CASE RECORDS AND COMMENTARIES IN

OBSTETRICS AND GYNAECOLOGY

SUBMITTED BY

DR SHIPHRAH KURIA

FOR THE DEGREE OF MASTERS IN MEDICINE

IN

OBSTETRICS AND GYNAECOLOLOGY

UNIVERSITY OF NAIROBI

2005

CONTENTS

Gynaecology Short Cases

Gynaecology Long Commentary:

DEDICATION

This book is dedicated to my parents Elizabeth and Hezron, who together with others brought me up in the way of the Lord and gave sacrificially to make me what I am today.

ACKNOWLEDGEMENT

I am grateful to the university of Nairobi for offering me the opportunity to train in Obstetrics and Gynaecology and to the Ministry of Health for the sponsorship. Thanks to the administration of the Aga Khan Hospital Nairobi for the opportunity to do elective term there.

For the wise critique on the commentaries and short cases by my supervisors: Dr E Bukusi, Dr Gachuno and Dr Tekle I am truly grateful. It would not have been possible to do this work without their guidance and dedication.

Special thanks to the members of the Obstetric and Gynaecology department including the chairman Dr Oyieke, all the consultants, my colleagues, other staff and the patients for all their support during the program without which it would have been impossible to learn.

I wish to thank the administration of Pumwani Maternity Hospital for allowing me to carry out the research on the quality of ANC in that hospital and for the nurses there who were very helpful. Special thanks to Dr Musili who was my supervisor in Pumwani.

I thank the KNH administration for allowing me to carry out the study on Domestic Violence in the hospital.

To the friends and family members who encouraged me and prayed with me I say thanks. Very special thanks to my husband Simon Ndiritu for his love, support and patience through the demanding program. To Susan who typed most of this material, Dick and Frankie who helped in Data analysis, I am grateful.

Finally and most important, to the almighty God from whom all good things come, for His strength and grace I am forever grateful; thus far has He brought me.

DECLARATION

This is to certify that the commentaries and the case records in this book are my original work. The case records recorded were managed under the supervision the senior members of the Obstetric and Gynaecology department, Kenyatta National Hospital.

DR SHIPHRAH KURIA

MBCHB

UNIVERSITY OF NAIROBI

SIGNATURE .....

DATE .....10.10.05.....

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria researched upon the long commentaries in this book under my guidance and supervision and that this book is submitted with my approval.

SIGNATURE .....

DATE ...SEPTEMBER...1...2005...

DR ELIZABETH BUKUSI,
MBCHB, MMED, MPH, Ph D (C)
RESEARCH OFFICER, KEMRI AND HONARARY LECTURER
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria researched upon the long commentaries in this book under my guidance and supervision and that this book is submitted with my approval.

SIGNATURE .....

DATE ..... 16/10/05 .....

DR GACHUNO ONESIMUS,
MBCHB, MMED (O/G)
LECTURER
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria managed Obstetric cases number 3,8,11,12,13,14 and Gynaecology cases number 3,4,5,13,15 under my guidance and supervision at Kenyatta National .

SIGNATURE .....

DATE 25.09.05 .

DR J.B.O OYIEKE
MBCHB, MMED (O/G)
SENIOR LECTURER AND CHAIRMAN
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria managed Obstetric cases number 2,4,5,6,10 and Gynaecology cases number 6,9,10,11, 14 under my guidance and supervision at Kenyatta National Hospital and Aga Khan Hospital.

SIGNATURE .....

DATE ..... 1/9/2005.

PROF. S.B.O OJWANG
MBCHB, MMED (O/G) DIP
PROFESSOR
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria managed Obstetric cases number 2,7,9,15 and Gynaecology cases number 1,2,7,8,12 under my guidance and supervision at Kenyatta National.

SIGNATURE .....

DATE 02/09/2005

PROFESSOR JOSEPH KARANJA
MBCHB, MMED (O/G)
ASSOCIATE PROFESSOR
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria managed Obstetric cases number 3,8,11,12,13,14 and Gynaecology cases number 3,4,5,13,15under my guidance and supervision at Kenyatta National.

SIGNATURE .....

DATE .....2/9/05.....

DR MUIA NDAVI
MBCHB, MMED MSC EPI,FHBR,DLSHTM.
SENIOR LECTURER
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

CERTIFICATION OF SUPERVISION

This is to certify that Dr Shiphrah N Kuria managed Obstetric cases number 2,7,9,15 and Gynaecology cases number 1,2,7,8,12 under my guidance and supervision at Kenyatta National .

SIGNATURE .....

DATE 7/9/05

DR SAMSON WANJALA
MBCHB, MMED (O/G)
SENIOR LECTURER
DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY
UNVERSITY OF NAIROBI.

INTRODUCTION

The obstetric and gynaecology short cases presented were managed at Kenyatta National Hospital (KNH) and in Aga Khan Hospital Nairobi. The Obstetric long commentary was researched at Pumwani maternity Hospital and the Gynaecology long commentary was researched at Kenyatta National Hospital.

The Kenyatta National Hospital is situated about 3 Km from the Nairobi city centre along Ngong road. This is the national referral hospital and also serves as a teaching hospital for the college of health sciences of the university of Nairobi offering facilities for undergraduate and postgraduate medical courses. Nursing and paramedical courses are also offered by this hospital.

Obstetric And Gynaecological Services

The obstetric and gynaecology outpatient services are provided at the antenatal and gynaecology clinic, casualty department and family welfare clinic. The inpatient services are provided in labour ward, Antenatal and Postnatal wards, the acute gynaecology ward and the elective gynaecology ward.

The Department of Obstetrics and Gynaecology offers laboratory services including semen analysis, hormonal radioimmunoassay, cytology, spectrophotometry, surfactant test and glucose tolerance test. Ultrasound foetal monitoring and radiological examinations 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 and all obstetric and gynaecological emergencies are screened here. Most patients are treated and discharged while serious cases are admitted either to labour ward or acute gynaecological ward; senior house officers offer obstetric and gynaecology coverage 24 hours a day.

Antenatal care

This offers routine antenatal care and also acts as the referral centre for high-risk patients from other clinics. The booking is done every Monday morning.

For those that are booked, a detailed history of the patient's past obstetrical and gynaecological, medical and social history is taken. The patients are then sent to the laboratory for antenatal profiles which include; blood group (including the Rhesus factor), serologic test for syphilis (VDRL), full haemogram (especially haemoglobin level), Urinalysis (protein/sugar) and Voluntary Counselling and testing for HIV. For first pregnancies or previous pregnancies more than 3 – year intervals, two tetanus toxoid doses are given 4 weeks apart; otherwise only booster is given during the second trimester. Protenuria, glycosuria, blood pressure, and weight gain are checked on every clinic visit.

During the antenatal period, any antenatal morbidity is managed accordingly either as outpatient or inpatient. At 36 weeks, clinical pelvic assessment is done on the primigravida. Those with breech presentation destined to deliver vaginally may also be done erect lateral pelvimetry. Amniocentesis for surfactant test is done at 38 weeks in those mothers who are planned for elective delivery. Also during this period of antenatal care, the appropriate health education on pregnancy and its related problems is provided. Emphasis is laid on better nutrition, clinic attendance, preparation for labour and delivery, post partum care, breast-feeding and family planning.

Voluntary counselling and testing for HIV

This is offered to all pregnant mothers; those who are negative are encouraged to continue being careful to avoid infection. Those who are positive are told about the various available methods of preventing mother to child transmission of HIV. They are put on zidovudine from 28 weeks gestation and get and Niverapine 200mg at the onset of labour. The infants are given Niverapine syrup stat dose after delivery and zidovudine syrup for one week. They are also encouraged not to breast feed. Those whose CD4 count is below 200 are started on the highly active anti retroviral therapy (HAART).

Hospital Admissions

Admissions fall into three categories namely; booked patients from our antenatal clinic, referrals from other hospitals or health centres and those without prior antenatal care. The last two categories constitute the majority of admissions. Booked

patients report directly to labour ward admission area when they are in labour or if they develop a problem when the clinics are closed e.g. weekends. Un booked patients are seen first in casualty before being sent to labour ward admission area. An intern doctor in conjunction with a senior house officer (registrar) sees the patients. Other members of staff are called if need arises. Those in labour are admitted to the labour ward while those not in labour are admitted to the ante natal ward if so required or discharged home. Patients who are very ill are admitted to the acute room in labour ward and managed accordingly.

Management Of Labour

Active management of labour is advocated. The components of active management of labour include; strict diagnostic criteria for labour, amniotomy at 4cm dilatation, early use of oxytoxin and continuous professional support. These measures are known to reduce the rates of caesarean sections and operative vaginal deliveries as well as prolonged labour and its attendant complications. In our set up due to the high prevalence of HIV infection routine amniotomy is discouraged especially when the HIV status is not established, as a measure to reduce vertical transmission of HIV.

The first stage of labour

Those patients who come from the lying in wards for induction of labour are given a soap enema and a warm bath. Patients who come from home in labour are assessed if they are in early labour with intact membranes soap – enema is given. Progress of labour is recorded graphically on a partogram where uterine contractions, foetal heart rate and maternal pulse rate are recorded every half hour; blood pressure and temperature every hour; and abdominal and vaginal examination every four hours. In vaginal examination the cervical dilation in centimetres is noted and recorded. In addition, descent of the presenting part and degree of moulding and the colour of the draining liquor is also noted and recorded. Urine analysis by dipstick is performed each time the patient passes urine to assess for protenuria and glycosuria mainly. An intramuscular injection of pethidine is given routinely for analgesia in the early phase of labour. Other alternatives include use of tramadol and buscopan.

The partogram has proved to be an indispensable tool in monitoring the progress of labour and predicting complications of labour to enable timely intervention. Descent

of the head is determined by the fifths of the palpable head above the pelvic brim. Cervical dilation of at least 1 cm per hour is expected and short of this rate in absence of any contraindication labour is augmented with oxytocin.

Speculum examination

Speculum examination is performed in patients complaining of haemorrhage or premature rupture of membranes. The patient is placed in lithotomy position, the vulva is cleaned with chlorohexidine solution and draped with sterile towels. A cusco's speculum is gently introduced in the vagina and the vagina slowly opened. Using a light source, lesions of the vaginal wall and cervix are sought. Bleeding or drainage of liquor through the cervical opening is noted.

The second stage of labour

When the patient is confirmed to be in second stage by both vaginal and abdominal examination and also has the urge to bear down, she is transferred to the delivery room and placed on a delivery bed. A midwife, a student midwife or a medical student under instruction usually conducts normal deliveries. The registrar conducts the high-risk cases (like multiple pregnancies, breech presentations, premature deliveries) and all operative vaginal deliveries. Clean delivery areas and strict aseptic technique is adhered to during each delivery. The person conducting the delivery is gowned and masked. The perineum is cleaned with chlorhexidine solution and sterile towels applied. The patient is encouraged to bear down with each contraction and to take deep breaths between contractions. Foetal heart is monitored every five minutes.

If the perineum is tight it is infiltrated with 10mls of 1% ligocaine hydrochloride and mediolateral episiotomy is performed when the head is about to crown. The person conducting the delivery inserts the index finger of the left hand into the vagina to protect the foetal head. Using a blunt – tipped Mayo's scissors and incision is made in the perineum starting in the midline and directed laterally and downwards.

When the foetal head distends the perineum the latter is supported by the right hand with a sterile pad while the left hand keeps the head flexed and prevents sudden expulsion. This prevents trauma to the perineum and foetal head in preterm babies. Once 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 passed around the neck to

rule out presence of the cord. If a cord is found and is loose it is slipped over the head. If it is tight double clamped and divided. The anterior shoulder is delivered followed by the posterior shoulder, trunk, and legs. If the umbilical cord was not clamped, it is done so now and the baby shown to the mother before handing over to another midwife who carries out oropharyngeal suction as required. In high – risk cases, a paediatrician is usually in attendance.

The Third stage of labour

At delivery of the anterior shoulder, 0.5mg ergometrine is given intramuscularly to effect contraction of the uterus. For patients with history of post – partum haemorrhage and grand multiparity it is given intravenously for a more rapid action. For cardiac and hypertensive patients, oxytocin 5 units' intravenous infusion is given if uterine contractions do not occur spontaneously.

The placenta and membranes are delivered by controlled cord traction after signs of separation (rise in uterine fundus, lengthening of umbilical cord and gush of blood) have occurred. The birth canal is inspected for any tears and the episiotomy is repaired. The patient is encouraged to empty the bladder. Post delivery blood pressure, pulse rate, uterine contraction, and lochia loss are observed and clearly recorded. The patient is further observed for one hour (4^{th} stage) and then transferred to the lying in ward for subsequent observations, "Rooming in" is encouraged and early initiation of breastfeeding within 30 min is advocated as long as there is no contraindication. The mothers are nursed together with their babies to establish good lactation and bonding. Patients with normal delivery are discharged once they are stable and their babies well usually within twenty hours due to pressure of bed space. The patient is advised on perineal hygiene and frequent sit baths until the episiotomy heals. The patients are also advised on neonatal and infant care and breastfeeding as well as the danger signs in the infant and themselves.

Repair Of Episiotomy

This is carried out in three layers using no 2/0 catgut stitch. The apex of the incision is identified and the repair of the vaginal mucosa carried out in a continuous suture while the muscle layer is approximated with interrupted sutures. The skin is apposed

using interrupted or continuous catgut no2/0 burying the knots and starting from the lateral edge.

Operative Vaginal Delivery (Vacuum Extraction)

The vacuum extractor is exclusively used to accomplish delivery in prolonged second stage due to poor maternal effort or where bearing down iscontraindicated as in cardiac and hypertensive disease or where expedite delivery is desired as in foetal distress occurring in the second stage of labour.

The patient is placed in lithotomy position. The vulva and perineum are cleaned with antiseptic solution and draped. Aseptic catheterisation of the bladder is done and repeat vaginal examination performed to rule out any contraindication to vacuum delivery such as cephlo-pelvic disproportion and malpresentation. The foetal head should be in the pelvis with only one fifth being palpable above the pelvic brim. An episiotomy is given during a contraction. The largest suitable vacuum cap is passed against the foetal scalp taking care not to include maternal soft tissues by running a finger round the cap. A negative pressure of 0.8 Kg/cm^2 is induced stepwise at intervals of 0.2 Kg/cm^2 every two minutes. At each increase in pressure a check is repeated for any maternal tissue around perimeter of the cap. During this process an artificial caput is created. When a caput is already present, the negative pressure may be achieved faster.

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 foetal head the pressure is released. The mouth and nose are wiped and delivery continued as for spontaneous delivery. The baby is handed over to the paediatrician for resuscitative measures as necessary.

Caesarean section

The commonest abdominal delivery done is the lower uterine segment caesarean section. Classical caesarean section is rarely done except for case of transverse lie with ruptured membranes.

Pre operative care

For elective caesarean section the patient is starved for at least six hours before operation. Blood is taken for grouping and cross matching and two units of blood are reserved. Informed consent for general anaesthesia and operation is taken. The abdominal wall, vulval and perineum are shaved clean. Premedication with atropine 0.6 mg is given intramuscularly half hour before going to theatre.

Surgical procedure

In theatre the patient is placed in supine position with the legs separated, the vulva and perineum are cleaned with chlorhexidine solution. Catheterisation is done and the catheter is left in situ after draining all the urine.

The anterior abdominal wall is cleaned with antiseptic lotion and iodine or spirit, then draped, general anaesthesia induced with intravenous thiopental sodium at a dosage, which is effective in sedating the patient, but it varies between 250-500 mg, succinyl choline 50 – 80 mg is also given intravenously for temporary muscle relaxation to enable endotracheal incubation. Anaesthesia is then maintained with nitrous oxide, oxygen and halothane. The abdomen is then opened in layers through a sub-umbilical midline incision, which extends an inch below the umbilicus, and above the pubic hairline or a transverse incision (commonly pfannenstiel). 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 muscle separated from their attachment to it, using a surgical blade, and then drawn to one side to expose the peritoneum. The latter is held up with 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 Doen's retractor is applied to reflect the bladder away as well as expose the uterovesical fold of peritoneum.

