous emphysema can occur involving the neck and face resulting in gas tracking to the thorax and mediastinum, therefore resulting in pneumothorax or pneumomediastinum.

Haemodvnamic changes in laparoscopic surgery also warrant anaesthetic considerations. The combination of Trendelenburg (head down) position and the pneumoperitoneum increases venous return and cardiac out-put and this leads to increase in arterial blood pressure Hypotension , dysrrthmia and cardiac arrest can occur due to alteration of arterial blood pressure. The incidence ofacute cardiovascular collapse during gynecologic laparoscopic surgery in Germany was reported to be 1 in 2000, with a mortality rate of 1 in 10,000 in early 1970's which decreased to 1 in 100,000 in the late **1970's** (48). The incidence ofdysrrythmia during laparoscopy is approximately 14% (49).

Bradyarrythmia, including significant bradycardia, atrioventricular dissociation, nodal rhythm and asystole have been reported. These are attributed to vagal stimulation caused by insertion of veress needle or the trocar, pneumoperitoneal induced peritoneal stretch, stimulation of the fallopian tubes during bipolar electric cauterization or C02 embolisation. The induction of pneumoperitoneum with the patient in the horizontal position (rather than in head-down position) can decrease the severity of these haemodynamic changes.

With the diaphragm displaced upwards, lung volume is reduced, there is a decrease in pulmonary compliance and an increase in peak airway pressure. Obesity and pre-existing cardio-pulmonarv dysfunction exacerbate these changes The increase in minute ventilation required to maintain normocapnia further increases peak airway pressure. The major pulmonary complications during laparoscopic surgery include significant hypoxaemia and hypercapnia.

In addition there is increased risk of lung injury owing to increase alveolar pressure Cephalad movement of the chin during creation of pneumoperitoneum and the Trendelenburg position can lead to endobronchial intubation. The position of the tracheal tube should therefore be reassessed after creation of the pneumoperitoneum and the position change. Pneumothorax is a rare but potentially life- threatening complication of laparoscopic surgery. It can occur during veress needle insertion and C02 insufflation.lt can occur with gas traversing into the thorax either through a tear in the visceral peritoneum or in a congenital defect in the diaphragm(patent pleuroperitoneal canal).

There have also been reports of pneumomediastinum and pneumopericardium during laparoscopy. It is proposed that the mechanisms of pneumomediastinum are similar to that of pneumothorax. Pneumopericardium can occur either when the CO2 is forced through the inferior venacava into the mediastinum and pericardium or when CO2 through the defect in the membranous portion of the diaphragm, which can have embryonic communication between the pericardial cavities.

Gas embolism is a rare but potentially lethal complication of laparoscopic surgery. Profound hypotension, cyanosis and asystole have been described following embolism. The incidence of gas embolism during laparoscopy is difficult to ascertain because the criteria for diagnosis of gas embolism are inexact. Most commonly embolism episodes occur during creation of pneumoperitoneum. Gas embolism can occur through a tear in a vessel in the abdominal wall or on the peritoneum. Embolism can also occur due to inadvertent placement of the veress needle directly into the parenchymal organ

Laparoscopic surgery is associated with a high mini-morbidity One early survey in 1984 (50) found that over 95% of patients had some symptoms at 24 hrs with neck and shoulder pain (80%) and abdominal pains (70%) being most common. A high percentage of patients also reported sore throat (49%), back ache (29%), nausea (22%) and weakness (10%). Many of these symptoms were still present on the second post-operative day and 31% of patients reported that they would have preferred not to have been discharged (50).

Improvement in anaesthetic technique can help because a more recent survey (1994) found similar rates of abdominal pains but shoulder pains (45%), sorethroat (26%), headache (12%) and nausea (3%) all were less common on first post-operative day and only 8% of the patients would have preferred not to have been discharged (51).

Several initial studies reported a lower incidence of pulmonary complications after laparoscopic surgery compared with open surgery (5% versus 25%) (52). However recent studies have not observed any difference in post-operative pulmonary function between laparoscopic and open surgery (53). The most likely causes of pulmonary impairment in post-operative period are diaphragmatic dysfiinction(because of stretching from pneumoperitoneum and C02 insufflation) and mediocre pain relief. Upward displacement of the paralyzed hemi-diaphragm following creation of pneumoperitoneum can cause pulmonary atelectasis, which cannot be clinically apparent peri-operativelv owing to Positive Pressure Ventilation(PPV). With resumption of spontaneous ventilation, the patient can become hypoxemic and require PPV until the collapsed lung can be re-expanded.

The lithotomy and Trendelenburg position impede blood flow in the lower extremities and result in venous statsis. Prolonged surgery in the lithotomy position can cause a lower extremity compartmental syndrome. High intra-abdominal pressure(>20mmHg) can cause compression of the terminal veins and reduced terminal vein flow velocity can increase the potential for deep venous thrombosis and pulmonary embolism. Perhaps the minimal tissue damage and early ambulation associated with laparoscopic procedures reduces post-operative venous stasis. Adverse patient positioning during lithotomy and Trendelenburg position can result in brachial nerve injuries (54).

Post-operative nausea and vomiting (PONV) is extremely common after laparoscopic surgery. The symptoms are unpleasant and can delay discharge. Some of the causal factors such as peritoneal gas insufflation and bowel manipulation are essentially

unavoidable. Some aspects of anaesthetic technique could influence the incidence of PONV, therefore, a low risk technique should obviously be used whenever possible.

The armantareum available to the surgeons of the future increases daily, and no doubt minimal access surgery (MAS) is the way forward. Majority of the surgeons in Kenya would require additional training and skills development in the modalities of MAS As time goes by and as more specialists embrace the new methods, MAS will gain a remarkable reputation in Kenya. As we enter into the new millennium it would be worthwhile and befitting to see the new generations of gynaecologists taking up the challenges of MAS in our region and keeping up with the trends worldwide.

I

CONTRAINDICATIONS TO LAPAROSCOPIC SURGERY

As for all surgical procedures, there are well defined absolute and relative contraindications to laparoscopic surgery

ABSOLUTE CONTRAINDICATIONS

- Mechanical and paralytic ileus.
- Large abdominal mass.
- Generalized peritonitis.
- Irreducible external hernia.
- Cardiac failure.
- Recent myocardial infarction.
- Cardiac conduction defects.
- Respiratory failure.
- Severe obstructive airways disease.
- Shock.

RELATIVE CONTRAINDICATIONS.

- Multiple abdominal incisions
- Gross obesity
- Hiatus hernia
- Ishaemic heart disease
- Blood dyscrasias and coagulopathies.

STUDY JUSTIFICATION.

Laparoscopic surgery is relatively new in Kenya. The advantages of laparoscopic surgery have long been documented and various clinical trials have attested to this. At the turn of the century, local Kenyan data on this new technique has slowly been emerging, though it is still scarce The data obtained on laparoscopic surgery has been mainly from the private hospitals in Nairobi. More so, the studies have included both data from general and gynaecological surgery.

This study intends to specifically describe the utilization of the laparoscope in operative gynaecological surgery in the largest public referral hospital in this country. Not only will it reveal the trend of operative laparoscopic gynaecological surgery in KNH, but it will elucidate on the usefulness of the laparoscope in gynaecological surgery in a public hospital. From the results it will be possible to assess the impact of the laparoscope in gynaecology and to look into the future needs in this field of surgery.

STUDY BROAD OBJECTIVE.

To describe the utilization of the laparoscope in operative gynaecological surgery at the Kenyatta National Hospital.

STUDY SPECIFIC OBJECTIVES.

1 To determine the total number of cases done and case rate per month over the 6 year study period

2. To determine the procedures done and the indications for the procedures.

3 To determine the prevalence of both intra-operative and post-operative complications

4. To determine the intra-operative failure rates of the procedures done.

5. To determine the average hospital stay after laparoscopic surgery

6. To give recommendations based on the findings of the study

STUDY METHODOLOGY

A. Study Design

Hospital-based, descriptive retrospective case analysis.

B. Study Area and Study Population.

The study was conducted at the Kenyatta National Hospital. This is the national referral hospital and also serves as the University of Nairobi teaching hospital. It is the largest public hospital in the country located 3 kilometers north of Nairobi city centre The hospital is the training centre for postgraduate students in Obstetrics and Gynaecology among other degree training programmes. It has a bed capacity of 2,000 and serves a population of more than 2.5 million people living in Nairobi and its environs.