The peritoneal fold is picked with a non-toothed dissecting forceps and opened at the middle using a curved Mayo's scissors. The incision is then extended on either side and the peritoneum stripped off the lower uterine segment with mounted swab. The Doen'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 2 cm is made in the lower segment about 2 cm below the uterine attachment of the uterovesical peritoneal fold. Once the membranes are reached the incision is extended laterally on either side using curved scissors directed by two fingers of the left hand. The opening is in an upward directed semi lunar incision to avoid uterine arteries at the angles. The incision is enlarged enough to allow delivery of the head and trunk. The membranes are then ruptured.

If the placenta is encountered in the line of incision it is either deflected or incised but in the latter case severe foetal maternal haemorrhage may occur and therefore the cord has to be clamped quickly. The retractor is then removed. If the presentation is vertex, a hand is slipped into the uterus between the foetal head and symphysis pubis and lifted gently with fingers and palm through the incision while a modest transabdominal pressure is applied. After delivery of the head, the nostrils and mouth are sucked. The shoulders are then delivered using gentle traction and still with some fundal pressures. The trunk delivery follows readily.

Intravenous ergometrine 0.5mg is given as the shoulders are delivered. After the infant is born the cord is clamped and divided then the baby is handed over to an assistant for resuscitation. In case of need, a paediatrician is on standby.

The placenta is delivered manually unless it separates spontaneously. The cut edges of the uterus are held with Green Armitage uterine clamps to control any bleeding that might be occurring as the inside of uterus is wiped of blood and other placental tissue such as membranes. The placenta is also inspected for completeness. The uterus is lifted out of the abdominal cavity through the incision and covered with a wet abdominal pack.

The uterus is then closed in 2 layers with No 2 chromic catgut as a continuous stitch for both layers. The second layer is stitched such that it buries the first one and extended beyond the lateral edges of the stitch. The visceral peritoneum is then closed with no. 1 chromic catgut.

The abdomen is mopped and the abdominal packs are removed. The pelvic viscera are then inspected for any abnormalities. Instruments and swabs are counted and if they tally with the initial count then the abdomen is closed in 3 layers. Peritoneum is closed with continuous No1 chromic catgut stitch, rectus sheath is similarly closed with No. 2 chromic catgut and skin with interrupted silk or nylon. The wound is cleaned and then dressed. The catheter is removed and uterus is massaged and clots evacuated from the vagina. General anaesthesia is reversed with 1.2mg of atropine and 2.5 mg of neostigmine intravenously. Extubation is done and oropharyngeal suctioning done.

Blood loss is estimated from what is in the suction pump container and amount in wet swab and mops. The patient is then transferred from the theatre to labour ward.

Post caesarean section care

The patient is observed quarter-hourly for one hour, then half-hourly for 2 hours, then 4 hourly thereafter, noting the blood pressure, temperature, pulse rate and respiratory rate on a chart, until she is fully awake, then four hourly, intramuscular pethidine 50 – 100mg 6 hourly is given for 48 hours to relieve pain. Intravenous 5% dextrose and normal saline are given alternately as 500mls four hourly untill bowel sounds are re-established. Prophylactic antibiotics are given to those at high risk of getting sepsis.

On the third post-operative day often haemoglobin level is checked. The stitches are removed after seven days of operation, after which the patient is discharged home with a case summary and having been explained to about the nature and findings of operation. The mother is seen in the post-natal clinic after one week and the baby is also seen in the child welfare clinic.

Post Natal Follow Up

The clinic is held every Friday morning. 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 immunisation of the baby. The patient is then examined and any problem managed. Mothers who are HIV positive have a special clinic where the necessary follow up is done. Family planning advice is given and the patient referred to the family planning clinic for the various methods available.

Care Of The New Born

All the newborn babies who are normal join their mothers after delivery unless the mother is moribund. A paediatric registrar reviews all the babies with problems or where complications are anticipated together with babies delivered by operative vaginal delivery or by caesarean section. Those having problems or who may develop some problems are transferred to nursery in a warm incubator. The premature babies are managed in nursery until their weight is about 2,000 gm when they are discharged. All mothers with babies in nursery are lodged in a mother's hostel.

The Gynaecology Unit

This consists of the out patient wing at clinic No 18 and two gynaecological wards; 1B and 1D on the first floor of the tower block. Ward 1D is the acute gynaecology ward whereas ward 1B is the elective gynaecology ward. The three firms in the department run the unit.

The Gynaecology Outpatient Services

These are mostly conducted in the clinics, which are three per week: firm 1 on Tuesday, Firm III on Wednesday and Firm II on Thursday. The clinics are run by consultants, senior registrars and registrars. Medical students are usually in attendance. There is also oncology clinic, which is on Friday mornings for following up of patients discharged from the ward. A colposcopy clinic is held every Friday morning. The majority of patients attending the gynaecology clinic are referred from casualty and emergency gynaecology ward after emergency consultation and treatment.

Postoperative patients also attend this clinic. Some patients are referred from other specialist clinics in Kenyatta National Hospital, other hospitals in and around Nairobi and from district and provincial hospitals.

Infertility cases constitute about two thirds of the gynaecology consultation followed by uterine fibroids, abnormal uterine bleeding, and adnexal masses. In the clinic, history is taken, a thorough physical examination is conducted, and most of the investigations are done as outpatient in the clinic to eventually reduce the hospital stay. These include haemogram, urea, and electrolytes semen analysis, Pap smear, pregnancy test among others.

Family Planning Clinic

It is situated at the family welfare centre. The methods of Family Planning offered include pills, the implants, injectables and voluntary surgical contraception procedures.

Gynaecology In-Patient Services

Elective gynaecology admissions – ward IB

This is the elective ward to which patients are usually admitted from the clinic or are transferred from the acute gynaecology ward for further management. The ward has 36 beds. Commonly the patients admitted here have uterine fibroids, gynaecological malignancies, and infertility among others.

Acute Gynaecological Admission – Ward ID

This is the emergency gynaecological ward having 32 beds but at the time of writing this introduction it averages 60 patients. Averages of about 10 patients are admitted daily, and about two thirds of these are cases having abortion complications. They are admitted through the gynaecology casualty, which is located in ward ID.

All the patients are clerked by the houseman and reviewed by the registrar who undertakes the management in consultation with senior members of the department. Apart from incomplete abortion, 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 ward ID, using Karman's Cannula and syringe. They are discharged home immediately. Patients who have undergone emergency laporotomies for pelvic abscess, ectopic pregnancy or pelvic masses have a minimum stay of four days post operatively. All patients with incomplete abortion and have uterine evacuation are counselled about contraception before discharge.

Patients with suspected carcinoma of the cervix are admitted at the first instance to ward ID, where they receive emergency care i.e. blood transfusion, antibiotics etc. routine clerking and investigations are started. Examination under anaesthesia, staging and biopsy is done. When histology report becomes available they are either transferred to ward IB or radiotherapy unit for definitive management. The patients also receive continuous care from the patient support centre and the hospice.

Gynaecological Operations

A theatre is reserved in main theatre for emergency gynaecological operations daily. Laparotomies for ectopic pregnancies (ruptured and non ruptured), pelvic abscesses, ovarian cyst and other tubo-ovarian masses are done here. Smaller procedures like diagnostic dilation and curettage of the uterus, removal of misplaced contraceptive devices and suction curettage are also performed.

Elective operations are done on firm basis, firm II on Mondays and Firms III and I on Thursdays. The operations are done from 8.00 am to 5.00 pm. 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 such as Vesicovaginal fistulae repairs are carried out under spinal anaesthesia.

Postoperative Care

After the operation general anaesthesia is reversed and the patient wheeled to the recovery room where quarter hourly observation of blood pressure, pulse rate,

respiratory rate and temperature are taken for half an hour, then half hourly until she is fully awake. The patient is then transferred to the ward where observations are done four hourly.

Most laparotomy patients are kept in the ward for four 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. Prophylatic antibiotics are given routinely. A check haemoglobin level is determined on the third postoperative day.

Before discharge the patient is informed about the findings at operation and a discharge summary is issued. Patients are reviewed in the gynaecological clinic after six weeks or earlier when there is an indication. Total abdominal hysterectomy is the commonest cold gynaecological operation. It is described below.

Total abdominal hysterectomy

General anaesthesia induction and maintenance is done as described above. A vulvo – vaginal toilet is performed with cetavlon lotion. Under aseptic conditions the patient is catheterised and the catheter left insitu to maintain continuous bladder drainage during the operation. Pelvic examination under anaesthesia is performed and pathological and normal findings noted. The vagina is painted with methylene blue dye. The abdomen is thoroughly cleaned with chlorohexidine and painted with iodine and then draped with sterile towels.

As described under caesarean section, the abdomen is opened in layers. The round ligaments are identified and beginning on either side using straight long artery forceps the round ligament is clamped and divided between the two forceps. The lateral stump is transfixed with no. 0 or no. 1 chromic catgut. This procedure opens the anterior leaf of the broad ligament, which is pushed forwards through this opening with the surgeon's finger and incised with scissors. The same is done for the opposite side. The next step depends on whether the tube and the ovary are to be preserved or removed. If they are to be preserved 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 vessels. This stump is ligated using

transfixed chromic catgut no 1. The same is done for the opposite side. If the tube and the ovary are to be removed with the uterus the in fundibulo 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 broad ligament together with the ovarian vessels is divided between the clamps and ligated using chromic catgut no1. 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 dissecting of the fascial fibres beneath the bladder wall. Usually the bladder can be displaced into the lower pelvis easily, but if it is adherent, it is surgically released.

In the next step, 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. These are double clamped and cut using a scalpel and freed form 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 tissues beyond the tip of the clamp, as this could permit bleeding from the collateral vessels that are not always included in the clamp, before clamping and cutting the uterine vessels it is always advisable to palpate 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 chromic catgut no 2.

The uterus is retracted forward and upward to demonstrate and stretch the uterosacral ligaments posteriorly. 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, mobilising it past the cervix to the posterior vaginal fornix. Usually this procedure is associated with haemorrhage as a proper loose areolar plane is entered. Care is taken not to dissect extensively laterally where the haemorrhoidal vessels are inserted into the rectum. Each uterosacral ligament is double clamped, cut, and ligated with no 1 chromic catgut sutures. 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 open technique, in which the anterior vaginal fornix is opened initially with the scalpel and a sharp knife or scissors circumcises the vagina. As the anterior, posterior and lateral angles of the vagina are opened straight artery forceps are used to secure the vaginal margins. These margins are then closed using a series of figure eight futures. Particular care is taken when tying the lateral angles to ensure that the descending vagina branches of the uterine vessel are securely ligated.

Suspension of the vaginal vault is done by tying the peritonealisation suture to the lateral and mid sutures for the vault. Peritonealisation is accomplished by means of a continuous no1 chromic catgut suture that first pierces the vaginal walls near the midline and passes through the posterior leaf of the broad ligament, the free margin of round 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 loosened separately with a purse string of no 2/0 chromic catgut and the free margin of the pedicle is high against the pelvic wall and are not anchored to the vaginal vault. This is advised in order to avoid subsequent dyspareunia and to avoid stretching of the ovarian vessels with possible thrombosis and chemical and cystic changes of the ovary. After this, abdominal viscera are well inspected. If haemostasis has been achieved and instruments and swabs counts are correct, the abdomen is closed in anatomical layer. The post-operative management is the same as described earlier.

Counselling clinics

There are several such clinics 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 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 and even those who do not want to bring up their children. The counsellors include trained nurses, sociologists and consultant obstetrician / gynaecologists. They counsel their patients, 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 Mothers Hostel

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

OBSTETRIC CASE 1

DEEP VENUOS THROMBOSIS – LIVE BABY

Ip No.	0987240	D.O.A.	14. 04. 02
Name	L K	D.O.D	19. 04. 02
Age	40	Parity	2 + 0

Presenting complaints

The patient was admitted with complaints of swelling and pain in the left lower limb for one week.

History Presenting Illness

She had been well until one week prior to admission when she noted some swelling in the left lower limb, associated with pain. The swelling and the pain increased progressively until walking became difficult. There was no associated hotness of body. There was no history of trauma.

History of current Pregnancy

Her last monthly period was on 15.9.02and the expected date of delivery was 22.3.03. She was at the gestation of 30 weeks. She attended clinic in Kenyatta National Hospital from 16 weeks gestation. The antenatal profile was as follows.

HB – 2.1 g/dl	VDRL – negative
Blood group – A +ve	Elisa for HIV – negative

Obstetric and Gynaecological history

She was a para 2 + 0. Her last delivery was in 1999 while her first delivery was in 1996.The previous deliveries were spontaneous vertex deliveries and the babies were alive and well. She attained menarche at 14 years. Her cycle was regular every 28 – 30 days with a normal flow of 3 – 4 days. She had used oral pills for contraception for two years prior to the pregnancy.

Past medical history

This was not significant

Family and social history

She worked in the hospital as a patient attendant. Her husband was a teacher. She did not take alcohol or smoke cigarettes. She had no history of chronic illness in her family.

Examination

She was in good general condition, not pale or jaundiced. She had no lymphadenopathy or oedema. Her vital signs were: BP – 100 / 65 mmHg, PR – 84/min PR 22/min and temp 36.8oC

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

Abdominal examination

The abdomen was uniformly distended with fundal height at term gestation. The foetus was in longitudinal lie, cephalic presentation and the head was 4/5 above the pelvic brim. There were no contractions palpable. The foetal heart rate was 144/min regular.

Musculo-skeletal system

The right lower limb appeared normal. It was not swollen and was non-tender. The left lower limb was massively swollen, shiny and tender. Circumferential calf and thigh measurements were taken for both lower limbs. The results revealed that the left limb had much bigger circumference.

Diagnosis

A clinical diagnosis of deep venous thrombosis was made.

Management

The patient was admitted for investigations and treatment. She was started on intravenous heparin infusion of 40 000 IU in 24 hours and mefenamic acid 500 mg 8 hourly. The affected limb was elevated and she was put on bed rest. Daily thigh and calf measurements were taken.

Serial measurements were as follows;

Date	right thigh	right calf	left thigh	left calf
14. 04.02	50	35	56	38
15. 04.02	50	35	55	37
16. 04.02	50.5	35	55	37
17. 04.02	50.5	35	54	36
18. 04.02	50	35	52	36
19. 04.02	50	35	51	35

The pain and swelling progressively resolved. She was then maintained on heparin 5000 iu twice daily, which she continued on discharge.

Investigations

KCCT	test	control
14. 04.02	30.8 sec	34.5
16.04. 02	35.3 sec	34.0
20.05. 02	45.4 sec	33.8
15.06. 02	40.2 sec	34.5
05. 07.02	38.7 sec	30

Haemogram

Hb 13.4 gm/l WBC 6.1 x 10^9/l platelets 400 x 10^{12}/l

Doppler flow studies revealed normal venous flow in the right lower limb. There was a large thrombus in the left common iliac and left femoral veins. Flow in the superficial femoral veins was sluggish. This confirmed the diagnosis of deep venous thrombosis.

Obstetric ultrasound showed a single intrauterine foetus at 30 weeks gestation.

Further management

The patient was taught how to inject herself. She was allowed home on subcutaneous heparin 5000 IU twice daily. She was seen in the antenatal clinic after one week. She was well with no complains. She continued with the heparin and routine antenatal clinic. Repeat Doppler flow studies after 2 weeks revealed that the thrombi had

resolved. The left femoral vein was dilated with sluggish flow but no thrombus. During the visits contraception options were discussed with her and she opted for post-partum bilateral tubal ligation (BTL).

The patient then came to the labour ward at 39 weeks gestation in early labour. The morning dose of heparin was omitted and a sample of blood for grouping and cross matching was taken. She progressed well to have a spontaneous vaginal delivery to a live male infant. The birth weight was 3400 gm, score 8/1 and 10/5. The placenta was delivered by controlled cord traction. The estimated blood loss was 300 ml. The following day a BTL was done under local anaesthesia. Twelve hours later heparin 5000 iu subcutaneously was restarted together with 5 mg of warfarin. After 3 days the heparin was stopped. She was discharged on warfarin to be seen in the postnatal clinic after one week.