The hospital's Department of Obstetrics and Gynaecology is managed by staff employed by both KNH and the University of Nairobi. The Department is housed in the hospital complex and consists of a labour ward, 3 antenatal and postnatal wards, an emergency gynaecology ward, and one cold gynaecology ward. Recently a private maternity wing has been introduced in the hospital to cater for private patients. The staff also manage the gynaecology, antenatal and postnatal out-patient clinics. The family planning clinic and the high risk adolescent clinic are also incorporated in the Department of Obstetrics and Gynaecology

Patients who undergo laparoscopic surgery are first seen in the gynaecology out patient clinic or the family planning clinic. Here they are clerked by a senior house officer where the presenting medical history, the past medical history, family and social history are recorded. A physical examination is performed and the findings recorded. Relevant laboratory and radiological examinations are ordered and once they are satisfactory then

the patient is scheduled for theatre. The senior house officer then liaises with the consultant running the laparoscopy theatre and an operation day is then scheduled

In theatre the time the operation begins and the time it ends is recorded. The procedure notes are then documented after the operation. The operation is then recorded in the theatre registry book. There after the patient's progress in the ward postoperatively is noted in the file The nurses also document the patient's progress in the nurses' cardex. A discharge summary is then written by the senior house officer. Results of investigations done including histopathology results are filed in then patient's file. Patient's files are then kept in the central records department where they can be retrieved easily.

C. Sample Size

The equipment to perform operative laparoscopic surgery was installed in KNH in the second half of the 1990's. Initially it was mainly used for diagnostic purposes. Preliminary inquiries indicated that the bulk of operative gynaecologic laparoscopic surgeries were done from the year 1998, though very few procedures were done prior to this year The sample size thus included all patients who underwent operative gynaecological laparoscopic surgery at Kenyatta National Hospital between January 1998 to December 2004.

D. Inclusion Criteria

1 All cases done operative gynaecological laparoscopic surgery between January 1998 to December 2004 at the Kenyatta National hospital

E. Exclusion Criteria

1. Non-gynaecological laparoscopic surgery done within the specified period
2. Diagnostic gynaecological laparoscopic surgery.
3. Cases whose notes were incomplete or partially or completely lost.
4. Cases done prior to January 1998 and after December 2004.

DATA COLLECTION.

Prior to data collection the principal investigator went through the theatre registry He then identified and recorded all the file numbers of patients who underwent operative gynaecological laparoscopic surgery within the specified period being studied

The files were then retrieved from the central records department. Data was collected by completion of a set out questionnaire by the principal investigator. The set out questionnaire was divided in broad categories namely:

a. Sociodemographic data
b. Gynaecological data
c. Intraoperative data
d. Post-operative data

DATA ANALYSIS

The raw data from the questionnaire was compiled and entered into the computer using the SPSS program. Totals, means, modes and frequencies were calculated. Data analysis was done through cross tabulation of the variables of pre-operative, intra-operative and post-operative data versus the different types of operative gynaecological laparoscopic procedures done and their indications. The data was summarized in the narrative form, pie charts, histograms, tables and graphs.

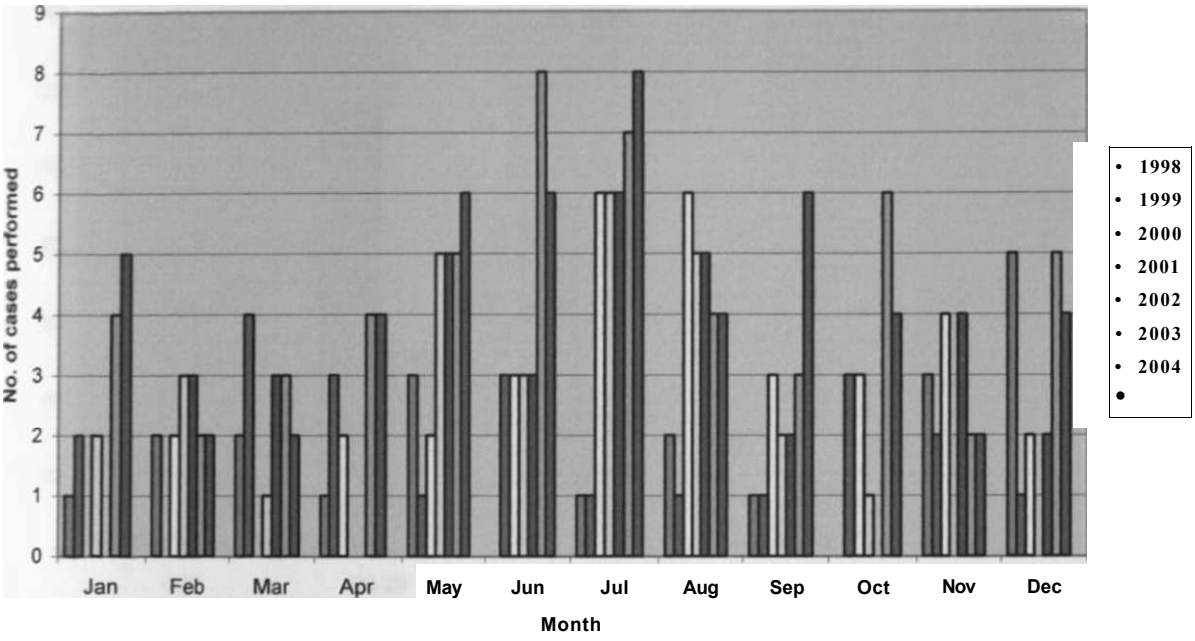
STUDY LIMITATIONS.

1. Data collection covered the period January 1998 to December 2004 Operative gynaecological laparoscopic procedures done prior to this date were not included, though the numbers were expected to be very small according to the preliminary inqueries.
2 The study did not take into consideration the level of experience of the surgeons. This has been known to have an effect on the outcome, not only on laparoscopy but also on other invasive procedures.
3 The study did not describe the long term outcomes of the various procedures done

ETHICAL CONSIDERATIONS

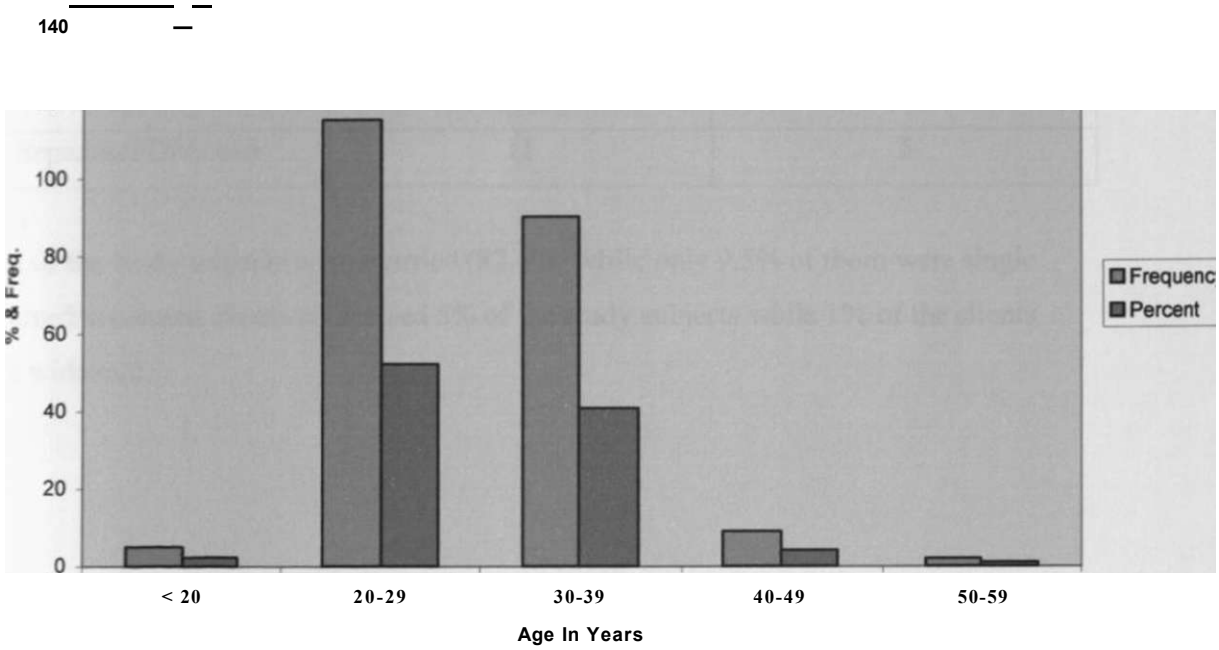
- The study was conducted on approval by the Kenyatta National Hospital Ethical and Research Committees.
- Data and information was treated with utmost confidentiality and names of clients and their surgeons was excluded from the questionnaire.
- There was no need for consent from the patients since this was a retrospective study with no risks
- The hospital and the clients together with the Department of Obstetrics and Gynaecology and the Ministry of Health stand to benefit from this study as recommendations have been given based on the findings of the study.

Chart 1: laparoscopic surgery cases by month



A total of 243 operative gynecologic laparoscopic procedures were done over the 6 year period. An average of 3 cases were done per month and an average of 20 cases per year. The trend of operative cases done increased over the years The case load rate increased from 1.3 per month to peak at 5.3 per month in 2004. This was due to increased number and experience of the surgeons and more patients opting for laparoscopic surgery. A total of 12 files were not retrieved and 10 files had no notes or the notes were incomplete. Analysis was therefore done on 221 files.

Chart 2: Age



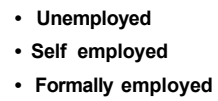
The age range of the subjects was 18-59 years with a mean of 29±6.047 (1SD) years. 115 clients (52%) fell in the 20-29 age group category. 90 clients (40.7%) fell in the 30-39 age category, 9 (4.1%) in the 40-49 age group category, 5 (2.3%) in the < 20 age group category and 2(1%) in the 50-59 age group category.

TABLE 2: Marital Status

Characteristic	Frequency	Percent
Single	21	9.5
Married	186	84.2
Widowed	2	1
Separated/Di vorced	11	5

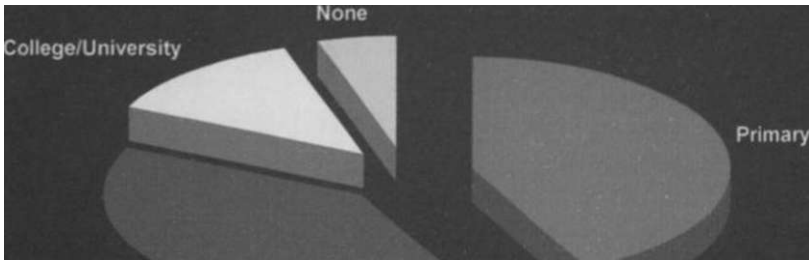
Most of the study subjects were married (82.4%) while only 9.5% of them were single. Divorced/separated clients comprised 5% of the study subjects while 1% of the clients were widowed.

Chart 3: Occupation status



Most of the study subjects were housewives/ unemployed (35.7%), while 33.9% of the study subjects were self employed. Only 28.1% of the subjects were formally employed.

(hart 4: Education Status



Majority of the study subjects had primary education (41.6%), while 38.5% had secondary education. Clients who had college or university education were 12.7% while 5% of the clients had no formal education.

TABLE 3: PARITY

Characteristic	Frequency	Percent
Nullipara	102	46.5
Para one	55	24.9
Para two	28	12.7
Para three	21	9.5
> Para 4	15	6.8

The parity ranged from 0-6 with a mean of 1.8. Nulliparous women comprised 46.2% of the study subjects while subjects who were para 4 and above comprised of 6.8% of the study subjects.

TABLE 4: LAPAROSCOPIC SURGERY INDICATIONS.

INDICATION	FREQUENCY	PERCENT
Primary Infertility	86	34.4
Secondary Infertility	87	34.8
Desired Family Size	3	1.2
Ectopic Pregnancy	7	2.8
Pelvic Inflammatory Disease	10	4
Chronic Pelvic Pain	9	3.6
Uterine Fibroids	13	5.2
Polycystic Ovarian Disease	16	6.4
Ovarian Cyst	12	4.8
Dvsfuctional Uterine Bleeding	6	2.4
Endometriosis	1	0.04

Table 4 shows the various indications for operative gynecologic laparoscopic surgery The most common indication for operative gynecologic laparoscopic surgery was secondary infertility (34.8%) which was followed closely by primary infertility (34.4%). Endometriosis as an indication accounted for only 0.04% of the cases

TABLE 5: OPERATIVE PROCEDURES DONE.

PROCEDURE	FREQUENCY	PERCENT
Bilateral Tubal Ligation	3	0.9
Adhesiolysis	150	45.3
Tuboplasty	83	25.1
Removal of Ectopic Pregnancy	7	2.1
Ovarian Cystectomy	16	4.8
Ovarian Drilling	47	14.2
Myomectomy	6	1.8
Laparoscopic Assisted Vaginal Hysterectomy	11	3.3
Total Laparoscopic Hysterectomy	1	0.3
Drainage of Pelvic Abscess	4	1.2
Ablation of Endometriotic Foci	3	0.9

A total of 331 operative gynecologic laparoscopic surgeries were done over the study period. This comprised 40.5% of all laparoscopic procedures done over the study period Adhesiolysis was done in 45.5% of the cases. This was in combination with other procedures especially tuboplasty. Adhesiolysis performed alone accounted for 19 3% (64 cases) of the total cases done.

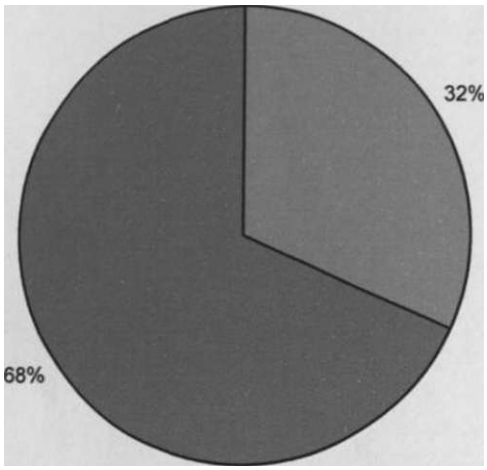
There were 83 cases (25.1%) of tuboplasty performed for primary and secondary infertility. The commonest procedure done during tuboplasty was cuff salpingostomy. Others included gutter salpingostomy, salpingotomy and neosalpingostomy

There were 7 cases (2.1%) of removal of ectopic pregnancy. In 4 cases of ectopic pregnancy, salpingectomy was done, while in 2 cases salpingostomy was done and 1 case had salpingotomy. Ovarian cystectomy was performed in 16 (4.8%) of the total cases. The size of the ovarian cysts ranged from 5cm diameter to 12cm diameter
47 (14.2%) cases of laparoscopic ovarian drilling were performed. The indications were primary and secondary infertility and in cases where a diagnosis of symptomatic ovarian cyst had been made.

Myomectomy for uterine fibroids accounted for 1.8% of the cases done. There were 11 (3 3%) cases of laparoscopic assisted vaginal hysterectomy(LAVH) done in this series. The indications were uterine fibroids dysfunctional uterine bleeding and abnormal cervical cytology. There was only one (0.3%) case of total laparoscopic hysterectomy

Pelvic abscesses were also drained using the laparoscope. There were 4
(1 2%) cases of pelvic abscesses which were drained. Ablation of endometriotic foci was performed in 3 cases which comprised 0 9% of the cases done. There were also 3 cases of laparoscopic tubal ligation (BTL) and this comprised 0.9% of total operative laparoscopic gynecologic surgeries done.

Chart 5: Previous Abdominal Surgery



The chart above shows that 32% (70 cases) had previous abdominal surgery while 68% (151 cases) had no previous abdominal surgery.