Follow up

She came for review in the postnatal clinic after one week. She had no complains. A coagulation screen was done and was acceptable. She was seen again after 5 more weeks and was well. She was then discharged to continue with warfarin through the haematology clinic for further follow up.

Discussion

The patient discussed was a para 2+0 who developed deep venous thrombosis in pregnancy, was managed and had a good outcome.

Thrombosis is the process by which blood flowing through the vascular system coagulates into a solid mass. Such coagulation without apparent antecedent inflammation is known as phlebothrombosis. When the coagulation results from vessel inflammation, it is referred to as thrombo-phlebitis [1, 2]

The incidence of deep vein thrombosis (DVT) varies and ranges 1 – 5 per 1000 pregnancies.[3] In KNH it was reported to be 1.6 in 1000. Pulmonary embolism can arise following thrombosis with a mortality rate of 12 – 15% .[4]

Pregnancy is a physiologic state in which many changes that occur favour thrombosis. The three elements of the virchous triad (circulatory stasis, vascular damage and hypercoagulability) are all enhanced during pregnancy. Vascular stasis is encouraged by increase in calibre of vessels and pressure on the lower extremities from the gravid uterus. High levels of progesterone lead to decreased vasomotor tone. Several factors contribute to hypercoagualable state. Coagulation factors VII, VIII and fibrinogen are increased markedly. Natural anticoagulants, antithrombosis III and proteins are decreased. Vascular damage is likely to occur at delivery. [1,7] The patient discussed was pregnant and hence had a hypercoagulable state.

Anaemia, haemorrhage, cardiovascular diseases and hypertensive diseases increase the risk of developing thrombosis. Other risk factors include past history of thrombosis, maternal age more than 35 years, smoking, cancers, surgery, immobilisation and use of oral contraceptive pills. [1,5,6,7] The patient discussed was pregnant and over 35 years but did not have these other risks. Inherited thrombophilias lead to an increased tendency to thrombosis. They include deficiencies of antithrombin 111, protein C protein S and factor V leiden mutation. [7, 8] The patient presented was not known to have any thrombophilias and there was no history of thrombophilias in the family.

Almost 90% of DVT in pregnancy affects the left side compared to 55% in non-pregnant women. This may be due to compression of the iliac vein by the right iliac and the ovarian arteries, which cross the vein on the left side. Most of the DVT occurs on the iliofemoral venous sinuses (72%) rather than the calf veins (9%). Iliofemoral thrombosis poses a greater risk of pulmonary thrombo-embolism. With calf vein thrombosis, the risk is negligible. However there is a tendency of the thrombi to extend proximally if not treated. [6, 7] The patient discussed had left limb thrombosis with the thrombi in the left common iliac and left femoral veins but did not develop pulmonary thrombo-embolism.

Classically DVT presents with swelling of the affected limb, pain, tenderness, local cyanosis and fever. However clinical diagnosis is neither sensitive nor specific. Most deep vein thrombi are completely asymptomatic. The symptoms severity does not co-relate with the risk of thrombosis. Clinical evidence of DVT precedes embolism in only about half of the cases. Thus nearly half of patients remain asymptomatic until the pulmonary embolism has occurred. Symptoms of pulmonary embolism include dyspnoea, chest pain, tachypnoea, cough and haemoptysis. Chest X-ray, ventilation perfusion scintigraphy and pulmonary angiogram are useful investigations. [1, 8, 9] This patient presented with lower limb swelling and pain. She did not have any symptoms of pulmonary embolism.

Various techniques are available to facilitate objective diagnosis of DVT. They include contrast venography, impendence plethysmorgraphy, I^{125} – fibrinogen scanning, biochemical assays and doppler ultrasonography. Contrast venography is the gold standard but it is expensive, cumbersome and time consuming. The non-invasive doppler ultrasound is now widely used for diagnosis. It has sensitivity of 76-94% and specificity of 90-95% for proximal DVT, but a lower sensitivity for calf DVT. Magnetic resonance imaging and the computed tomographic scanning can also be used. In the patient presented doppler flow studies were done to confirm the diagnosis. L K had the diagnosis confirmed using doppler flow studies.

Treatment of DVT consists of anticoagulation analgesia and bed rest. Heparin is the drug of choice. It markedly increases the activity of antithrombin III. Initial treatment is usually by continuous infusion with a loading dose of 5000 units followed by

25,000 – 40,000 units over 24 hours. Heparin may also be given subcutaneously. The therapeutic effect of heparin should be monitored to ensure sufficient anticoagulation without causing spontaneous bleeding. The activated partial thromboplastin time is used for the monitoring and should be 1.5 – 2.5 times the control value.
Low molecular weight heparin can also be used safely. It has the advantage of once or twice daily administration with fewer bleeding complications. [8,10] Most authors recommend that the drug does not have to be monitored .[11, 12] Heparin does not cross the placenta and is not secreted into breast milk and there is no evidence suggesting any adverse foetal effects with its use. [13] The patient presented was treated with heparin with good results.

With this management, pain soon is relieved. Once the symptoms have subsidised graded ambulation should be started. Warfarin is an oral anticoagulant that is suitable for treatment of DVT in puerperium. It is usually started when heparin treatment is continuing since it takes at least 72 hours to take effect. Heparin therapy is then discontinued and warfarin is continued for 3 – 6 months into the pueperium. [14]
Warfarin crosses the placenta and has been associated with teratogenic effects. It is therefore best avoided during pregnancy. The teratogenic effects include nasal hypoplasia, skeletal abnormalities, multiple central nervous system abnormalities and retarded development. Foetal and placental bleeding leading to intrauterine foetal demise have been described. Another disadvantage of warfarin is that its antidote, the vitamin K, works in at least 24 hours. For faster reversal of its effects fresh plasma is needed [1, 2, 15] If warfarin must be used in pregnancy then it should be avoided in the first trimester, to reduce chances of embryopathies, and after 36 weeks when chances of going into labour without due preparation for delivery are higher. Warfarin therapy is monitored by the use of the prothrobin time, which should be 1.5 – 2.5 times the control. [1, 15] The patient discussed was maintained on heparin during the pregnancy and warfarin was used in the puerperium.

In the mother heparin causes osteoporosis, thrombocytopenia and bleeding disorders. It should not be given if the platelet count is below 50 x 10^9/L.Osteoporosis is reduced by administration of calcium and vitamin D. the antidote of heparin is protamine sulphate. It should be given at the dose of 1mg per 100 units of heparin. Excess protamine sulphate should be avoided since it has anticoagulant effects. [1, 7, 15]

During the time of labour and delivery, the heparin should be withheld. If the uterus is well contracted and there is negligible trauma to the genital tract heparin may be restarted within 6 hours post partum. Warfarin can be initiated together and heparin withdrawn within 5 days. Warfarin should be continued for 3 – 6 months postpartum.[8,14] In our patient heparin and warfarin were started 24 hours later since she had a post partum BTL the morning after delivery, to reduce the risk of bleeding.

Contraception containing estrogens should be avoided because they increase the risk of thromboebolism. [16,17] The patient discussed had a bilateral tubal-ligation done since she had attained her desired family size.

References

1. Manoj K B, Perloff D. Hematologic disorders in pregnancy in currents Obstetric & Gynecologic Diagnosis and Treatment, Appleton Lange 8th Edition 1994 22; 448-457.
2. Bonnar J. Venous thrombosis and pulmonary embolism. Turnbulls obstetric 2nd edition church hill Livingstone 1996; 44 789 – 802
3. Rutherfords, Montora M, Mc Grechee W, storng J: Thromboembolic disease associated with pregnancy. In all year review. *Am J obstet gynaecol 1991; 164 supp 286*
4. Waweru J M. Deep venous Thrombosis MMed thesis UON 1981
5. Anthonie L W, A Prandoni P, Martin HP et al; Deep venous thrombosis *Lancet 1999; 353 479-85*
6. Smith R S, Lee W cotton D B. Critical care in obstetrics in Current Obstetric & Gynecologic diagnosis and management, Appleton Lange 8th Edition 59; 1064-1085.
7. Greer I A; Thrombosis in pregnancy: Maternal and foetal issues *Lancet* 353; 1258, 1999
8. Cunningham G R Gant F N et al; Preterm birth in Williams Obstetrics Mc Gray Hill 21st Edition. 27; 690-718, 2003.
9. Pioped Investigators: Value of the ventilation perfusion scan in acute pulmonary embolism results of prospective investigation of pulmonary embolism diagnosis (PIOPED) JAMA 263; 2753 1990
10. Columbus investigators low molecular weight heparin in the treatment of patients with venous thromoembolism *N Engl J Med 337; 657, 1997*
11. American college of obstetricians and gynaecologists; Anticoagulation with low molecular weight heparin during pregnancy committee on obstetric practice committee opinion no 211, Nov 1998
12. Lensing A W, buller HR et al; Deep-vein thrombosis Lancet 353; 479, 1999
13. Lock wood C T; Thromboembolic disoders of pregnancy. Medicine of the foetus and mother. 2nd edition Lippincott, Philadelphia 1999; 1181 – 1189
14. Kearon C, Gent M et al; A comparison of 3 months of anticoagulation with extended anticoagulation for a first episode of idiopathic venous thromboembolism N Engl J med 340; 901, 1999

15. Janice E W, Mitchell P D; Venous thrombosis and pulmonary embolism, obstetrics normal and problem pregnancies 4th edition. Churchill, new York 2002; 1046 – 1053
16. Ronald T Burkman, Contraception and family planning in Current Obstetric & Gynecologic diagnosis and management, Appleton Lange 8th Edition 1999 33; 670-686.
17. Venous thromboembolism and hormonal contraception; The Royal college of obstetricians and gynaecology clinical guideline, Oct 2004.

OBSTETRIC CASE 2

CORD PROLAPSE - EMERGENCY CAESERIAN SECTION DONE

Ip no.	0920432	D.O.A.	18.9.03
Name	F W	D.O.D	23.9.03
Age	25	Parity	1 + 0
Sex	Female		

Presenting complaints

The patient was admitted through casualty with complains of labour pains for 5 hours at 38 weeks gestation. She had not drained any liquor. While in the labour ward she had spontaneous rupture of membranes.

History of present pregnancy

Her last menstrual period was on 23.12.02 and the EDD was on 30.9.03. At admission she was at 38 weeks gestation. She had attended antenatal clinic in KNH from 24 weeks gestation. Her antenatal profile was

HB – 12.5 g/dl	VDRL – negative
Blood Group - A +Ve	Elisa for HIV – negative

Past obstetric and gynaecological history

She was para 1 + 0. Her last delivery was in 2000 by spontaneous vertex delivery. The baby was alive and well. She attained menarche at 15 years. Her cycle is regular every 27 – 29 days with a normal flow of 2 – 3 days. She had used Depo-Provera after the previous delivery.

Past medical history

This was non-contributory.

Family and social history

She was a housewife, married to a carpenter. They lived in Kibera. She did not smoke cigarettes or drink alcohol. There was no history of chronic illness in the family.

General examination

She was in good general condition not pale. She had no oedema or lymphadenopathy. Her vital signs were BP 110/70mhg, Pr – 86/min RR 22/min temp 36.8°C

Her respiratory, cardiovascular and central nervous systems were within normal.

Abdominal examination

The abdomen was uniformly distended, and moving with respiration. The fundal height was term, the foetus was in longitudinal lie and cephalic presentation. The foetal heart rate was 144 beats per minute, regular. She had moderate contractions 3 in 10 minutes.

Vaginal examination

While on the examination couch, she experienced spontaneous rapture of membranes and clear liquor gushed out wetting the bed. The external genitalia was normal. The vaginal cavity was filled with a warm pulsating prolapsed cord. The cervix was about 5 cm dilated.

Diagnosis

A diagnosis of cord proplapse was made.

Management

The patient was placed in the Trendelelenburg position and oxygen was administered. The condition and the need for an emergency caesarean section were explained to her. Theatre was alerted immediately. The patient signed the consent form. Blood was taken for grouping and cross matching and an intravenous infusion of 5% dextrose was started. She was premeditated with atropine 0.6 mg intramuscularly and taken to theatre.

When she was placed supine on the operation table, the patient's hips were raised using a pillow. A caesarean section was done. The outcome was alive male infant, birth weight of 3300 gm and scored 8/1, 10/5. The baby did well and stayed with the mother. Post operatively the patient did well and both her and the baby were discharged on the 4th postoperative day.

Post natal clinic

The patient was lost to follow up

Discussion

The patient presented had umbilical cord prolapse following spontaneous rupture of membranes while in the hospital. An emergency caesarean section was done and the baby had a good apgar score.

Umbilical cord prolapse (UCP) is the descent of the umbilical cord into the lower segment of the uterus. It is termed as overt when it lies below the presenting part. In occult cord prolapse the cord lies adjacent to the presenting part. Funic presentation refers the UCP below the level of the presenting part before the rupture of the membranes. This patient had overt UCP.

Umbilical cord prolapse occurs when the cord descends beyond or alongside the presenting part. The cord is displaced in to the vagina or even through the introitus once the rapture of membranes occurs. Umbilical prolapse is an uncommon obstetrical emergency. The incidence varies and is about 1 in 200 – 300 deliveries. In the Nairobi birth survey Mati reported an incidence of 1.8% while Ochiel reported and incidence of 0.57% in KNH. [2, 3] While the incidence of UCP has not changed much over the years, the associated prenatal mortality has fallen significantly in the last several decades. [4] The outcome for our patient was a live baby.

Umbilical cord is more likely to prolapse when the foetal presenting part is not well applied to the cervix. Frequency of cord prolapse increases with small foetus or malpresentation. Breech presentation is the leading cause of UCP accounting for 40 – 50 % of the cases. With frank breech presentation the incidence is about 0.5% while with footling breech the incidence is 15% and 5% with complete breech. [5, 6] Other presentations associated with increased risk of UCP are transverse lie, face and brow presentations.[1] Our patient had cephalic presentation at term, but the head was not well applied to the cervix.

A contracted pelvis predisposes to mal-presentation and to the presenting part remaining high. With a contracted pelvis, face and shoulder presentations are encountered 3 – 4 times more frequently and cord prolapse occurs 4 – 6 times more frequently.[6] Pre-maturity accounts for 20% of UCP. This is related to the relatively

small size of the foetus and higher incidence of mal-presentation. Multiple gestations are associated with higher incidences of pre-maturity and mal-presentation and are therefore associated with higher incidences of UCP. Other factors associated with UCP include polyhydramnios, placenta praevia and pelvic tumours. Forceful gush of amniotic fluid after membrane rupture may sweep the cord along leading to prolapse. [1,7,8,9] The patient presented had a forceful gush of liquor with no other notable risks of cord prolapse.

UCP is associated with significant perinatal morbidity and mortality. With overt prolapse the perinatal mortality rate approaches 20%. Once a cord has prolapsed, there is increased risk of compression of the cord between the presenting part and the pelvic inlet, the cervix or the vagina. Compression may occur especially during the uterine contractions. Cord compression results in foetal circulation compromise. Immediate caesarean section delivery is indicated. Cord prolapsed through the introitus exposes it to cooling and irritation that leads to vasospasm of the vessels. It is important to replace the cord back to the warm moist vagina or wrap it with a warm moist towel.[7,9] In this patient the out come was good.

Patients with risk factors for cord prolapse should be treated with the appropriate precautions to prevent UCP. Continuous monitoring in labour will detect foetal heart rate abnormalities resulting from cord compression. When the presenting part is high, artificial rupture of membranes (ARM) should be avoided. If ARM is required with the presenting part high, then the presenting part should be stabilized and slow controlled release of the amniotic fluid done until the presenting part settles against the cervix. Some prefer to rupture the membranes during a contraction to ensure the presenting part is pushed towards the pelvis. If spontaneous rapture of membrane occurs, prompt careful pelvic examination should be performed to rule out cord prolapse. The patient presented had spontaneous rupture of membranes, prompt examination was done and the cord prolapse was detected. During the pelvic examination the strength and frequency of the pulsations should be ascertained. The degree of cervical dilation, the nature and station of the presenting part and the adequacy of the pelvis should also be noted. [7,9]

Overt cord prolapse is a condition demanding immediate action. Preparation for a rapid abdominal delivery should be made. Meanwhile the patient should be placed in the knee-chest position and continuous upward pressure applied against the presenting part to maintain the foetus away form the prolapsed cord. Oxygen should be given to the mother. However if the cervix is fully dilated there is no cephalo-pelvic disproportion and delivery is imminent, then a vaginal delivery may be accomplished. Delay of more than 30 minutes increases the morbidity and mortality four fold. [1, 7, 9] The patient discussed was delivered within 30 minutes and the outcome was good.