Chart 6: Type of Surgery

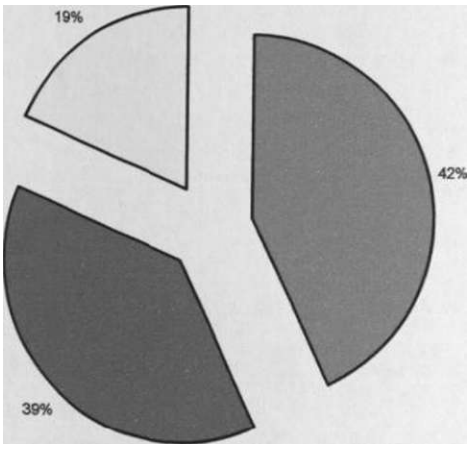


Chart 6 shows that 42% of the subjects had laparotomy, 39% had minilaparotomy and 19% had caesarean section.

chart 7: Intraoperative conversion to laparotomy

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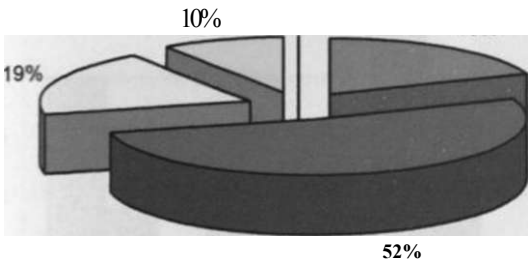


- **Intraoperative complications**
- **Inability of laparoscopic procedure to adequately treat condition**
- **Failed 1st entry**
- **Equipment malfunction**

There were 16 (7.2%) cases where laparoscopic surgery was abandoned and open laparotomy done. In 19% conversion to laparotomy was due to failed 1^{sl} entry, 19% due to intra-operative complications, 52% due to inability of the laparoscopic procedure to adequately treat the condition and in 10% it was due to equipment malfunction.

Chart 8: Major intra-operative complications

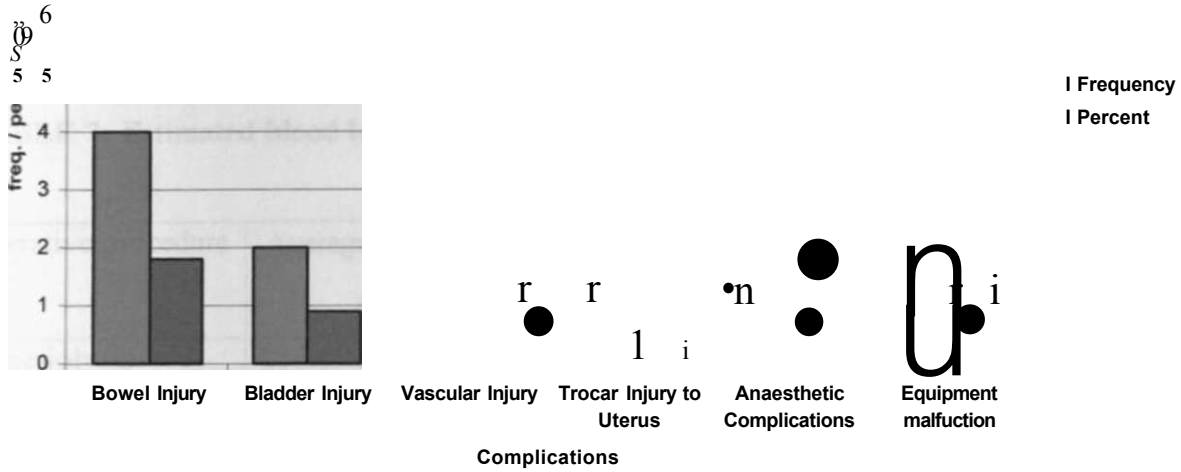


Chart 8 shows the intra-operative complications during laparoscopic surgery. Intra-operative complications occurred in 18 (8.1%) of the cases analyzed. The rate was 1.8% for bowel injury. 0.9% for bladder injury, 3.6% for vascular injury, 0.5% for trocar injury to the uterus, 0.9% for anaesthetic complications and 0.9% for equipment malfunction. There were no mortalities encountered intra-operatively.

TABLE 6: Complication rates by previous abdominal surgery

Intra-operative complications												
Previous abdominal surgery	Anesthetic complications		Vascular injury		Bowel injury		Bladder injury		Non recorded		Total	
	N	%	N	%	N	%	N	%	N	%	N	%
Yes	1	1.4	3	4.3	4	5.7	2	2.9	60	85.7	70	31.6
No	0	0	5	3.4	0	0	0	0	142	95.9	149	67.4
Total	1	0.5	8	3.7	4	1.8	2	0.9	202	92.7	219	99.1

Table 6 above shows the intra-operative complication rates by previous abdominal surgery. Bowel and bladder injury occurred in patients with previous abdominal surgery. Anesthetic complications also occurred in patients with previous abdominal surgery.

Vascular injury occurred in both groups but slightly more in patients who had not had previous abdominal surgery Patients with previous abdominal surgery suffered more intra-operative complications (14 3% Vs. 4.1%) compared to those who had not had previous surgery and this difference was significant (p = 0 007 X^2 = 7.32.).

TABLE 7: Estimated blood loss by laparoscopic procedure

Operative procedure	Average EBL±1 SD (mis)	No. of cases EBL recorded	No. that had blood transfusion.
Adhesiolysis	218.18 ± 175.03	32	1
Tuboplasty	206.66 ± 167.72	20	-
LAVH	252.00 ± 110.73	11	-
TLH	350.00 ±0.00	1	-
Ectopic pregnancy	303.33 ±206.55	8	1
Myomectomy	333.33 ±57.73	3	-
Ovarian drilling	104.00 ±63.87	10	-
Ovarian cystectomy	275.00 ±35.35	6	1
Drainage of pelvic abscess	280.01 ± 18.85	2	2
TOTAL N=221	Mean= 267.46±143.02	93 (42.1%)	5 (2.3%)

There were 93 (42.1%) files with EBL recordings. The mean blood loss was **267** 46±143.02 mis. On average the highest EBL recorded was from total laparoscopic hysterectomy (350±0.00mls) while the lowest was on ovarian drilling (104.00±63.87mls). There were 5 (2.3%) laparoscopic procedures where blood transfusion was required.

TABLE 8: Duration spent in theatre by laparoscopic surgery

Operative procedure	Procedure time mean time (mins)± 1 SD	Time in recovery room mean time (mins)	N
Adhesiolysis	110.3 ±49.9	40	58
Tuboplasty	152.5 ±34.2	48	37
LAVH	165 ±24.6	54	11
TLH	185.0 ±0.0	45	1
Ectopic pregnancy	125.0 ±38.9	45	7
Myomectomy	166.6 ±20.8	58	3
Ovarian drilling	70.6 ±26.3	27	18
Ovarian cystectomy	123.3 ±35.1	45	9
Bilateral Tubal Ligation	67.5 ±31.8	50	2
Drainage of pelvic abscess.	121.6 ± 56.8	65	**J**

Table 8 above shows the average amount of time spent in theatre for the various laparoscopic procedures undertaken. The overall trend is increased operation time with increased complexity of the procedure. Total laparoscopic hysterectomy took the longest time (185.0±0.00 mins), while the shortest procedure was bilateral tubal ligation **(67**.5±38.1 mins). The mean time in the recovery room for all the cases analyzed was 38 99 minutes. There was no difference in time spent in the recovery room for the various cases done.