Occult cord prolapse may be suspected from cord compression pattern (variable decelerations) of the foetal heart rate. If overt UCP is ruled out by a pelvic examination, the patient should be placed on the lateral Sims or trendelenburg position. Oxygen should be administered to the mother. If the cord compression pattern persists, the patient should be delivered via a rapid caesarean section.

A patient noted to have funic presentation at term should be delivered by a caesarean section. If the foetus is premature, hospitalisation with bed rest in the Sims or trendelenburg position should be done. Serial ultrasound should be done to determine any changes in the cord presentation and the gestation age. Elective caeserian section for non-cephalic presentation reduces the incidence of cord prolapse. [10]

Rapid forceful instillation of 500 – 700 ml of saline into the urinary bladder via a foleys catheter has been shown to be useful in reducing cord compression. The distended bladder maintains the raised position of the presenting part.

Depending on the degree and duration of cord compression, the neonate at delivery may be hypoxic, acidotic or moribund. A paediatric team should be present for resuscitation of the newborn. For our patient the paediatrician was present to resuscitate the newborn. Fortunately the infant was healthy and did well.

References

1. Whitefield C R, Cord prolapse in Dewhursts textbook of obstetrics and gynaecology for postgraduates. 5th Edition Blackwell Scientific Publication.
2. Mati J K G, Sanghvi HSG, Aggarwal UP et al; Nairobi birth survey. *Jour obstet gynaecol East and Cent Africa 2: 47, 1983*
3. Ochiel O; Management of 112 cases of cord prolapse at KNH; MMed Thesis UON
4. Panter K R, Hannah M E, Umbilical cord prolapse; So far so good? *Lancet 1996 347:74*
5. Fischer R. Breech Presentation. May 2004. www.e medicine
6. Crichlow L W, Leet T L. Benedetti B J, Daling J R; Risk factors and infant outcomes associated with umbilical cord prolapse. A population based case control study among births in Washington State. *A M J obstet gynaecol 170: 613, 1994*
7. Cunningham F G, Leveno J K, et al; Breech presentation and delivery; Williams obstetrics 21st edition McGraw hill 2003 22: 513-528
8. Collea J V, Malpresentation and cord prolapse in Current Obstetric & Gynecoloic Diagnosis and Treatment 8th Edition Appleton Lange 1994 21; 424-426.
9. Usta I M, Mercer B M, Siban B M, Current obstetrical practice and umbilical cord prolapse *Am J prenatal 1999 16:479*
10. D C Dutta, Malposition, Malpresentation & Cord Prolapse in Text book of Obstetric, 6 th Edition Central 2004, 25: 398-401

OBSTETRIC CASE 3

VAGINAL BIRTH AFTER PREVIOUS CAESAREAN SECTION – SUCCESSFUL TRIAL OF SCAR

Name	M N	IP No	0981435
Parity	2 + 0	DOA	22.10.02
Age	30yrs	DOD	24.10.02

Presenting complain

The patient was admitted with complains of labour pains for 6 hours.

History of presenting complain

M N had developed low abdominal pain 6 hours prior to admission. The pain radiated to the back. It had been increasing in intensity and frequency. There was no associated drainage of liquor or per vaginal bleeding.

Obstetric and gynaecological history

She attained menarche at 15 years. Her cycle was 28 days regular with a flow lasting 3 – 4 days. She had used oral contraceptive pills prior to this pregnancy. She was para 2 + 0. Her first delivery was in 1999. It was a spontaneous vertex delivery to a live male, birth weight of 3700gms. In 2000 she had her second delivery by caesarean section due to foetal distress. The baby weighed 3400 gm and was alive and well.

History of current pregnancy

Her last menstrual period was on the 21.1.02 and her EDD was on 28.10.02 so she was at 39 weeks gestation by dates. She had attended antenatal clinic since 24 weeks gestations in a city council clinic. She had made a total of 10 visits. The antenatal profile was as follows

Hb – 12.2 g/dl	VDRL – negative
Blood group – B+ve	Elisa for HIV – negative

An erect lateral pelvimetry was not done, neither was an ultra sound done. She had uneventful antenatal period.

Family and social history

She was married and worked as a clerk in the city council. She lived in Umoja with her family. There was no known chronic illness on her family. She did not take alcohol or smoke cigarettes.

Past medical history

This was not contributory

Physical examination

She was in good general condition and not pale. Her vital signs were PR – 22/mg PR – 82/min BP – $^{120}/_{80}$mhg tem – 36.8°C

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

Abdominal examination

The abdomen was uniformly distended with a sub umbilical midline scar. The fundal height corresponded to term gestation. The lie was longitudinal with a cephalic presentation. The presenting part was $^{3}/_{5}$ above the pelvic brim. The foetal heartbeat was heard and was regular at 144 beats/ min. she was noted to have moderate contractions 3 every 10 minutes. There were no areas of tenderness.

Pelvic examination

The external genitalia was normal. The cervix was 4 cm dilated, soft, in the central position and 60% effaced. The pelvis was clinically adequate. The head was well applied to the cervix with no caput or moulding. In view of the fact that she was in active phase of labour, with one previous scar due to a not necessarily occurring indication and a previous vaginal delivery, a decision for trial of scar was made. The patient was explained to the findings and she consented for trial of scar.

Artificial rapture of membrane was done and clear liquor was obtained.

Further management

Blood was taken for grouping and cross matching. An intravenous infusion of 5% dextrose was set up. The patient was monitored closely with special attention to the foetal heart tones, her general condition pulse rate and for any uterine tenderness. The labour progressed well with no features of maternal or foetal distress. The cervix

dilated well, being 7cm after 2 hrs. Two hours later she had the urge to push and was taken to the 2nd stage room. She had a spontaneous vertex delivery to a live female infant birth weight of 3300gms and apgar score of $^{9}/_{1}$, $^{10}/_{5}$. The placenta was delivered complete with membranes by the cervix vaginal walls and perineum were examined and found to be intact since. The estimated blood loss was 250 ml. Exploration of the uterine scar was not done. Vital signs examination continued being monitored and she remained stable. After close observation for 4 hours she was discharged to the postnatal ward. Both her and the baby remained well. They were discharged home after being observed in the postnatal ward for 24 hours.

Follow up

She came for postnatal clinic after 6 weeks. She had no complaints. The baby was breastfeeding well and the mother was in good condition. She was discharged through the family planning clinic.

DISCUSSION

M N was a para 2 + 0 who had undergone a caesarean section during her last delivery. She presented in active labour and had a successful vaginal delivery.

The caesarean section (C/S) delivery rate has increased throughout the world.[1] For many years the scarred uterus was believed to be a contra indication to vaginal delivery. However reports of successful trials have lead to significant increase in attempts of vaginal birth after (VBAC).[2] A success rate of vaginal delivery after caesarean section is reported to be 53.1% at Kenyatta National Hospital.[3] In the US there was a 14 – fold increase of women with prior caesareans delivering vaginally by 1996.[4]

Vaginal delivery is associated with fewer delivery risks, requires less anaesthesia, poses a lower potential for postpartum morbidity, involves shorter hospital stay, saves money and encourages earlier and often smoother interaction between mother and infant.[5] Therefore since a significant number of caesarean sections are repeats, if VBAC is widely adopted appropriately there would be a substantial and beneficial reduction in the incidence on caesarean section. Success rtes of trial of scar have been reported to be 60 – 80% in the USA when the obstetrician promotes VBAC. In Kenyatta National Hospital a success rate of VBAC was reported as 73.9% in 1982. [6] Walton found previous scars to be the commonest indication for elective caesarean section (53.3%) followed by contracted pelvis (37.4%) [7]

The success of the trial depends on the obstetric history and the indications for the previous caesarean section. When previous section was due to breech presentation or foetal distress, the trial of scar is more likely to be successful than when the indication of previous scar was cephalopelvic disruption (CPD).[4] However CPD as the cause of the previous scar is not a contraindication for trial.[8] A woman who has ever had a vaginal delivery whether prior to or after the caesarean section is more likely to have a successful trial than one who has never delivered vaginally [9]. The patient discussed had a previous vaginal delivery and the indication for the previous C/S was foetal distress. She was therefore a good candidate for successful trial of scar.

Criteria for vaginal delivery following a previous caesarean section may include the following, according t the American college of obstetricians and gynaecologists.[10]

1. One or two prior low transverse caesarean deliveries
2. No other uterine scars or previous rupture
3. Physician immediately available throughout active labour capable of monitoring labour and performing an emergency caesarean delivery
4. Availability for anaesthesia and personnel for emergency caesarean delivery
5. Clinically adequate pelvis

Other requirements for vaginal delivery after a previous caesarean section include the patients consent to the procedure, original indication not to be a necessarily recurring one, the post operative course after the caesarean section to have been benign and the current pregnancy not to be complicated by conditions (such as macrosomia, malposition, multiple gestation etc) that could likely preclude vaginal delivery. In our set up VBAC is confined to women with only one previous caesarean section not two. M N met the criteria for trial of VBAC.[10]

Use of oxytocin to induce or augment labour has been implicated in uterine raptures in women with prior caesarean deliveries. [12] However its use is not contraindicated but dose monitoring needs to be done.[10,11,13] The use of epidural anaesthesia is controversial. It is argued that its use would potentially mask rupture thereby jeopardizing both the mother and the infant. Those in favour of the epidural anaesthesia argue that its use removes the fear of labour and helps encourage VBAC.[11] Our patient did not need augmentation. She was not given any analgesia but she tolerated labour well.

The most feared complication of VBAC is uterine rupture. This is not very common when the uterine incision was low transverse. Uterine rupture is a small but significant risk and is associated with poor outcomes for both mother and infant. During a trial of scar measures for managing a rupture should it occur must be in place. Equipment for maternal and electronic foetal monitoring is helpful in diagnosis of an impending rupture or early rupture. A large bore needle for intravenous access and blood for maternal transfusion should stay ready. Facilities for immediate laparotomy and neonatal resuscitation should be readily available. [4, 10, 11, 13] Our patient had a large

bore needle in place during the trial. Though there was no electronic foetal monitor clinical monitoring of both the mother and the foetus was done. Facilities for immediate laparotomy were available if the need arose.

Following a successful VBAC it is important to look out for significant bleeding that may an indication for surgical repair of any scar desistence. Some authorities advocate for routine exploration of the scar to feel for any dehiscence.[14] Some, for fear of enlarging small separations and increasing the risk of endometritis, discourages this practice. [4] M N did not have significant bleeding and exploration of the scar was not done. She did develop any complications in the post partum period.

REFERENCES

1. National Centre for Health Statistics: Rates of caesarean delivery--United States, 1991.
2. Rosen MG, Dickinson JC: Vaginal birth after caesarean: A meta-analysis of indicators for success. Obstet Gynecol 1990 Nov; 76(5 Pt 1): 865-9
3. Githuru PK; Value of erect lateral pelvimetry in management of patients with one previous scar. Mmed Thesis U.O.N 1992
4. Cunningham GF, Grant FN, Leveno JK et al Caesarean delivery and postpartum hysterectomy, Williams obstetrics 21st Edition 2003 McGraw hill 23:538-553
5. National Institute Of Health Consensus Development Task Force 1981 statement on caesarean childbirth. *Am Jour obstet and gynaecol 139; 903 7*
6. Karanja J. G. Review of caesarean delivery at Kenyatta national hospital in 1980 Mmed Thesis UON 1982
7. Walton SM, The antenatal and intrapartum management of patients with one previous scar. *East African Medical Journal vol 55 No 1. pp3 1978*
8. Bedoya C Bartha J Rodigne et al. a trial of scar after caesarean section inpatients with or without prior vaginal delivery. *Int J gynaecol obstet 1992; 38 285 – 289* (level iii)
9. Handle CB, Menutti MJ, Gibbres S.G; Evaluating of the relative risks of trial of labour Vs elective caesarean section AVA. Journal of perinatal 1986; 3 107 – 114
10. American college of obstetricians and gynaecologists vaginal birth after previous caesarean delivery practice bulletin no. 5 July 1999.
11. Hale W.R.; Operative delivery, Current Obstetric & Gynaecological Diagnosis and Treatment. 8th Edition 1994 Appleton & Lange, 571
12. Turner MJ Delivery after one previous caesarean section AM J obstetric gynaecol 176; 741, 1997
13. D C Dutta, Text book of Obstetric, 6 th Edition Central 2004; 22: 327-330

OBSTETRIC CASE 4

DIABETES IN PREGNANCY

Name	A K	Sex	Female
Ip No.	438452	D.O.A.	09.12.03
Age	28	D.O.D	14.12.03
Parity	0 + 0		

Presenting Complaints

She was admitted through the antenatal clinic due to high blood sugar.

She did not have any complains. She reported no history of polyuria, polydipsia or polyphagia.

History Of Current Pregnancy

Her last menstrual period was on 29.05.03 her expected date of delivery was 05.03.04 she was therefore at 28 weeks at admission. She was attending antenatal clinic in the hospital since 15 weeks of gestation. She had been counselled on the need for compliance during the pregnancy. The nutritionist had reviewed her diet.

The antenatal profile was as follows.

HB – 12.5 g/dl	Blood Group – B +Ve
VDRL – negative	ELISA for HIV –not done

An ultra sound done at 20 weeks gestation revealed a normal intra uterine foetus, grossly normal, appropriately grown for the age. The average ultra sound gestation corresponded to the dates. The liquor was adequate, and the placenta fundal posterior

Gynaecological History

She attained menarche at 15 years. Her cycle was regular, lasting 3-4 days in a cycle of 28-30 days, normal flow with mild dysmenorrhoea. She had never used contraceptives.

Past Medical History

She was known to have diabetes for 5 years. She had been well controlled on insulin outside of the pregnancy. She did not have any other chronic illness.

Family and Social History

A married lady for two years and was living with her husband. She was a banker by profession. Her aunt was diabetic but no other chronic illness in the family. She did not consume alcohol or smoke cigarettes.

Physical Examination

She was in good general condition. She was not pale and had no oedema or lymphadenopathy. Her vital signs were BP – 110/70mhg PR – 84/min PR-22/min temp – 36.8oC

The random blood sugar was 22 mmol/l

Her respiratory, cardiovascular and central nervous systems were normal.

Abdominal Examination

The abdomen was uniformly distended. The fundal height was corresponded to 30 weeks gestation, with a longitudinal lie and cephalic presentation. The abdomen was soft and non-tender. The foetal heart rate was 144 beats per minute.

Pelvic Examination

A sterile speculum exam was done. Her external genitalia was normal. The cervix was long, posterior and closed. Grossly it appeared normal with no signs of infection.

Diagnosis

A diagnosis of insulin dependent diabetes out of control at 28 weeks gestation was made.

Management

She was admitted and rehydration with normal saline intravenously started. She received 10 iu of soluble insulin and was started on mixtard insulin 20 iu in the morning, 10 iu evening and 8 iu of soluble insulin at lunch. The insulin was titrated against the blood sugar until satisfactory control was achieved.

Investigations

Serial blood sugars were done and insulin titrated accordingly.

Haemogram parameters were within normal. Urea, electrolytes and creatinine were all normal. An electrocardiogram done was normal.

Hb_{IC} was 8, urinalysis revealed ketones +, sugar +++

The blood sugar fell to be consistently below 6 mmol/l and she was discharged home after five days of hospitalisation to be followed up in the antenatal clinic.
She bought a glucometer and was shown how to take her blood sugar at home and keep record. She already knew how to inject herself with insulin.
The nutritionist reviewed her diet and was encouraged to be compliant even at home.
She continued follow up in the clinic without any major problems. She had 2 episodes of vaginal candidiasis and was successfully treated with clotrimazole vaginal pessaries.

An ultra sound scan was repeated at 36 weeks gestation. It revealed an appropriately grown foetus for the gestation, with no abnormality. The placenta was fundal posterior, not low lying. There was no cord round the neck. There was mild polyhydramnios. The biophysical profile was 8/8. The estimated foetal weight was 2500 gm.

At 38 weeks she was admitted complaining of labour pain. A vaginal examination done revealed a cervix that was 4 cm dilated, soft, central, 70% effaced with slightly bulging membranes. No cord was felt. Artificial rupture of membranes was done and clear liquor was obtained. She was started on syntocinon drip, 5 IU in 5% dextrose. Another drip, half litre 5% dextrose with 5 units of insulin was set up. Blood sugar estimation was done every hour and the insulin drip adjusted accordingly. Vaginal examination was done after 4 hrs. She was found to be 9 cm dilated the head had descended and normal vaginal delivery was anticipated. She reached second stage soon after and had a spontaneous vertex delivery to a live male infant, who weighed 3400 gm and the APGAR score was 9 at 1 and 10 at 5. Blood sugar monitoring continued in the post partum period and the insulin dose was adjusted. The mother did well and was discharged home on the 4th post partum day to come again for follow up in the postnatal clinic after one week.

Follow up

A K came back for review after one week. She did not have any complaints. The baby was breastfeeding well. The lochia was now serosa and not foul smelling. The blood sugar was well controlled. She came again foe review after 5 weeks. During this visit the contraception choices were discussed with her. She opted to use condoms with spermicides. She was then discharged through the family planning and the medical outpatient clinics to continue with follow up.

Discussion

A K was known to have diabetes. During the routine antenatal follow up she was noted have elevated blood sugar. She was managed and had a normal delivery.

Diabetes mellitus is a chronic disorder of metabolism affecting carbohydrates, proteins and fats. The incidence varies in different populations. In the USA it complicates about 2.6% of all live births and is the most common medical complication of pregnancy[2]. Prior to introduction of insulin diabetic women suffered infertility and the few who conceived faced a dismal prognosis. With proper control maternal and foetal outcomes have improved significantly. A K had good outcome.

Diabetes is classified as type 1 (insulin dependent) or type 2 (non insulin dependent). Type 1 diabetes is immune mediate developing in genetically susceptible persons. It is associated with low vertical transmission. Type 2 diabetes has familiar occurrence. It is due to abnormal insulin secretion and insulin resistance in target tissues. Most patients are overtly obese. Patients can be grouped into those who were known to have diabetes before pregnancy (overt) and those diagnosed during pregnancy (gestational). A K was known to have diabetes type 1 before the pregnancy.

The physiological changes in pregnancy in diabetics result in a decrease of carbohydrates control reserves. Pregnancy is therefore a unique opportunity for screening for diabetes. This allows the woman to discover or prevent diabetes. Since diabetes has adverse effects on the foetus and the mother it is imperative to discover and control diabetes in pregnancy. Ketoacidocis is a threat to the maternal life and hyperglycaemia increases the risk of midline birth defects 6 fold in a developing embryo. Diabetes is clinically recognized by a relative paucity of insulin, hyperglycaemia, glucosuria and ketoacidosis. Women with high plasma glucose levels may present with classical signs and symptoms of polydipsia, polyuria and unexplained weight loss. Their fasting blood sugar will be 7 mmol/l or more. Such women should be considered to have overt diabetes. [3] A K very had very high blood sugar levels but did not have the classical symptoms of polyuria and polydipsia.

Detection of glucosuria in pregnancy may reflect augmented glomerular filtration but it warrants further investigation to establish if it is as a result of impaired glucose tolerance.[4] Women with a strong familial history of diabetes have given birth to large infants or have unexplained foetal losses need to be investigated for impaired glucose tolerance.

Gestational diabetes mellitus refers to carbohydrate intolerance with onset or first recognition during pregnancy. Studies suggest that women with fasting hypergylycaemia diagnosed before 24 weeks likely have overt diabetes .[5] Controversy exists regarding the optimal screening for gestational diabetes. Some authorities have recommended universal screening while others recommend selective screening. In our set up due to cost, selective screening is practised. Screening is best done between 24 and 28 weeks. [6] In our set up the 100 g 3 hour oral glucose tolerance test after an overnight fast is used for screening. The most important perinatal concern in women with gestational diabetes is excessive foetal growth. This leads to increased risk of birth trauma due to shoulder dystocia. [7] Unlike in women with overt diabetes foetal anomalies are not increased. [8]

Overt diabetes has significant impact on pregnancy outcome. The outcome is affected by the degree of the glycaemic control but more importantly by the intensity of any underlying maternal cardiovascular or renal disease. Improved foetal surveillance, neonatal intensive care and maternal metabolic control reduce perinatal morbidity and mortality. Foetal deaths resulting from congenital malformations and those that are unexplained remain unchanged by medical intervention .[9] our patient did not have any underlying cardiovascular or renal disease.

First trimester poor glycaemia control may lead to spontaneous abortion. Later in pregnancy the risk of spontaneous or induced premature delivery is increased.[10] Poor diabetic control periconceptionally is associated with increased risk of foetal anomalies. [11] Other complications associated with infants born to diabetic mothers include respiratory distress. Hypoglycaemia and hyperbilirubinaemia. These infants may have cardiac hypertrophy that progress to congestive heart failure. Generally the infants birth weight is skewed consistently towards being heavier compared with

normal pregnancies. Our patient had good glycaemic control in the preconception period and the first trimester.

With pregnancy maternal welfare of the overtly diabetic mother can be seriously jeopardized. However with the exception of retinopathy, the long-term course of diabetes is not affected. With diabetic nephropathy and pre-eclampsia indicated preterm delivery increase substantially. The integrity of renal function is the best single predicator of success in pregnancy. Women with diabetic neuropathy may have gastropathy. This leads to troublesome nausea and vomiting, nutritional problems and difficulty with glucose control. Hypertension induced or exacerbated by pregnancy is a major complication in diabetic women. Diabetic pre-eclamptic patients have a 20-fold increase of perinatal mortality. The risk factors for pre-eclampsia are vascular complications, pre–existing protenuria and/or chronic hypertension. It is not related to glucose control. [12] Ketoacidosis is one of the most serious complications of diabetic pregnancies. It may be triggered by hyper-emesis gravidarum, tocolytics (B Sympathomimetic drugs), infections and use of corticosteroids to induce lung maturity. Ketoacidosis may be as a result of non-compliance. It is a poor prognostic sign for the pregnancy. [13] Diabetic women have increased incidence of infections. The commonest infections include Candida vulvoginitis, urinary tract infections, puerperal infections and respiratory tract infections. These infections may lead to preterm birth [14, 15] Our patient had vaginal candidiasis but did not develop other complications.

The management of diabetes should start before conception, to prevent early pregnancy loss and reduce the risk of congenital malformations. The patients should be well educated.[16] Unfortunately in our set up many pregnancies are unplanned for and patients only come for prenatal care late in pregnancy. The patient discussed did not come early for antenatal clinic. However she had been on follow up by her physician and her blood sugar was relatively well-controlled periconceptionally. Her haemoglobin A_{IC} was 7. Folate should be given periconceptionally and in early pregnancy to reduce the risk of neural tube defects. [17]

Blood glucose control is best done by use of multiple daily insulin injections and diet adjustment. Oral hypoglycaemia agents are generally not used because they may cause foetal hyper-insulinaemia. They have also been associated with an increased

rate of congenital malformations.[18] The amount of insulin needed is titrated according to the blood glucose levels. Care should be taken to avoid nocturnal hypoglycaemia that is more likely in pregnancy. The woman should be taught how to take her own blood sugar using a glucometer and how to inject herself with insulin. Dietary advice should also be given. When a patient is involved in her management, she tends to be more responsible and hence more compliant. In our set many patients do not afford glucometers and even the cost of insulin is prohibitive. This makes management of diabetes in pregnancy a big challenge. Fortunately the patient discussed was able to buy a glucometer and to afford other necessities such as the suggested foods.

Diabetes tends to be unstable in the first trimester followed by a stable period. From 24 weeks gestation insulin requirements increases.[19] These differences are due to high levels of pregnancy hormones that are insulin antagonists. The patient discussed went out of control at 28 weeks of gestation. An ultrasound to search for malformation is done at 20 weeks. Amniocentesis to detect neural tube malfunctions and other anomalies are done routinely in some set ups. Our patient had an ultrasound at 20 weeks gestation that revealed a grossly normal foetus. Amniocentesis was not done.

In the third trimester ultrasound scanning to evaluate for excessive or insufficient foetal growth and to evaluate amniotic fluid volume is advisable. [20] The frequency of evaluations depends on the risk factors present and the affordability. Foetal surveillance is done depending on different protocols. The patient discussed had an ultrasound done at 20 weeks and at 36 weeks gestation. She was done non stress test during the admission.

Delivery is best done after 38 completed weeks to ensure foetal lung maturity. In case of complications such as pre-eclampsia, then amniocentesis is done to determine if lung maturity has been attained. However depending on the indication delivery may be instituted without documented foetal lung maturity. In case of premature labour β sympathomimetic drugs are best avoided since they worsen glucose control and could lead to ketoacidosis. Similarly use of corticosteroids to hasten lung maturity should be done with caution.[6] A caesarean section may be done if macrosomia is suspected to avoid birth injury bur diabetes is not an indication.[21] The patient discussed had a normal vaginal delivery at 38 weeks.

In labour it is important to use short acting insulin because the insulin need will drop drastically after delivery. During the labour and after delivery adequate hydration is important as well as glucose in sufficient amounts to avoid hypoglycaemia. In the first 24 hours post delivery insulin may not be required and thereafter the requirements fluctuate for the following few days. A K had a drip of soluble insulin and dextrose running during labour. Her blood sugar remained well controlled and she continued with a lower dose of insulin in the post partum period.

In labour it is important to use short acting insulin because the insulin need will drop drastically after delivery. During the labour and after delivery adequate hydration is important as well as glucose in sufficient amounts to avoid hypoglycaemia. In the first 24 hours post delivery insulin may not be required and thereafter the requirements fluctuate for the following few days. A K had a drip of soluble insulin and dextrose running during labour. Her blood sugar remained well controlled and she continued with a lower dose of insulin in the post partum period.

REFERENCES

1. Ventura SJ. Martin J A, Et al: Births: Final data for 1998. National vital statistics reports, vol 48 No. 3 Hyattsville 2000

2. Cunningham G. F. Norman F.G leveno K J et al; Diabetes, in Williams obstetrics 21st edition 2003 McGraw hill publishers, 51: 1360-1398.

3. American Diabetes Association: Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes care 22/ Suppl 1): 512, 1999 b

4. Gribble R K. Meir PR, Berg RL: the value of vine screening for glucose at each prenatal visit. Obstetric Gynaecology 86: 405, 1995

5. Sheffield JJ. Casey B M Lucas M J et al Gestational Diabetes. Effects of the degree of hyperglycaemia and the gestational age at diagnosis. J Soc Gyn Inv 6: 6A, 1999

6. American college of Obstetricians and Gynaecologists; Diabetes and pregnancy. Technical Bulletin No. 200, December 1994

7. McFarland M B, Langer O, Fazionit E. Et al: Anthropometric and body composition differences in large for gestational age, but not appropriate for gestational age infants of mothers with and without. J Joc Gynaecological invest 7: 231, 2000

8. Reec E A, Hobbins J C: Diabetic embryopathy: pathogenesis, prenatal diagnosis and prevention. Obstet Gynaecol surv 41: 325, 1986

9. Garner P: Type 1 diabetes mellitus and pregnancy lancet 346: 157, 1995a.

10. Hanson U, Persson B: Outcome of pregnancies complicated by type 1 insulin dependent diabetes in Sweden: Acute pregnancy complications, neonatal mortality and morbidity. Am J perinatal 10: 330, 1993

11. Kitznullier J L Gavin L A et al Preconception care of diabetes: Glycaemic control prevents congenital anomalies. JAMA 265: 731, 1991

12. Garner PR, O'alton ME Dudley DIC Et al Pre-eclampsia in diabetic pregnancies. AM J obstetric gynaecol 163: 505, 1990

13. Pederson J. Andersen et al: Assessors of foetal perinatal mortality in diabetic pregnancy. Analysis of 1332 pregnancies in the Copenhagen series, 1946 – 1972. Diabetes 23 ; 302, 1974

14. Stamlel EF, cruz ML Mimdum F et al: high infections morbidity in pregnant women with insulin dependent diabetes. An under stated complication. Am obstet gynaecol 163: 1217, 1990

OBSTETRIC CASE 5

RHESUS NEGATIVE MOTHER

Name	B M	D.O.A.	16.1.04
Ip No.	438734	D.O.D	19.1.04
Age	28	Parity	0 + 0

Presenting complains

B M was admitted through the antenatal clinic with complains of low abdominal pain for 6 hours

History of presenting illness

She had been well until that day when she starting experiencing low abdominal pain. The pain was intermittent and mild. The pain increased in intensity and frequency. She had been scheduled to come to hospital that day for induction of labour.

History of current pregnancy

Her last menstrual period was 10.4.03 and her expected date of delivery was 17.1.04. She was 40 weeks gestation by dates. She had booked antenatal clinic at 24 weeks gestation. She had made 9 visits to the clinic. The antenatal period was uneventful.

The antenatal profile was as follows;

VDRL negative

Blood group O rhesus negative

Indirect coombs test negative at 28 and 36 weeks.

Her husband's blood group was B rhesus positive.

Normal ultrasound at 24 weeks and at 36 weeks

She received 300 mg of anti D at 28 weeks

Haemoglobin level - 12.8 g/dl

Normal urinalysis

ELISA for HIV - negative

Past medical history

This was not contributory

Past obstetric and gynaecological history

A primigravida. She attained menarche at 14 years. Her cycles were regular 3 – 4 days, every 28 days. She had used oral pills prior to conception.

Family and social history

She was married. Worked as a secretary. Her husband was a pastor. She did not smoke cigarettes or take alcohol. There was no history of chronic illness in the family.

Physical examination

She was in good general condition not pale, not jaundiced and was a febrile. Her vital signs were all normal.

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

Abdominal examination

The abdomen was uniformly distended and moving with respiration. The foetal height was term, longitudinal lie, cephalic presentation and the head was 4/5 above the pelvic born. The foetal heart rate was 144 beats /min regular. Mild contractions were noted.

Pelvic examination

The external genitalia was normal. The cervix was 4 cm dilated, about 80% effaced soft and central. She was draining clear liquor. There was no caput or moulding of the foetal head. The pelvis was adequate clinically.

Management

She was explained to the findings and the plan of management.

On vaginal examination 4 hours later her cervix was 8 cm dilated. An hour later she felt the urge to push. She had a spontaneous vertex delivery to a live male infant, birth weight of 3300gms. He scored 8/1 and 10/5. The placenta and membranes were delivered complete by controlled cord traction. The estimated blood loss was 250 ml.

The baby's cord blood was taken for blood grouping, direct coombs test, bilirubin levels and haemoglobin estimation. The baby was taken to the newborn unit for observation. The direct combs test was negative, the haemoglobin was within normal and the blood group was B rhesus positive. The mother received 300 mg of anti D, the following day. The mother and baby were discharged home to be seen in the postnatal clinic after 6 weeks.

Postpartum follow up

When she came for review after 6 weeks she had no complaints. She was not pale and her general condition was good. The uterus had involuted well. Advice on family planning was given. She chose to use the Depo-Provera and she was discharged through the family planning clinic.

Discussion

The patient presented was a primigravida who was rhesus negative. She was unsensitised and delivered a live baby.

Over 400 red cells antigens have been identified. Individuals who lack a specific red cell antigen may produce antibodies when exposed to that antigen. The rhesus (RH) blood group is the most complex human blood group. The Rh antigens include Dd, Cc and Ee. The antigen D is of great clinical importance. If a woman without the Rh antigen (Rh-negative) carries a rhesus positive foetus, if foetal red blood cells cross to the maternal circulation, then she produces antibodies. This is referred to as isoimmunization or sensitisation. Haemolytic disease of the newborn may then occur. Isoimmunization can also occur following transfusion with incompatible blood.[1,2] B M was a rhesus negative mother who carried a rhesus positive baby but by the time of delivery sensitisation had not occurred.