Chart 9: Post-operative mini-morbidity

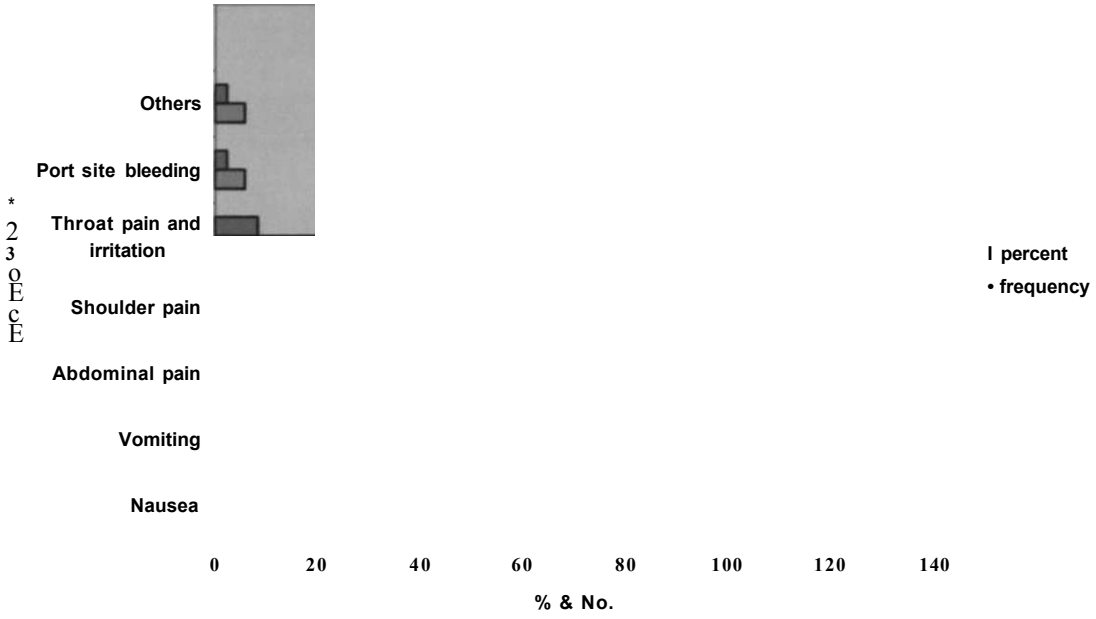
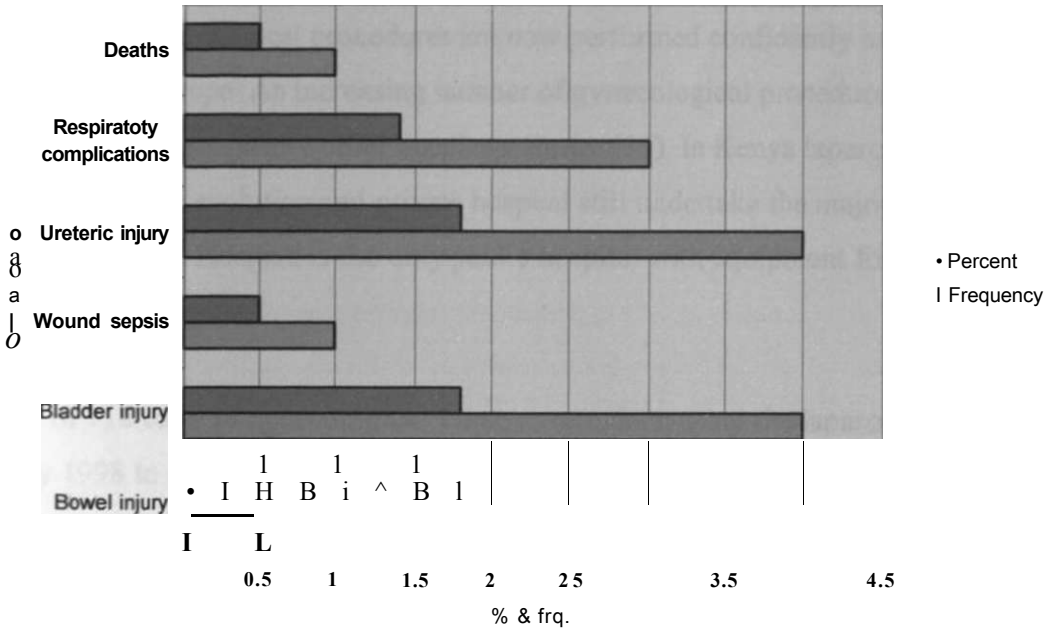


Chart 9 above shows the rate of mini-morbidities recorded for the study subjects. The commonest mini-morbidity was abdominal pain (50.2%) followed by nausea (19.4%), shoulder pain (9.3%), throat pain and irritation (8.5%), vomiting (7.8%) and port site bleeding (2.4%). Others accounting for 2.4% included; urinary retention, per vaginal spotting, diarrhoea, backache and dizziness.

Chart 10: Major post-operative complications



Major post-operative complications included; bowel injury (1.8%), respiratory complications (1.4%). ureteric injury (1.8%), wound sepsis (0.5%), bladder injury (1.8%) and mortality of 0.5%. The death was due to complications of bowel injury.

DISCUSSION

Laparoscopic surgery has become an independent surgical discipline in recent times. Surgical and gynecological procedures are now performed confidently and competently using the laparoscope. An increasing number of gynecological procedures are now being undertaken laparoscopically other than laparotomy (15). In Kenya laparoscopy is still in its early phases of evolution and private hospital still undertake the majority of cases. Kenyatta National Hospital is the only public hospital with equipment for laparoscopic surgery.

A total of 718 cases of gynecological surgery were done using the laparoscope between January 1998 to December 2004 at the Kenyatta National Hospital. The majority of the cases were diagnostic (66.1%) while the rest were operative (33 8%). In the Aga Khan hospital Nairobi in 2002, 52.% of the total gynecological surgeries were performed via the laparoscope while 47.3% was through open laparotomy(55). Only 15.5% of the laparsopic surgeries done at the Aga Khan hospital in the same year were diagnostic.

In our study the case load rate increased from 1.3 per month in 1998 to 5.3 per month in December 2004. In his series of operative laparoscopic gynecology done from 1996 to **2000,** Parker(2) and colleagues had an impressive increase in case load rate from 2.4 per month in 1996 to 27.5 per month in 1999. In a different study by Parker and colleagues(2) at the Aga Khan Hospital in Nairobi between May 2000 to May 2002 their case load rate increased from 7.0 cases per month in 2000 to 22 cases per month in 2001. This comparison clearly shows that the rate of utilization of the laparoscope at the Kenyatta National Hospital falls far below the expected rate compared to other hospitals. This should not be the case since KNH is the largest referral hospital in this region of Africa and also it is the teaching hospital for the University of Nairobi faculty of medicine

The age range of the study patients was from 18-59 years with a mean of 29±6 years. In our study 52% of the patients fell in the 20-29 age group. These were young patients compared to the ones in one of Parker's (2) study where 62.7% of the patients fell in

the30-45 age bracket and another study (18) where 50.5% were in the 31-40 years age bracket.

In our study most of the patients were nulliparous (46.5%). Para one were 24.9%, Para two 12.7% and more than para 4 6.8%. In both of Parkers' studies (2,18) patients who were para two comprised 49.2% and 38.6% and were the majority of the study subjects, respectively.

Unemployed patients and housewives (35.7%) with primary education (41.6%) were the majority of patients who underwent laparoscopic surgery. This could be due to the subsidized price the hospital offers to the patients since it is a public referral hospital. In Rono's study (55) most of the patients were employed in the formal sector (52.6%) while only 33% were unemployed.

The main indication for gynecologic laparoscopic surgery was secondary infertility **(34 4%)** followed closely by primary infertility (34.4%). Other main indications included; polycystic ovaries (6.4%), uterine fibroids (5.2%), ovarian cyst (4 8%), pelvic inflammatory disease (4.0%), chronic pelvic pain (3.6%), ectopic pregnancy (2.8%), dysfunctional uterine bleeding (2.4%), desired family size(1.2%) and endometriosis^.4%). Infertility was also the major indication for laparoscopic surgery at the Aga Khan Hospital, Nairobi (55).

Adhesioysis of pelvic adhesions causing compromised fertility and pelvic pain accounted for 45.3% of the total procedures done. Pelvic adhesions due to infection are a major cause of sub fertility in females in this region. Post-operative adhesions are also less in patients who have undergone laparoscopic surgery compared to Iaparotomy($p<0.0001$. Lundorf et al. 13).

In our study 83 (25.%) tuboplasty procedures were undertaken for both primary and secondary infertility. The main techniques used were cuff salpingostomy, linear and gutter salpingostomy, neoaslpingostomy and fimbrioplasty. The pregnancy rate after tubal surgery was not established in this study. In Gomel's series (56), 89 microsurgical salpingotomies were followed for more than 1 year; 30 patients (33.7%) achieved one or more pregnancy, 28 (31.5%) had one or more live births and 8 (9%) had ectopic

pregnancy. Dubuisson (57) followed 76 patients with microsurgical salpingotomies for more than 2 years; 28 patients(36.8%) achieved one or more pregnancies and 17 (22.3%) had ectopic pregnancy. In his series, Parker (18)had 234 (39.2%) women conceiving following laparoscopic adhesiolysis, tuboplasty , ovarian drilling and /or myomectomy.

Laparoscopic surgery is currently considered the gold standard in the management of ectopic pregnancy This is especially so for hemodynamically stable patients In our study 7(2 1%) cases of ectopic pregnancy were undertaken laparoscopically Salpingectomy was performed in 4 of the cases while salpigostomy for 3 of the cases. At the Aga Khan Hospital in Nairobi, 33 (8.08%) cases of ectopic pregnancy were managed laparoscopically between May 2000 to May 2002(2). In this series in 10 cases salpingostomy was done, 21 salpingectomy and 2 salpingotomy.