The incidence of rhesus negativity varies with races. It is highest among the Caucasian population (15-16%), low in Africans (4%) and non-existent in mongoloids.[1] In Nairobi the incidence was 5% of the mothers attending antenatal clinics and at KNH it was 4.1%[3,4]

The overall risk if isommunization for a RH-positive ABO incompatible infant with a Rh-negative mother is about 16%. 1.5-2% of the reactions occur antepartum, 7% within 6 months of delivery and 7% early in the second pregnancy.[1,2]

Factors that encourage foetal maternal haemorrhage increase the risk of isoimmunization. They include abortions, amniocentesis, abdominal trauma, placenta praevia, abruptio placentae, foetal death and caesarean section. Our patient did not have any of these factors. As little as 0.1 ml of Rh-positive cells can cause sensitisation. This amount occurs in less than half of pregnancies with delivery. Of rhesus negative persons 30% are non- responders and will not get sensitised even when exposed to rhesus positive cells.[1]

Haemolytic disease of the newborn occurs when maternal antibodies destroy the Rh-positive foetal red cells. On exposure to Rh-positive cells, the Rhesus negative maternal immune response produces low levels of immunoglobin M (Ig M). Within 6 weeks to 6 months Ig G antibodies become detectable. It is the 1g G antibodies that cross the placenta to attack the foetal red cells. Since 1g G tend to be formed later in the sensitisation process, most first borns are not affected. The absorbed antibodies act as haemolysins leading to accelerated red cell destruction. The excessive or prolonged haemolysis stimulates extramedullary haematopoiesis especially in the liver and spleen. This leads to hepatic dysfunction.[5]

Destruction of the red cells results in release of haem, which is converted to bilirubin. Both haem and bilirubin are neurotoxic but the placenta and maternal liver metabolise them while the foetus is in utero. Soon after birth kernicterus may occur since the foetal liver is not mature enough to clear all the bilirubin in the foetal body. When the destruction of the foetal red blood cells exceeds the production, the resulting severe anaemia results in erythroblastosis foetalis. This condition is characterised by extramedullary haematopoiesis, heart failure, oedema, ascites and pericardial effusion.

All pregnant women should be tested the ABO blood groups and rhesus during their first antenatal visit. For those found to be rhesus negative an indirect combs test should be done. The paternal blood group may be useful since if both parents are rhesus negative, their offspring will also be rhesus negative. At 28 weeks the indirect coombs test is repeated. If negative, 300μg of Rh immune globulin G (the anti D) prophylaxis is given. Without it 1.8% of those delivering Rh positive babies will be immunised as a consequence of spontaneous silent foetal maternal haemorrhages remote from delivery.[6] Our patient had a negative indirect coombs test at 28 weeks and anti D was given. At 35 weeks another indirect coombs test should be repeated, if negative observations continue, if positive the patient is managed as Rh sensitised. If uterine bleeding occurs or amniocentesis is performed more than 3 weeks after the prophylaxis but before delivery the anti D should be repeated.

After delivery, the baby's blood group is done. If Rh positive, the mother receives another dose of anti D, if the baby is Rh negative the anti D is not necessary. Anti D

immune globulin given to unsensitised D-negative women within 72 hours of delivery is highly protective. This prophylaxis should also be given to Rh negative mothers after miscarriage, abortion, molar pregnancy or ectopic pregnancy.[7,12] Perinatal deaths from haemolytic disease have decreased dramatically because of administration of D immune globulin.[8] For the rhesus positive baby, the serum bilirubin levels are estimated and a direct coombs test is done to determine if sensitisation has occurred. Our patient delivered a Rh-positive baby, the direct coombs test was negative. The bilirubin levels were within normal. The mother received 300µg of anti D a day after delivery.

For sensitised mothers close monitoring with ultrasound and amniotic fluid studies is needed. Ultrasound is performed at 14 – 16 weeks to search for signs of ascites and oedema. Amniocentesis is done at 18 –22 weeks. Amniotic fluid is analysed by spectrophotometry, the amount of light absorbed by the blood breakdown products is plotted versus the gestation age. The severity of the affliction is approximated from the Liley graph and is used to guide the management. The graph is divided into 3 zones. The mildly affected foetus falls in zone 1. Amniocentesis should be repeated every 2 – 3 weeks. Delivery should be done when the foetus achieves pulmonary maturity. Moderately affected foetuses fall in zone 2. Amniocentesis should be repeated every 1 – 2 weeks. Delivery usually will be before term as soon as pulmonary maturity is achieved. Severely affected foetus will fall into zone 3. Interventions such as intrauterine transfusion will be necessary to facilitate the foetus to reach a gestational where delivery can be done with minimal adverse effects of prematurity. If no treatment is given to sensitised negative mothers carrying Rh-positive foetus, the prenatal mortality is about 30 %. With treatment the mortality is lowered remarkably.[7,9] In carefully monitored cases, labour can be induced as soon as the pulmonary maturity is reached. In the severely compromised foetus a caesarean delivery is beneficial.[10] In our set up facilities for aggressive management of sensitised mothers are limited. One series reported the rate of sensitised women in KNH to be 600 per 1000 deliveries.[11] The unsensitised mother should not be allowed to go beyond term to reduce the risk of prenatal foetal maternal immunisation. Our patient went into spontaneous labour at 40 weeks on the day she had been scheduled for induction of labour.

References

1. Pernoil M L, Late pregnancy complications in Current Obstetric & Gynecological Diagnosis and Treatment. 8th Edition 1994, 15: 339-342.
2. Bowman JM: controversies in Rh prophylaxis who needs Rh immune globulin and when should it be given? AM J Obsetet Gynaecol 151; 289, 1985
3. Mati J K G Aggarwal UP Sanghvi HCG et al Nairobi birth survey: Antenatal care in Nairobi. Journ of Obs/Gynae east and cent Afri 2 (1): 1, 1983
4. Mulandi in a 2 year study to show the effectiveness of anti – D gammaglobulin in preventing Rhesus Iso-immunization in pregnant women in KNH. Mmed thesis UON, 1985
5. Collin U, Nicolaides P. Tannirandorn Y et al, Foetal liver dysfunction in Rh alloimmunization Bi J Obstet Gynaecol 98: 287, 1991
6. Bowman J.M, Pollock J M, Antenatal Rh prophylaxis 28-week gestation service programme. Can Med ass J 118:622, 1978
7. Freda V; Haemolytic disease, Clin Obstet Gynaecol 16:72, 1973
8. Fretts R,C Boyd M E, et al ; The changing pattern of foetal death, 1961 – 1988 obstet Gynaecol 79:35, 1992
9. Harman C R, Manning F A, et al severe Rh disease – poor outcome is not inevitable. A M J obstet Gynaecol145; 823, 1983
10. Freda V J, Gorman J G, Pollack W, Prevention of Haemolytic: disease Ten years clinical experience with Rh immune globulin N Engl J Med 292: 1014 1975.
11. Kagia JW; Review of the management of Rhesus negative women at Kenyatta National Hospital 1975-1980, MMed Thesis UON 1980.
12. Clinical Guidelines; The Royal College of obstetricians and gynaecologists. Revised May 2002.

OBSTETRIC CASE 6

POST PURTUM HAEMORRHAGE DUE TO CERVICAL TEAR – SUCCESFUL REPAIR

Ip No.	438350	Name	M J
Age	34	D.O.A.	20.1.04
Parity	3 + 0	D.O.D	23.1.04

Presenting complain

The patient was noted to have significant bleeding soon after a spontaneous vertex delivery.

History of presenting complain

She had been admitted in established labour at 39 weeks gestation. The labour progressed well and had a spontaneous vertex delivery to a live male infant who weighed 3.8 kg and scored 8/1, 10/5. The placenta was then delivered complete with the membranes. Despite a well contracted uterus. She continued to have significant postpartum haemorrhage.

History of the current pregnancy

Her last monthly period was on 22.4.03 and her expected date of delivery was 28.1.04. So she was at 39 weeks gestation at the time of delivery.

She had attended antenatal clinic at our hospital and had been uneventful. The antenatal profile was as follows: HB – 12.5 g/dl Blood group – O positive

VDRL – negative ELISA for HIV – negative

Obstetric and gynaecological history

She attained menarche at 13 years. Her menstrual cycle was regular every 27 – 29 days and the flow was normal for 2 – 4 days. She experienced no dysmenorrhoea. She had used oral contraceptives between her pregnancies.

She had 3 previous deliveries all being spontaneous vertex deliveries and the children were well. Her last delivery was 2 years ago.

Past medical history

This was not significant

Family and social history

She was married, an engineer at the Kenya power and lighting company. She did not smoke cigarettes or take alcohol. There was no history of chronic illness in her family.

Physical examination

She was in stable general condition. She was not pale, cyanosed or jaundiced. Her vital signs were: BP – 110 /70 mmhg Pulse Rate 90/min

Respiratory Rate – 22/min Temp 36.6°C

The cardiovascular, respiratory and the central; nervous systems were within normal.

Abdominal examination

The abdomen was moving with respiration. There was some uniform suprapubic fullness. The uterus was well contracted corresponding to 20 weeks gestation. There was no tenderness.

Pelvic examination

The external genitalia was normal but blood stained. The perineum was intact. A sterile speculum examination revealed a cervical tear at the 7.00 o clock position with active bleeding.

Diagnosis

An impression of cervical tear was made

Management

An intravenous infusion of normal saline was started. Blood was taken for grouping and cross matching and packed cell volume estimation. The patient was explained to the condition and the need for immediate repair under anaesthesia. A written consent was obtained. She was premeditated with atropine 0.6mg intramuscularly and shaved the pubic hair. Intravenous augmentin 1.2g was given. She was taken to the theatre.

In theatre, she was put under general anaesthesia. In the lithotomy position, vulvovaginal toilet was done. Aseptic catheterisation was done and clear urine was drained. Using a Sims speculum and vaginal retractors, the cervix was well exposed. On inspection an obvious tear was seen at 7.00 o clock position. It was bleeding slightly but actively. The two margins of the tear were held using sponge holding forceps and down traction exerted to expose the apex of the tear. Using vicryl number 2 – 0 on a round needle, the first stitch was applied, just above the apex. Running stitches were used and the whole length of the tear repaired. Bleeding from the site stopped. The vaginal walls were confirmed to be intact as well as the perineum. Anaesthesia was reversed successfully. She remained stable and did not need blood transfusion. She received 2 more doses of intravenous augmentin 1.2g 8 hours apart as antimicrobial prophylaxis.

On the second post-partum day both the mother and the baby were allowed home.

Follow up

She came for review after 6 weeks, she did not have any complaints. The baby was breastfeeding well. Examination revealed normal vital signs, breasts were bilaterally soft and active. The uterus had involuted. The external genitalia was normal and on digital examination, the cervix felt normal. Advice on family planning was given and she was booked to continue in the family planning clinic.

Discussion

The patient presented had a spontaneous vertex delivery but was noted to have post partum haemorrhage after the delivery of the placenta. Examination revealed a cervical tear that was successfully repaired.

Post partum haemorrhage (PPH) refers to blood loss in excess of 500 ml after a vaginal delivery. The incidence is 5 – 8 % of vaginal deliveries.[1] It is among the leading causes of maternal mortality in the developing world. At KNH, Makokha found 15% of the maternal deaths to have resulted from post-partum haemorrhage. [2] Even in the USA PPH is an important cause of maternal mortality being the third leading cause.[3]

The cause of the haemorrhage should be established quickly to allow definitive treatment. Uterine atony, various degrees of retained placenta and genitalia tract trauma are the leading causes of PPH. The patient presented was found to have a cervical tear. Genital trauma accounts for up to 20% of primary post-partum haemorrhage.[1]

Cervical lacerations occur in over half of vaginal deliveries. Most cervical tears are less than 0.5 cm and heal spontaneously. Deep cervical tears may extend to the upper vagina or involve the lower uterine segment or even the peritoneum. Laparotomy for repair may be necessary in some cases. Rarely the cervix may be entirely avulsed. Such serious injuries often follow difficult forceps deliveries or deliveries through an incompletely dilated cervix or precipitate labour. But lacerations up to 2 cm do occur during normal deliveries. The extensive tears or those bleeding actively need repair. Repair under anaesthesia is recommended to allow good exposure, relaxation and for examination to rule out other lacerations elsewhere. [4,5] The patient discussed had an apparently normal delivery but sustained a cervical tear that required repair.

Cervical tear should be suspected in case of profuse haemorrhage during or after the third stage labour especially if the uterus is well contracted. Adequate exposure and inspection of the cervix is required to confirm the diagnosis. [6] M J had a well uterus but continued bleeding. A speculum done revealed an actively bleeding cervical tear.

Repair requires good exposure. A Sims speculum and right-angled vaginal retractors are helpful. The bleeding usually comes from the angel (apex) of the tear and the first stitch should be slightly above this to ensure good homeostasis. Either an interrupted or continuous absorbable sutures can be used. [6] Overzealous stitching may lead to cervical stenosis while neglected tears can lead to cervical incompetence.[7] M J had successful repair under general anaesthesia.

Repair requires good exposure. A Sims speculum and right-angled vaginal retractors are helpful. The bleeding usually comes from the angel (apex) of the tear and the first stitch should be slightly above this to ensure good homeostasis. Either an interrupted or continuous absorbable sutures can be used. [6] Overzealous stitching may lead to cervical stenosis while neglected tears can lead to cervical incompetence.[7] M J had successful repair under general anaesthesia.

References

1. Sabrina DC, Kapernick PS; Postpartum haemorrhage and the abnormal puerperium in current Obstetric and Gynaecological diagnosis and management. Appleton and lange, 8th edition 1994, 20: 574-575
2. Makokha A E; Maternal mortality at Kenyatta National Hospital between 1977 – 1979 EA Med J 1980; 57: 451
3. Atrash HK et al Maternal mortality in the United States, 1979-1986. Obstet gynecol 1990; 76 : 1055.
4. Fahmy K, Sammour M, Nosair M, Salem A; Postpartum colposcopy of the cervix: Injury and healing. Int J Gynaecol Obstet 34:133, 1991
5. Liaberman B A repair to injury of the genital tract in clinical obstetrics and gynaecology 1980; 7; 621
6. Barbara K, Gomez K: Basic Maternal and Newborn Care: A guide for skilled providers; JHPIEGO, 2004: 4-36
7. Cunninghum FG, Leverno KJ at al Obstetrical haemorrhage in Williams obstetrics, McGraw Hill , 21st edition 2003, 25: 635-645

OBSTERIC CASE 7

PROLONGED PREGNANCY – SUCCESSFUL INDUCTION

Ip No.	0843042	D.O.A.	15.3.04
Name	J M	D.O.D	18.3.04
Age	28	Parity	1 + 0

Presenting complain

The patient was admitted from the antenatal clinic for induction of labour. She had no complains.

History of current pregnancy

Her last monthly period was on 29.5.03 and the expected date of delivery was 5. 3.04. She was at the gestation of 41 weeks and 3 days. She attended clinic in KNH since 20 weeks gestation. The antenatal period was uneventful though she was treated for vaginal candidiasis once. The antenatal profile was as follows.

HB – 2.1 g/dl	VDRL – negative
Blood group – A +ve	Elisa for HIV – negative

She had not had an ultra sound evaluation but clinically the fundal height corresponded to the dates. Her quickening was on 30.09.04

Obstetric and Gynaecological history

She was a para 1 + 0. Her last delivery was in 2000 to a female who was alive and well. It was a spontaneous vertex delivery at 39 weeks gestation. She attained menarche at 14 years. Her cycle was regular every 28 – 30 days with a normal flow of 3 – 4 days. She was not on any contraception prior to the pregnancy.

Past medical history

This was not significant

Family and social history

She was a housewife married to a teacher. She did not take alcohol or smoke cigarettes. She had no history of chronic illness in her family.

Examination

She was in good general condition, not pale or jaundiced .s he had no lymphadenopathy or oedema. Her vital signs were: BP – 100 / 65 mmHg, PR – 82/min PR 22/min and temp 36.6oC

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

Abdominal examination

The abdomen was uniformly distended with fundal height at term gestation. The foetus was in longitudinal lie, cephalic presentation and the head was 4/5 above the pelvic brim. There were no contractions palpable. The foetal heart rate was 144/min regular.