In another review Parker managed 28 (4.69%) cases of ectopic pregnancy laparoscopically out of a total of 596 laparoscopic surgeries performed in the coast province of Kenya(18). There is need to manage more cases of ectopic pregnancy by laparoscopic surgery in Kenyatta National Hospital. Brumstead et al (58) comparing laparoscopy versus laparotomy in the management of ectopic pregnancy reported significantly shorter periods of convalescence(8.7±7.8 days versus 25.7± 16.2 days p<0 01) and reduced post-operative analgesia requirements
(0 84±2.3 versus 4.64±2.9 doses; p<0.01) in the laparoscopy group compared to the laparotomy group. Yao and Tulandi (21) and Vermesh et al (8) reported similar findings.

The latter authors also reported higher rates of intrauterine pregnancies after laparoscopy (61% and 50% respectively).They also found lower rates of recurrent ectopic pregnancies in patients who had laparoscopic management as opposed to those who had laparotomy(8% vs. 14% and 6% vs. 11% respectively).

Endoscopic surgery is currently seen as a method of choice for the treatment of benign ovarian cysts. There is however some concern about the risk of cyst rupture in undiagnosed cases of malignancy. A survey by the American Association of Gynecologic Laparoscopist (1990) involving 13,739 cases of laparoscopic managed ovarian cysts

revealed only 53 (0.4%) cases proven to have had unsuspected ovarian cancer (16). The risk is therefore small.

In this study ovarian cystectomy was performed in 16 (4.8%) cases. The cysts ranged from 5cm to 12 cm in diameter. Several studies have documented the safety and feasibility of management of benign ovarian cysts via the laparoscope (59, 60).

Laparoscopic management of tubo-ovarian abscesses and pelvic abscesses is currently getting prominence since it was first described in France in the early 1970's (61). Laparoscopic treatment plus antibiotics is currently used to treat recent tubo-ovarian masses (less than 3 weeks old). Long duration tubo-ovarian abscesses (> 3 weeks old) can also be treated laparoscopically though incidence of complications is high In our review there were 4 (1.2%) cases of pelvic abscesses which were drained laparoscopically. This were recent abscesses, though one patient required blood transfusion due to intra-operative hemorrhage. Several studies have now documented the feasibility of laparoscopic management of pelvic abscesses (62,63).

Laparoscopy is currently considered to be the gold standard in the diagnosis and treatment of pelvic endometriosis. In our series only 3 (0.9%) cases of ablation of endometriotic foci were done. These was a low number compared with what is done in other centers worldwide. Under-or-mis-diagnosis of endometriosis may also be common in our set-up

There were 6 (1.8%)cases of myomectomy in our series. In his series in the coast province of Kenya Parker (18) performed a total of 59 (9.8%)cases of laparoscopic myomectomy due to uterine fibroids. Between May 2000 and May 2002, 63 cases of laparoscopic myomectomy were undertaken at the Aga Khan Hospital in Nairobi (2). Myomectomy is still performed by laparotomy in most of the cases at the Kenyatta National Hospital. Nezhat et al (26) observed that laparoscopic myomectomy is quite feasible especially if the patients are well selected, the surgeon has adequate experience, the equipment is of good quality and the surgical team is experienced. Jean Bernard Dubuisson (27) popularized and refined the technique of laparoscopic myomectomy by using a combination of monopolar and bipolar electrosurgery and intra-coporrheal sutures

to achieve hemostasis and tissue apposition. The use of ultrasonic (harmonic) scalpel by Miller in Chicago (28) has made laparoscopic myomectomy pretty bloodless.

In our review there were 11 (3.3%) cases of laparoscopic assisted vaginal hysterectomy (LAVH) and one case (0.3%) of total laparoscopic hysterectomy. LAVH was introduced to allow surgeons with limited experience in vaginal surgery to remove the uterus without and abdominal incision in the presence of pelvic adhesions, endometriosis, adnexal disease, or large-sized uterus. A total of 63 cases of LAVH were performed at the Aga Khan Hospital in Nairobi between May 2000 to May 2002 (2). These were more than in our series which was over a six year period. There were also 9 cases of total laparoscopic hysterectomy at the Aga Khan Hospital within the same period compared to only one case in our series

All patients scheduled for laparoscopy should consent for possible intra-operative conversion to laparotmy, since in any given unit as cited by Parker, 10 to 15% of all cases may be inadvertently converted to laparotomy (2). In our review, 16 (7.2%) cases were converted intra-operatively to laparotomy.

Intra-operative and post-operative complications in our series were slightly different from those reported by other authors in other parts of the world (36, 38). Bowel and vascular injuries were the most common complications each accounting for 3.6% of the total cases done. Bladder injury accounted for 2.7% and ureteric injury 1.8% of the total cases done. There was one case of death which was due to complications of bowel injury postoperatively. Mortality rate in laparoscopic surgery has been reported in one survey to be approximately 4 to 8 deaths per 100,000 cases (36). Bowel injuries have been reported to occur in between 0.1% to 4% of cases (39). These injuries are a major cause of morbidity and mortality as happened to our patients.

Our overall complication rate (8.1%) was slightly higher than those reported in literature (1-5%) (39). In our review complications were higher in patients who had undergone pervious surgery (14.3% vs.4.1% and this was significant (Fishers exact test = 0.007). Harkinen et al (11) cited reasons for increased complications in laparoscopic surgery as

rrevious abdominal surgery, obesity, gastric distension, active infection, use of electorcautery and cardiovascular disease.

Reduced blood loss has been cited as being amongst the major advantages of laparoscopic surgery The mean blood loss in our review was 267.46±143 02mls However out of the 221 files analyzed only 93(42.1%) had estimated blood loss recordings. Five patients in our study received blood transfusion. Four patients received 1 pint each while one patient received 2 pints. Possible reasons for not recording EBL included minimal blood loss (< 50mls) and difficulty in estimating blood loss due to constant suction and irrigation with saline during the operation.

The time taken in laparosopic procedures is generally long. The learning curve is also longer and it takes many cases and time for the surgeon to become competent. The mean operating time in our review was 115.11±46.56 minutes. A study in Finland (11) revealed the mean operating time for laparoscopic procedures was 85±14 minutes compared to **53rl3** minutes for laparotomies

The mean time for our review was slightly longer compared to reports in literature. Parker (2) and colleagues reported a mean operating time ranging from 50-310 minutes with an average of 95 minutes. In a different study Parker (18) reported a average operating time of 71-80 minutes. However the time recorded for his studies were from scope entry to scope removal. In our study the operating time included positioning of the patient, setting of laparoscopic equipment, induction of anaesthesia, actual operation up to anaesthesia reversal.

Post-operative mini-morbidities are quite common after laparoscopic surgery In our study abdominal pain was the most common post-operative mini-morbidity with 50.2% of patients complaining. Nausea accounted for 19.4%, shoulder pain 9.3%, throat pain and irritation 8 5%, vomiting 7.8% and port site bleeding 2.4%. Though most authors report less post-operative pain after laparoscopy the mini-morbidities are a concern both tor the physician and anaesthetist.

Wyekyer (64) in his study in Ghana found that the main causes of post-operative mini-morbidities were; abdominal pain, shoulder pain , nausea and vomiting. A report of Radcliff and colleagues in 1994 found post-operative abdominal and shoulder pain present in 45% of patients. Peritoneal insufflation, bowel manipulation and pelvic surgery all contribute to post-operative abdominal pain, nausea and vomiting.

In our study no patient was managed as a day case. This is not in keeping with the trends world-wide where quite a number of laparoscopic procedures are done as day care cases In Aga Khan Hospital (55) in Nairobi 37 (11.5%) patients were managed as day care and were discharged within 12 hours of surgery. The mean hospital stay in our study was 31 68±25 49 hours. The mean duration of stay in hospital appeared to increase with complexity of the procedure. The longest hospital stay was after total laparoscopic hysterectomy (84.00±0.00 hours) while the shortest was after sterilization (18.00±0.00 hours).

In a study comparing laparoscopy versus laparotomy and the mean length of hospital stay. (18) patients in the laparoscopy group stayed for a shorter time in hospital (2.1 ±0 3 days) compared to the laparotmy group (3.4±0.7 days) and this difference was significant ($p<0$ 001). Laparoscopic surgery is associated with smaller wounds and less tissue trauma hence recovery and discharge from hospital is early and resumption to normal activities takes a shorter time compared to laparotomy.

Over enthusiasm in early discharge can lead to premature discharge of patients from hospital before full recovery. Indeed Nkyekyer in Ghana (64) noted that 50% of patients who underwent laparoscopic surgery felt they were not well enough to be sent home at the time of discharge.

CONCLUSION

- Operative gynecologic laparoscopic surgery is yet to achieve its full potential at the Kenyatta National Hospital. The cases done over the six year period under review were very few compared to the local private hospital where twice as much of the cases were done over a two year period
- Adhesiolysis was the main intervention procedure done via the laparoscope The early laparoscopic procedure to be learnt by many surgeons is adhesiolysis since it is a prerequisite for many laparoscopic procedures. Uterine, tubal, ovarian and pelvic surgeries done via the laparoscope are small in numbers compared to other settings. Only one case of total laparoscopic hysterectomy was done over the six year period.
- Intra-operative and post-operative complications were slightly high compared to other settings both local and abroad.
- There was no day case performed yet in other hospitals both local and abroad day laparoscopic cases are the norm.
- Very few emergency gynaecolgical cases were undertaken laparoscopicallv
- Operation times were quite long but this is expected since very few cases are performed and most of the surgeons are in the learning phase for many procedures.

RECOMMENDATIONS.

- Minimal access surgery needs aggressive advocacy at the Kenyatta National Hospital.
- More surgeons need to be trained and re-trained on the modalities of minimal access surgery
- More emergency gynaecological cases need to be undertaken laparoscopicaliy.
- Collaboration with other institutions both local and abroad needs to be put in place so that many surgeons can be trained and also acquire the necessary experience on laparoscopic surgery
- Equipment maintenance needs to be aggressive and complete records of the procedures needs to be undertaken and files easily retrievable.

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APPENDIX I- WORLD HEALTH ORGANIZATION CLINICAL STAGING SYSTEM FOR HIV INFECTION

Stage **one / perfomance scale one** (asymptomatic normal activity)

1.Asymptomatic (ASY).
2 Persistent generalized lymphadenopathy(PGL).
3 Acute retroviral infection (seroconversion illness).

Stage two / performance scale two (symptoms but fully ambulatory).

4. Unintentional weight loss< 10% of body weight (WL4)
5. Minor mucocutaneous conditions (e g seborrhea, prurigo, fungal -nail, oral ulcers, angular cheilitis) (MMC).
6.Herpes Zoster within the last five years (HZV).
7. Recurrent upper respiratory tract infection (e g bacterial sinusitis) (URTI).

Stage three / performance scale three (bedridden < **50%** of normal day time)

8.Unintentional weight loss > **10 %** of body weight (WL **8).**
9. Chronic diarrhoea > one month. (DIA).
10. Prolonged fever > one month. (PRY)
11. Oral candidiasis (ORC)
12. Oral hairy leucoplakia (HLP)
13. Pulmonary TB in last year. (PTB).
14. Severe bacterial infections (pneumonia , pyomyositis) (BAC)
15. Vulvovaginal **Candida > 1** month / poor response to therapy (VVC).

Stage four / performance scale four (bedridden > 50% of day time in prior month)

16 HIV wasting (**8+9** or 10) (CAC).
17 Pneumocystis pneumonia (proven: PCP, presumptive: PPCP).
IS CNS toxoplasmosis (TOXO)
19. Cryptosporidiosis + diarrrhoea>one month (CRS).
20. Isosporiasis + diarrhoea (ISO).
21. Cryptococcus -non-pulmonary (CRC).
22. Cytomegalovirus infection other than liver, spleen or lymph node (CMV).
23. Herpes simplex infection; visceral or >1 month mucocutaneous (HSV).
24. Progressive multifocal leucoencephalopathy (PML).
25. Disseminated mycosis (MYC).
26. Candida esophageal / trachea / pulmonary (0EC)
27. Atypical mycobacteriosis disseminated (MOTT)
28 Non- Typhoidal salmonella septicaemia (SAL).
29 Extra- Pulmonary Tuberculosis (ETB).
30 Lymphoma (LYM).
31. Kapos's sarcoma. (KS).
32. HIV Encephalopathy (ADC).

APPENDIX II

CONSENT FORM

I do consent to participate in the research and to have my blood tested for HIV. I understand that this research is for educational purposes only and I do not stand to gain financially or otherwise from the research. I consent to have been adequately counseled on the condition of my health and the implications on myself and my baby. I do understand that it is my right to decline to participate in this study and this will not in any way affect my current or future treatment in this hospital.

SIGNATURE OF PARTICIPANT OR LEFT THUMB

PRINT

SIGNATURE OF WITNESS

DATE

Mimi ninakubali kujihusisha na utafiti huu nilioelezewa. Ninakubali damu yangu kupimwa virusi vya ukimwi Pia ninaelewa ya kwamba utafiti huu ni kwasababu ya masomo na sitapata pesa zozote au kitu kingine chochote kutokana na utafiti huu. Ninakubali nimeelezewa ugonjwa wangu vilivyo na madhara yake kwangu na mtoto wangu. Ninaelewa ya kwamba in haki yangu kukataa kujihusisha na utafiti huu na uamuzi wangu hautadhuru matibabu yangu ya sasa na ya baadaye katika hospitali hii.

SAHIHI YA MUHUSIKA AU ALAMA YA KDDOLE GUMBA YA KUSHOTO

SAHIHI YA SHAHIDI.....t.....

TAREHE

RESEARCH INFORMATION TO BE GIVEN TO THE PARTICIPANT.

This research forms part of my thesis for the masters degree in obstetrics and gynaecology The aim of the research is to find out what effect HIV disease has on pregnancy. HIV disease is affecting many pregnant women in our country and it is important to scientifically find out what these effects are especially in women with advanced HIV disease in pregnancy

A lot of research has been done in Europe and USA but our country still lags behind in doing research in this area yet we are the ones who are hardest hit by the disease.

The results of this research will enable us to plan our resources to fight the disease and in particular to strengthen our antenatal and delivery services to women with HIV disease in pregnancy.

You are therefore requested to voluntarily participate in this research in order to generate information in this area of study. This research will inflict no harm to yourself and to your baby. Your participation and the information you provide will be highly appreciated and treated with utmost confidentiality. By participating in this research you will have given crucial information to the scientists of this country on how to plan and help thousands of other women in our country who are pregnant and suffer from HIV disease. You also have a right to refuse to participate in this research and this will in no way affect your current or future treatment in this hospital.

Yours faithfully,

Dr. Joseph Wangira Musana,
Postgraduate student.
Department of Obstetrics and Gynecology,
University of Nairobi.

APPENDIX III

QUESTIONNAIRE I

Client Serial No. **DATE.**

SOCIODEMOGRAPHIC DATA

1 Age in years

2. Occupational status

   a. Not employed / Housewife

   b Self employed

   c. Formally employed

3. Marital status

   a. Single

   b Married

   c. Divorced / Separated

   d. Widowed

4. Level of education

   a. Primary

   b. Secondary

   c. College/University

   d. None

ANTENATAL DATA

5 Gestation at enrollment.

6 Number of previous deliveries

7. Number of previous abortions

8 HIV Seropositivity

a. Positive

b Negative.

9. WHO HIV Disease stage if positive

a. Stage 3

b. Stage 4

10 Reason for being stage 3 or 4.

a. Chronic diarrhoea for more than 1 month

b Prolonged fever for more than 1 month

c. Pulmonary Tuberculosis

d. Septicaemia

e. Severe bacterial pneumonia

f. Presumptive **Pneumocystis** carinii pneumonia

g. Bacterial meningitis

h. Cryptoccocal meningitis

i. Oropharyngeal candidiasis

j. Other. . . . . .Specify.

11 Is patient on HAART ?

a Yes

b. No.

12 Is patient on any PMTCT regime ?

a. Yes

b. No.

13. Haemoglobin level at booking.

14. VDRL Status.

15. Other sexually transmitted diseases noted at enrollment

a. Candidiasis

b Trichomoniasis

c Gonorrhoea

d Chlamydia

e. Non

QUESTIONNAIRE II

Client serial No. DATE.

LABOUR AND DELIVERY

16 Gestation at delivery in completed weeks.

17 Hypertensive disease. Diastolic blood pressure>90mmHg.

a. Yes.

b. No.

18 Labour started by

a. Spontaneous

b Induction

19 If induction state reason for induction.

20 Estimated blood loss in ml.

21 Mode of delivery

a Spontaneous

b. Breech delivery

c. Vaccum delivery

d Caesarean section

22 If caesarean section state reason for operation.

a. Due to PMTCT

b. Antepartum haemorrhage

c. Cephalopelvic disproportion

d. Fetal distress

e. Mai presentation

f. Other.....Specify

23 Other complications

a. Preterm rupture of membranes

b. Prolonged -preterm rupture of membranes

c. Prolonged rupture of membranes at term

d Prolonged labor

e Postpartum haemorrhage

f. Other.....Specify.