Pelvic examination

The external genitalia was normal. The cervix was 2 cm long, soft central and the os was closed clinically the pelvis was adequate.

Diagnosis

A diagnosis of post date pregnancy was made

Management

She was admitted to the ward and a prostaglandin E 2 pessary was inserted into the posterior fornix. She was reviewed after 8 hours and the cervix was soft, effaced and 2 cm dilated. Artificial rapture of membrane was done, clear liquor drained. A syntocinon infusion of 5 units in 500 ml in 5% dextrose was started at 10 drops per minute. The infusion rate was increased at 10 drops per minute until she had 3 strong contractions every 10 minutes. After 4 hours she was reviewed and the cervix was 6 cm dilated. 3 hours later she had an urge to push. On examination the cervix was fully dilated. She was taken to the second stage room where she had a spontaneous vertex delivery to a live male infant. The birth weight was 3300gms and score 8/1 and 10/5.

the baby did not have features of post maturity. The placenta was delivered by controlled cord traction. The estimated blood loss was 250 ml.

The mother and the baby remained stable and were transferred to the postnatal ward. They were then discharged home to be seen in the postnatal clinic after six weeks.

Follow up

She came for review after 6 weeks. She had no complains. Her breasts were active and soft. The uterus had involuted. She was advised on family planning and was discharged through the family planning clinic.

the baby did not have features of post maturity. The placenta was delivered by controlled cord traction. The estimated blood loss was 250 ml.

The mother and the baby remained stable and were transferred to the postnatal ward. They were then discharged home to be seen in the postnatal clinic after six weeks.

Follow up

She came for review after 6 weeks. She had no complains. Her breasts were active and soft. The uterus had involuted. She was advised on family planning and was discharged through the family planning clinic.

Discussion

The patient presented had a prolonged pregnancy. Induction of labour was successfully done and she delivered a healthy baby.

Prolonged pregnancy is defined as a pregnancy that is 42 completed weeks (294 days) or more from the first day of the last menstrual cycle. The incidence varies but an average is about 10%.[1] In Pumwani maternity hospital an incidence of 4.9 % was reported.[2] Some of these pregnancies are not actually prolonged but appear so due to wrong date estimation.

The cause of prolonged pregnancy is unknown, but some conditions have been associated. It has been shown to have a tendency to recur in the same women and to run in families. Conditions associated with low oestrogen levels in pregnancy have been noted to increase the incidence. Such include foetal adrenal hypoplasia, anencephaly, absence of foetal pituitary gland and placental sulphatase deficiency. Extra uterine pregnancy also tends to be prolonged. [1,3] J M did not have any of these risk factors.

For the diagnosis to be made, it is important to confirm the gestational age. The last menstrual period may be difficult to establish in women who were on hormonal contraception due to irregularities of bleeding caused by their withdrawal. The patient discussed was not on such contraception. Other factors can be used in establishing the gestational age. A pregnancy test done in early pregnancy (during the first 6 weeks of the last period) can be used. If 36 weeks have elapsed since then the baby is mature. Quickening in keen mothers is a helpful event. In primigravida it occurs at 18 – 20 weeks while with multiparous it occurs at 16 – 18 weeks gestation. Foetal heart tones can be detected with a fetoscope at 17 – 19 weeks. If 20 weeks of foetal heart tones have been recorded then maturity is sure. Ultrasound dating can be used. It is important to note the accuracy of ultrasound dating varies with gestation. For crown rump length for example is ± 1 week when the ultra sound is performed at 12 – 20 weeks gestation, at 20 – 30 weeks its ±2 weeks and after 30 weeks its ±3 weeks. Fundal height estimation clinically, between 18 – 30 weeks is correspondent when

bladder is empty. [1,4,5] The patient discussed had attended the antenatal clinic since 20 weeks gestation. She did not have an ultra sound evaluation or a pregnancy test early in the pregnancy. Her date of quickening, and clinical fundal height estimation during the clinic visits confirmed maturity of her pregnancy.

Prolonged pregnancies are associated with certain complications. With placental ageing, utero-placental insufficiency may set in leading to growth restriction. However many post-term foetus continue to gain weight. This could lead to complications of macrosomia such as cephalopelvic disproportion and shoulder dystocia during delivery.[1,4,5] Our patient did not develop these complications.

Post-mature infants have wrinkled patchy peeling skin with a long thin wasted body. Many have higher morbidity due to birth asphyxia and meconium aspiration. Higher prenatal mortality has been reported to be increase. [6] Oligohydramnios associated with post term pregnancies increases the risk of morbidity. There is more risk of cord compression. Foetal release of meconium into already reduced amniotic fluid leads to more thick and viscous meconium thus increasing the risk of meconium aspiration syndrome.[7] J M delivered a baby who did not have any of these features.

The management of prolonged pregnancy is controversial. Due to the complications associated with prolonged pregnancy many authorities agree that well dated pregnancies should not be allowed to progress beyond 42 weeks. [4] There is data to suggest that routine induction after 41 weeks gestation is associated with reduction in prenatal mortality with no increase in the rate of instrumentation or caesarean deliveries. [7] Induction of labour was feared to result in increased operative deliveries without preventing prenatal death. Some clinicians prefer to employ foetal surveillance instead of induction. An amniotic fluid index of more than 8 cm and a reactive foetal heart rate tracing are reassuring. [6,7] This foetal surveillance is expensive and may not be readily available in our set up. In our hospital induction of labour is done once a pregnancy is ascertained to be at 41 weeks and 3 days or more. The patient presented was at 41 weeks and 3 days. She was induced successfully with good outcome.

Once the decision to deliver is made the date of delivery depend on the individual case. If the cervix is unfavourable, ripening is done to reduce the chance of failed induction. Prostaglandin preparations are available for this purpose. When the cervix is favourable amniotomy is done and labour augmented with oxytocin agents. Once induction has begun, close intrapartum monitoring is necessary to recognise and deal with any complications. Such may include presence of meconium, macrosomia and foetal intolerance to labour.

Our patient responded well to the prostaglandin pessaries for cervical ripening and augmentation with syntocinon. She had a normal delivery to a healthy infant.

References

1. Cunninghan F.G, Norman FG, Leverno K S et al; Post term pregnancy in Williams Obstetrics Mc Graw Hill 21st edition. 2001, Pg 730-741
2. Elfenesh B. Post Date pregnancy and foetal out come. Mmed Thesis, UON 1998
3. Phelan J P Myoung O A, Eden RE et al; Post datism, clinical obstetrics and gynaecology 1989; 32 (2)l
4. Paul T Walkes, Post Date Pregnancy, E medicine, Aug 8 2002
5. Pernoll M L, Late pregnancy complications in Current Obstetric & Gynecological Diagnosis and Treatment. 8th Edition, 15; 338-339
6. Mannino F; Neonatal complications of post term gestation. J reprod med 1988 Mar; 33 (3) 27 1 – 6
7. Leverno K J Quirk J G Cunningham F G et al prolonged pregnancy, observations concerning the causes of foetal distress. AM J Obstet Gynaecology 150: 465, 1984
8. Crowley P. Interventions for preventing or improving outcome of delivery at or beyond term. Cochran database syst rev 2000; (2) CD 000170

OBSTETRIC CASE 8

MULTIPLE GESTATION- LIVE BABIES

Name	L W	Parity	1 + 0
Ip No.	0984325	D.O.A.	09.12.04
Age	25	D.O.D	12.12.04

Presenting Complains

LW was admitted through casualty with complains of low abdominal pain and backache for 3 hours.

History Of Presenting Illness

The patient had developed low abdominal pain that morning. The pain was intermittent and radiated to the back. It was increasing in intensity and frequency. There was no associated drainage of liquor but she had seen show.

History Of Current Pregnancy

Her last menstrual period was on 24.03.03 and her expected date of delivery was 31.12.04. She was therefore at 37 weeks by dates at admission. She was attending antenatal clinic in a city council clinic since 24 weeks of gestation. The antenatal profile was as follows.

HB – 11.2 g/dl Blood Group – B +Ve

VDRL – negative ELISA for HIV – negative

During the antenatal period the fundal height was noted to be higher than dates. An ultra sound was not done but she was advised to deliver in Kenyatta National Hospital (KNH). She did not book clinic in KNH but turned up in labour.

She reported to have experienced excessive vomiting in the first trimester though she was never admitted. She also more pressure symptoms than the earlier pregnancy.

Obstetric and Gynaecological History

She was para 1+0. Her last delivery was in 2001 to live female infant at term who weighed 3300 gm. The baby was alive and well. She attained menarche at 15 years. Her cycle was regular, lasting 3-4 days in a cycle of 28-30 days, normal flow with mild dysmenorrhoea. She had never used contraceptives.

Past Medical History

She did not have any chronic illness or allergies.

Family and Social History

A housewife was living with her husband. Her mother had delivered twins. Her aunt was diabetic but no other chronic illness in the family. She did not consume alcohol or smoke cigarettes.

Physical Examination

She was in good general condition. She was not pale and had no oedema or lymphadenopathy. Her vital signs were BP – 110/70mhg PR – 84/min PR-22/min temp – 36.8oC

Her respiratory, cardiovascular and central nervous systems were normal.

Abdominal Examination

The abdomen was uniformly and massively distended. The fundal height was corresponded term gestation. Multiple foetal parts were palpated and 2 heads felt in different quadrants. The lie for both twins was longitudinal with the first twin in cephalic presentation and the second twin in breech presentation. Two distinct foetal tones were heard one at 132 beats per min and the other at 144 beats per minute. She had moderate contractions 3-4 in 10 minutes

Pelvic Examination

Her external genitalia was normal. The cervix was 4 cm dilated, soft and effaced. The first twin was confirmed to be in cephalic presentation. The pelvis was clinically adequate.

Diagnosis

A diagnosis of twin gestation in labour was made.

Management

The patient was explained to the findings and the possibility of that she had a twin gestation. Since she was at 37 weeks gestation, the first twin was in cephalic presentation and the pelvis was adequate, labour was allowed to proceed normally. A dextrose infusion was set up.

Vaginal examination was done after 4 hrs. She was found to be 9 cm dilated the head had descended. Soon after she felt the urge to push and vaginal examination confirmed she was fully dilated. She had a spontaneous vertex delivery to a live male infant, who weighed 2100 gm and the APGAR score was 9 at 1 and 10 at 5. She continued to have strong contractions. The second twin engaged and artificial rupture of membranes was done. The second twin was then delivered by spontaneous vertex delivery, a live female infant who weighed 2250 gm and scored 8 at 1 and 10 at 5. the placenta was delivered by controlled cord traction. On observation the placentas were confirmed to be 2 separate ones. Both were normal and complete with membranes. The uterus contracted well. The estimated blood loss was 400 ml. A paediatrician

Discussion

The patient discussed was a para 1+ 0 who came to Kenyatta hospital in labour and was found to be having twin gestation. She progressed well and delivered both twins vaginally.

Multiple gestation refers to the occurrence of more than one foetus in the uterus at the same time. Two foetuses are referred to as twins, three foetuses triplets, four foetuses quadruplets and so forth. Twin foetuses result from fertilisation of two separate ova (dizygotic twins) or from a single fertilized ovum dividing (monozygotic twins). Either or both process are involved in the formation of higher number of foetuses.[1] LW had a twin gestation

The rate of naturally occurring monozygotic twins is remarkably constant occurring in about 2-3-4 per 1000 pregnancies in all races. It is not influenced by heredity, age of the mother or parity.[2] However there is evidence now to suggest that the division of the fertilized ovum may occur as a result of delayed developmental events. Delayed transport through the tube increases the risk of twinning. Contraceptives containing progesterone lead to delayed tubal transport of the ovum and are believed to increase the risk of twinning in pregnancies conceived close to contraception use.[3] It is also suggested that minor trauma to the blastocyst during assisted reproduction techniques leads to the increased incidence of monozygote twins.[4] About 30% of twins are monozygote. Our patient conceived spontaneously and was not on any contraception prior, her twins were dizygotic.

Nearly 70% of twins are dizygote. Race, heredity, maternal age, parity and especially fertility drugs influence the incidence of dizygotic twins remarkably. Dizygotic twins are most common in blacks, least common in Asians and of intermediate occurrence in whites. In Japan the rate has been found to be 1 in 155 births, while in Nigeria it was as high as 1 in 20 births.[5] In Kenyatta National Hospital, the rate of twinning was found to be 1 in 50 – 60 births.[6,7] Dizygotic twin pregnancies tend to recur; a woman who has had one has a 10-fold chance of another. It may be influenced by a recessive autosomal trait carried by the female descendants. Aging of the mother tends to increase the chance of a dizygotic twin pregnancy with a peak at 35 – 40 years of age. Though young our patient was an African and had history of twinning in her family,

the mother had twins. Most probably her propensity to twinning was inherited. Twining rate is related to nutritional status as reflected by maternal size. Taller heavier women have a higher rate of twinning than the nutritionally deprived.[8] Our patient was in good nutritional status.

The common factor linking race, age and fertility to multiple gestational is higher levels of follicle stimulating hormone levels.[9] Women who conceive within a month of stopping oral contraceptive pills have a higher rate of twinning. This is thought to result from rebound increase of follicle stimulating hormone.[10] Ovulation induction with clomiphene or the human menopausal gonadotrophin has been noted to increase the rate of twinning both dizygotic and monozygotic.[4] The percentage of male conceptuses decreases as the number of foetuses per pregnancy increases.

It is important to make the diagnosis of twin pregnancy and determine the zygosity antenatally. This helps in assessing obstetric risks and guides in the management of multiple gestations. For obstetrical purposes it is important to determine the number of chorions and this can be done in the first trimester, presence of two separate placental sites and a thick dividing membrane more than 2mm supports dichorionicity. Foetuses of different gender suggest dizygotic twins,[10] Ultra sound determination of zygosity has been reported to have 91% sensitivity and specificity. [11] Our patient did not have an ultra sound examination antenatally.

Placental examination after delivery can give further evidence of the zygocity. Foetuses with one common amniotic sac or juxtaposed amnions not separated by chorion are monozygotic. If amnions are separated by a chorion, the foetus could be either monozygotic or dizygotic. Cord blood typing is helpful. Different blood groups confirm dizygosity but similar blood groups do not confirm monozygosity. Definitive diagnosis is done by DNA studies but it is rarely indicated for medical reasons. For our patient the placentas were separate and the babies of different sex confirming dizygosity. [1,2]

Several factors are associated with multiple gestations and may suggest the diagnosis. A high maternal age, high parity, large maternal size and previous history of twins are weak clues. Stronger clues are ovulation induction or assisted reproductive technology. With multiple gestation the uterine size is larger than the expected

gestational age in the second trimester.[12] Between 20 and 30 weeks fundal heights are an average 5cm greater than expected for singletons of the same age. [13] With careful ultrasonic examination separate gestation sacs can be identified in early pregnancy. Clinically multiple gestation may be suspected by palpation of two foetal heads, usually in different quadrants. But it is difficult to identify twins if one overlies the other, if the woman is obese or if there is hydramnios. Different foetal heart sounds asynchronous with the mothers pulse and with each other by at least 8 beats per minute may be heard. Our patient had larger uterine size than dates in the second trimester. The diagnosis was made on admission. Multiple foetal parts were palpated, 2 heads felt in different quadrants and 2 distinct foetal tones were heard.

Generally the degree of maternal physiological changes is greater with multiple gestation. In the first trimester, the nausea and vomiting may be in excess, the physiological anaemia is more pronounced and there is more likelihood of hydramnios.[12] In multiple gestation the urinary chorionic gonadotropin, estriol and pregnanedial levels are elevated. The maternal serum alpha-feto protein levels average twice the median level for singleton pregnancy.[1,2] LW reported to have experienced excessive vomiting in the first trimester. The mother will usually experience earlier and more severe pressure in the pelvis, backache, varicosities, constipation, haemorrhoids, abdominal distension and difficulty in breathing. The foetal activity is greater and more persistent. It is important to distinguish multiple gestation from certain conditions that may present with similar signs or symptoms. Singleton pregnancy with inaccurate dating may appear larger than expected. Polyhydramnios may lead to a bigger uterus than expected. Hydatidiform mole should be considered in an early pregnancy with exaggerated symptoms. Abdominal tumours complicating pregnancy can cause the uterus to be bigger than dates.[8,9] Our patient had marked pressure symptoms in the third trimester.