2J Presence of chorioamnionitis by foul smelling liquor at delivery [9]

a Yes

b. No.

25 Grade of liquor at delivery,

a. Clear

b Grade I MSL

c. Grade II MSL

d. Grade III MSL

POSTPARTUM PERIOD.

26 Placental weight at delivery in grammes.

27 Febrile illness present after delivery

a. Yes

b. No

28 If yes, state maximum temperature

29 Maternal hospital stay for more than a week after delivery [9]

a. Yes

b. No

30 If yes state reason for stay.

31 Maternal mortality present within the follow-up period ?

a. Yes

b No.

32 If yes state

a. Number of days after delivery.

b. Cause of death.

CHILD**'S QUANTITATIVE CHARACTERISTICS AT BIRTH.**

33 Fetal outcome.

a. Live birth

b Fresh still birth

c Macerated still birth

34 Weight of baby at birth in grammes.

35 Infant sex

a. Male

b. Female

36 Crown-rump length of baby at birth, (cm).

37 Head circumference (cm).

38 Apgar score

a. At 1 min.

b At 5 min.

c. At **10** min.

39 External congenital anomalies present [9]

a. Yes.

b. No.

40 If yes specify.

41 Status of Infant at discharge or at end of follow-

a. Alive and well

b Alive and unwell

c. Deceased

42 If unwell or deceased state reason

a. Respiratory distress syndrome

b Neonatal sepsis

c. Severe congenital malformations

d. Others.....Specify.

APPENDIX IV

QUESTIONNAIRE

OPERATIVE GYNAECOLOGIC LAPAROSCOPIC SURGERY AT THE KENYATTA

NATIONAL HOSPITAL.

A RETROSPECTIVE ANALYSIS

SOCIODEMOGRAPHIC DATA

Study No. **File No.** **Date.**

1. Age in years
2. Marital status
    1. Single
    2. Married
    3. Widowed
    4. Separated/ Divorced

3. Occupation
    1. Unemployed
    2. Self-employed
    3. Formally employed
    4. Other

4 Level of Education.

1. Primary

2. Secondary

3. College/ University

4. None

GYNAECOLOGICAL DATA

5. Number of previous deliveries

6. Number of previous abortions

PRE OPERATIVE DATA

7 Admission status of patient.

1. Day care

2. In-patient

8 Pre-operative diagnosis.

1. Desired family size

2. Primary infertility

3. Secondary infertility

4. Pelvic inflammatory disease

5. Ectopic pregnancy

6. Chronic pelvic pain

7. Dysfunctional uterine bleeding.

8. Uterine fibroid

9. Polycystic ovarian disease

10. Ovarian cyst

10. Abnormal cervical cytology |___1

12. Other..... (specify).

9. Has the patient had previous abdominal surgery

1. Yes

2. No.

10. If yes, state:

a. Indication for surgery

1. Gynaecological

2. Obstetrical

3. Surgical

4. Other..... (specify) CZ

b. Type of surgery done

1. Laparotomy EZ

2. Mini-Iaparotomy I

3. Caesarean section

4. Other..... (specify)

INTRA-OPERATIVE DATA

11. Operative procedure done.

1. Bilateral Tubal Ligation

2. Adhesiolysis

3. Tuboplasty

4. Removal of ectopic pregnancy |____|

5. Ovarian cystectomy

6. Ovarian drilling |____|

7. Myomectomy |____|

8 Laparoscopic Assisted Vaginal Hysterectomy____j

9. Total Laparoscopic Hysterectomy

10 Other.....(specify). |____|

12. Intraoperative complications.

1. Non recorded |____[

2. Vascular injury [____|

3. Bowel injury 1____1

4 Bladder injury 1____1

5. Other.....(specify)_

13 Estimated blood loss.

1. Recorded

2. Not recorded

14. If recorded state blood loss in ml.

15. Was the operation changed to laparotomy ?

1. Yes |____[

2. No. |____|

16. If yes, why was the laparoscopic procedure changed to laparotomy[9]

1. Failed first entry technique_

2. Intra-operative complications

3. Inability of the laparoscopic procedure to adequately treat the condition I I

4. Anaesthetic complications

5. Other.....(specify). I I

17 Range of intra-operative cardiovascular parameters.

1. Blood pressure in mmHg.

i. Systolic_

ii. Diastolic_

2. Pulse rate per minute_

18 How long was the operation [9] (minutes)

POST-OPERATIVE DATA.

19. Length of time in recovery room

1. Recorded

2. Not recorded

20 If recorded state time in minutes.

21. Where was the patient taken from recovery room [9]

1. Discharged home via ward Q

2. Back to ward [_

3. ICU/HDU

4 Other. . . . . (specify).

22. Did the patient require blood transfusion ?

1. Yes I—

2. No I—

23. What was the post-operative mini-morbidity?

1. Non recorded

2. Nausea |

3. Vomiting |

4. Abdominal pain |

5. Bleeding from surgical site

6. Shoulder pain |

7. Other. . . . . (specify) I

24. Were there any major post-operative complications ?

1. Yes. I H

2. No. —

25. If yes, what were they ?

1. Vascular injury f—

2. Bowel injury

3. Bladder injury

4. Respiratory complications [

5. Cardiovascular complications

6. Other. . . . . (specify) [

26 How was the patient managed for the complications ?

1. Conservatively
2. Laparoscopic surgery
3. Laparotomy |____|
4. Other.....(specify) |____|

27. Total length of hospital stay in hours. |____|

28 Was the patient re-admitted after discharge

1. Yes |
2. No |____1

29 If yes state:

a. Duration after discharge in days,

b Reason for readmission

I Due to laparoscopic related complications

2. For further operation for the diagnosed condition

3. For non-laparoscopic related condition

4. Other.....(specify).

APPENDIX V

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along. Ngong Rd
P.O. Box 20723, Nairobi.

Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi

CSCG-

Ref: KNH-ERC/01/2407 **Date: 22nd October 2004**

Dr Joseph W Musana
Deptof Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr. Musuna.

RE "OPERATIVE GYNAECOLOGIC LAPAROSCOPIC SURGERY KENYATI A NATIONAL HOSPITAL A RETROSPF.CTI VE AN ALYSIS" (P99/7/2004)

This is to inform you that the Kenyatla National Hospital Ethics and Research Committee lias reviewed and apmoved the revised version of your above cited research proposal for the period 22nd October 2004 - 21 st October 2005 You will lie required to request for a renewal of the appro\al if you intend to continue with the study beyond the deadline given

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summarv of the research findings upon completion of the study

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances ofstudy duplication

Vours sincerely,

PROF A N GUANTAI
&XRLIAKY. KNII-I R(

Cc Prof. K Bliatt, Chairperson, KNII-ERC
The Deputy Director (C/S), KNH
The Dean, Faculty of Medicine. UON
The Chairman, Dept. of Obs/Gynae, UON
CMRO
Supervisors: Dr. Weston Khisa, Dept.of Obs/Gynae, UON
Prof. S.B. Ojwang, DeptofJ#*/Gynae, UON

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.

Tel: 726300-9
Fax: 725272
Telegrams: "MEDSUP", Nairobi.
Email: **KNHplan@Ken.Healthnel.org**

Ref: KNH-ERC/01/2351

Date: 10'h September 2004

Dr. Joseph W. Musana
Dept. of Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr. Musana,

RESEARCH PROPOSAL "PREGNANCY OUTCOMES IN MOTHERS WITH ADVANCED HUMAN IMMUNODEFICIENCY VIRUS DISEASE^ (r59/5/2004)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and approved revised version of your above cited research proposal for the period 10 September 2004 - 9 September 2005. You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely /

PROF. A'N GUANTAI
SECRETARY, KNI1-ERC

cc Prof K M Bhatt, Chairperson, KNH-ERC
The Deputy Director (C/S), KNH
The Dean, Faculty of Medicine, UON
The Chairman, Dept. of Obs/Gynae, UON
CMRO
Supervisors: Prof. SBO Ojwang, Dept.of Obs/Gynae, UON
Dr. Weston Khisa, Dept. of Obs, Gynae, UON