Multiple pregnancies are associated with several complications. They are associated with increased risks of spontaneous abortions, congenital malformations, low birth weight, restricted foetal growth and preterm deliveries. [14] The degree of growth restriction is likely to be greater in monozygotic twins than in dizygotic twins. Maternal hypertension and placental abruption are higher in multiple gestation.

Perinatal morbidity and mortality is increased significantly in multiple gestations accounting for 12.6% of the perinatal mortality. [15]

There are complications that are unique to multiple foetuses. Monoamniotic twins are prone to intertwining of their umbilical cords, which complicates at least half of the cases. This is a common cause of death.[16] Conjoined twins result from incomplete separation of the embryonic disc. Surgical separation of nearly complete conjoined twins is successful if essential organs are not shared. Our patient had dizygotic twins.

To prevent the complications of multiple pregnancy, it is imperative to accomplish the diagnosis early and to enhance the antenatal care. Nutritional support to supply the increased requirements is important. Iron supplementation, vitamin and folic acid administration and high protein diet are recommended. Limited physical exercise, early work leave and structured maternal education have been advocated to reduce preterm births. [1,2] Early and prompt therapy for any complications should be provided.

Delivery of multiple foetuses may be associated with many complications such as uterine dysfunction, abnormal presentations, umbilical cord prolapse, premature separation of placenta and immediate postpartum haemorrhage. It is therefore important to take certain precautions and make some special arrangements when delivery of two or more foetuses is expected. The woman should be admitted to the hospital as soon as she develops signs of labour, drainage of liquor or per-vaginal bleeding. Labour should be monitored closely and immediate caesarean section done if need arises.[1,2,17]

An intravenous line should be established and at least 2 units of blood made available in case need arise. Rehydration with normal saline or dextrose solution should be done. Vaginal delivery is usually planned for cephalic – cephalic presenting twins. The optimal delivery route for the cephalic – non-cephalic twins is controversial. There are reports that vaginal delivery of first cephalic twin with breech extraction of the second non-cephalic twin resulted insignificantly shorter maternal and neonatal hospital stay than with external version or caesarean delivery of twin B. [1,17]

When the first twin is non-cephalic the usual mode of delivery is elective caesarean section except in the case of very immature twins.[18] As soon as the first twin is

delivered the presenting part of the second twin, its size and its relationship to the birth canal should be quickly ascertained. If the foetal head or breech is fixed to the birth canal, then artificial rupture of membranes should be done. Labour should then be allowed to continue with foetal heart rate monitoring. If contractions do not resume within 10 minutes, dilute oxytocin may be used. If the presenting part is not fixed to the birth canal, then it can be guided into the birth canal by a hand through the vaginal and fundal pressure by an assistant. Internal podallic version may be done and the foetus quickly delivered by breech extraction. Caesarean section should be done for twin B if this second twin is much bigger than the first and is a non-cephalic position or if the cervix promptly contracts and thickness after delivery of the first twin and does not dilate subsequently. Caesarean section is usually done for high order multiple gestations.[1,2,16,18]LW had cephalic - cephalic presentation. Her labour was observed closely and she delivered vaginally to healthy babies. She did not develop any complications in the puerperium.

References

1. Cunningham F G, Grant F N, J et al; Multifoetal pregnancy in Williams obstetric 21st edition 2001, McGraw Hill 765-805.
2. Pernoll L M., Bush M; Multiple pregnancy in current Obstetric & Gynaecologic diagnosis & treatment 9th edition Appleton & Lange 315-325, 2003
3. Bressers W M, E, Ksson A W, et al; Increasing trend in the monozygotic twinning rate, Actagenet Med Gramellol (Roma) 36; 397, 1983
4. SchachterM, Friedler A et al; Monozygotic twinning after assisted reproductive techniques: A phenomenon independent of micromanipulation. Human Reproduction 16 (6): 124-1269, 2001
5. Knox G. Morley D; twinning in Yoruba women J obstetric Gynaecology Br Em p 67; 981, 1960
6. Mutungi A; Prospective study of twin delivery at KNH and PMH; ANC and delivery Mmed Thesis UON 1990.
7. Oyieke J. B. A two and half year review of some aspects of twin pregnancy at KNH Mmed Thesis UON 1990.
8. Nylander PPS; Biosocial aspects of multiple births; J Biosocial sci 3; 29, 1971.
9. Benirschke K, Kim C K; multiple pregnancy N Engl J med 288; 1276, 1973
10. Rothman K J; Foetal loss, twinning and birth weight after oral contraception use. N Engl J Med 297; 468, 1977
11. Scardo J A, Ellings J M, Newman R B; Prospective determination of chrionicity, amnmniocity and zygosity in twin gestations. AM J Obstetric Gynaecology 173; 1376, 1995
12. National guidelines for quality obstetrics and perinatal care, MOH Division of Reproductive Health; Nov 2004
13. Rouse D J Skopec G. J, 2 Latmic F J; fundal height as a predictor of preterm twin delivery obstetric gynaecology 81; 211, 1993
14. Buekens P, Wilcox A; Why do small twins have a lower mortality rate then small singletons? A M J obstetric gynaecology 168; 937, 1993
15. Gardner MO, Goldenberg R L et al; The origin and outcome of preterm twin pregnancies obstetric gynaecology 85; 553, 1991

16. Gilbert WM, Davis S E, Kaplan et al; morbidity associated with prenatal disruption of the dividing membrane in twin gestations. Obstetric gynaecology 78; 623, 1991

17. Mauldin J G, Newman R B, Mauldin PD; cost effective delivery management of the vertex and non vertex twin gestation. AMJ obstetric gynaecology 179; 864, 1998.

18. American college of obstetricians and gynaecologists; special problems of multiple gestation. Education bulletin No. 253, 1998.

OBSTETRIC CASE 9

BREECH PRESENTATION – CAESAREAN SECTION DONE

Ip No.	0924233	D.O.A.	15.5.04
Name	B W	D.O.D	20.5.04
Age	21	Parity	0 + 0

Presenting complain

The patient was admitted through casualty with complaints of labour pain for 4 hours. She had been draining liquor for 2 hours.

History of present pregnancy

She had her last menstrual period on 19.8.03 and her expected date of delivery was 26.5.04. By dates she was 38 weeks and 2 days on admission. She had attended antenatal clinic in Kayole. The antenatal profile done was HB 11.2 g/dl, Blood group B +Ve, VDRL –negative Elisa for HIV – negative. She was noted to have breech presentation and was advised to seek attention in a hospital where a caesarean section could be done. She did not go to any hospital until labour pains set in.

Obstetric and gynaecological history

She attained menarche at 14 years. She had a regular cycle every 25 – 29 days with a normal flow of 2 – 3 days. She had never used contraception.

Past medical history

This was non-contributory.

Family and social history

She was married, a housewife. The husband was a casual labourer. She did not smoke cigarettes or drink alcohol. Her grand mother was hypertensive.

Examination

She was in a stable general condition, not pale or oedematous. She had no lymphadenopathy. Her vital signs were Bp 120/80 mg Hg temp 36.6°C
PR 82/min RR 22/min

Abdominal examination

The abdomen was uniformly distended. The fundal height was term. The foetus was in longitudinal lie, breech presentation. The breech was 4/5 above the pelvic brim. She had moderate contractions, 3 every 10 minutes. The foetal heart rate was 144 B/min

Vaginal examination

She had normal external genitalia. The cervix was 3 cm dilated, soft and central. The presenting part was not well applied to the cervix. She was draining meconium stained liquor grade 2.

Diagnosis

A diagnosis of breech presentation in a primigravida in labour was made.

Management

The need for emergency caesarean section was explained to the patient. She gave an informed consent. Blood was taken for grouping and cross matching and an intravenous infusion of 5% dextrose started. She was shaved the pubic hair and premeditated with 0.5 mg of atropine intramuscularly. In theatre a caesarean section was done and breech extraction performed. The outcome was a live female infant, birth weight of 3400 gm and apgar score of 8/1 and 10/5.

Post operative recovery

The patient remained stable post operatively. She started oral sips and light diet on the first day post operatively. The wound healed well. The baby did well and remained with the mother in the ward. On the 4th postoperative day, she was discharged home to come again to the postnatal clinic after 6 weeks.

Post natal review

She came to the clinic for review. She had no complains. The wound had healed well and the uterus had involuted. The baby was breastfeeding well. She was counselled on contraception and was discharged through the family-planning clinic.

Discussion

B W was a primigravida who presented with breech presentation in labour. An emergency caesarean section was done with good outcome.

Breech presentation occurs when the foetal pelvis or lower extremities engage in the maternal pelvic inlet. Breech presentation occurs in 3-4% of all deliveries. The incidence decreases with advancing gestational age. The incidence is estimated to be 35% before 28 weeks gestation, 25% between 28-32 weeks, 20% between 32-34 weeks, 8% between 34 –36 weeks and 2-3% after 36 weeks gestation.[1] Locally Mati found an incidence of 2.7% in the Nairobi Birth survey while in KNH Njuki found an incidence of 3.5%.[2,3]

There are three types of breeches: [1, 4]

- Frank breech (50-70%) in which the hips are flexed and knees extended. It is also known as the pike position.
- Complete breech (5-10%) in which the hips are flexed and the knees are flexed, known as the cannonball position.
- Footling or incomplete (10-30%) in this type one or both hips extended and foot presenting.

Predisposing factors for breech presentation include prematurity, the lower the gestation the higher the incidence. Uterine abnormalities such as bicornute or septate uterus as well as fibroids have been implicated. Foetal abnormalities such as CNS malformations, neck masses and aneuploidy are risk factors. Multiple gestations are associated with limited uterine space leading the foetuses to align themselves in breech presentation. Other factors include placenta praevia, pelvic tumours, polyhydramnios, and high parity. With previous breech delivery the likely hood of a recurrence is high if the predisposing factor is persistent as in the case of uterine malformations or high parity. [1,4] our patient did not have any of these risks.

The diagnosis of breech presentation is usually made antenatally by abdominal examination. Since the diagnosis error is common ultrasound is useful for confirmation or a pelvic examination for patients in labour. In abdominal examination the softer ill-defined breech is palpated in the lower uterine segment while the hard, globular readily ballotable head is found in the fundus. The foetal heart tones are heard are heard clearly above the umbilicus unlike in cephalic presentation when the foetal heart tones are heard clearly below the umbilicus. When a patient is in labour, during the vaginal examination, the soft irregular breech is easily differentiated from the firm round smooth head. If the breech is complete or frank, the ischial tuberosities, sacrum, anus and/or the external genitals are palpable. The feet may be palpated with the buttocks in complete breech while one or both feet or knee are palpated in footling breech. An emergency ultra sound examination confirms the presentation as well as providing more information. The altitude, number and size of the foetus(es) can be assessed. The location of the placenta and amount of the amniotic fluid can also be determined. Skeletal and soft tissue malformations may be seen on ultra sound. [1,4,5] The patient discussed had the breech presentation diagnosed clinically by abdominal examination antenatally. It was confirmed by pelvic examination in labour.

CT or conventional radiographic pelvimetry may be done. These investigations give information on the foetal presentation, the kind of breech, the degree of the head flexion as well as the pelvic measurements. These investigations expose the foetus to some radiations and are not routinely done. [1,6]

The mode of delivery for breech presentation is controversial. Perinatal mortality is increased 2- to 4-fold with breech presentation, regardless of the mode of delivery. Deaths are most often associated with malformations and prematurity. Abnormalities are observed in 17% of preterm deliveries that have breech presentation and in 9% of term gestations with breech presentation. [1,4]

Different studies have been carried out and reveal that the perinatal mortality, neonatal mortality, or serious neonatal morbidity is significantly lower in the planned caesarean group than in the planned vaginal deliveries. The difference in perinatal outcome was after controlling for the experience level of the obstetrician. On the basis of these studies many authorities hold that planned vaginal delivery of a singleton

term breech may no longer be appropriate. This does not apply to those mothers presenting in advanced labour with a term breech and imminent delivery or to the second twin that is nonvertex. [1,5,7,8] B W was a primigravida she presented with breech presentation in early labour and can emergency caesarean section was done.

If vaginal breech delivery is to be performed some candidates are more suitable than others. Between 32 and 36 weeks' gestation, vaginal breech delivery may be considered. A frank breech presentation is preferred when vaginal delivery is to be attempted. Complete breeches and footling breeches are still candidates as long as the breech infant is well applied to the cervix. The foetus should show no neck hyperextension on ultrasound, flexed or military position is acceptable. The estimated foetal weight should be between 2000 – 3500 gm. There should be no other maternal or foetal indication for caesarean section. Previable foetus or one with documented lethal malformations should be delivered vaginally. Mothers who are at an advanced stage of labour that is progressing normally should be allowed to progress with vaginal delivery. Recommended pelvimetry criteria include that the inlet has a transverse diameter larger than 11.5 cm and an anteroposterior diameter larger than 10.5 cm. The mid pelvis should have a transverse diameter (between ischial spines) larger than 10 cm and an anteroposterior diameter larger than 11.5 cm. [1,4,5,6,9]

For vaginal breech delivery leaving the foetal membranes intact as long as possible to act as a dilating wedge and to prevent overt cord prolapse is advisable. Oxytocin induction and augmentation are controversial. Some studies show successful induction and augmentation using oxytocin. Results from other studies indicate that nonphysiologic forceful contractions could result in an incompletely dilated cervix and an entrapped head. An anaesthesiologist and paediatrician should be present for all vaginal breech deliveries. A paediatrician is needed because of the higher prevalence of neonatal depression and the increased risk for unrecognised foetal anomalies. An anaesthesiologist may be needed if intrapartum complications develop and the patient requires general anaesthesia. An episiotomy should be performed when crowning is evident. This is advocated by many authors for all breech deliveries even in multiparas, to prevent soft tissue dystocia. [7,8]

Three types of vaginal breech deliveries are described, as follows: [1,4,6]

1. Spontaneous breech delivery: No traction or manipulation of the infant is used. This occurs predominantly in very preterm deliveries.
2. Assisted breech delivery: This is the most common type of vaginal breech delivery. The infant is allowed to spontaneously deliver up to the umbilicus, and then manoeuvres are initiated to assist in the delivery of the remainder of the body, arms, and head.
3. Total breech extraction: The foetal feet are grasped, and the entire foetus is extracted. Total breech extraction should be used only for a noncephalic second twin; it should not be used for singleton foetuses because the cervix may not be adequately dilated to allow passage of the foetal head. If the feet prolapse through the vagina, treat expectantly as long as the foetal heart rate is stable to allow the cervix to completely dilate around the breech. Total breech extraction for the singleton breech is associated with a birth injury rate of 25% and a mortality rate of approximately 10%.

Vaginal breech deliveries are associated with lower Apgar scores, especially at 1 minute. An entrapped foetal head may result from an incompletely dilated cervix or from a head that lacked time to mould in the maternal pelvis. This occurs in 0-8.5% of vaginal breech deliveries. This percentage is higher with preterm foetuses (<32 wk), when the head is larger than the body and may become trapped in an incompletely dilated cervix. Nuchal arms are present in 0-5% of vaginal breech deliveries and in 9% of breech extractions. Nuchal arms result in neonatal trauma in 25% of cases. Avoiding rapid extraction of the infant during delivery of the body may reduce risks. Cervical spine injury is predominantly observed when the foetus is noted to have a hyper-extended head prior to delivery. Cord prolapse occurs in 7.5% of all breeches. Of cord prolapses, 0-2% occur with frank breech, 5-10% occur with complete breech, and 10-25% occur with footling breech. Cord prolapse occurs twice as often in multiparas (6%) than in primigravidas (3%). Cord prolapse may not result in severe foetal heart rate decelerations because of the lack of presenting parts compressing the umbilical cord. [1,4,5,6]

Many breech deliveries are now by caesarean section. The point of caesarean delivery is to avoid trauma to the fragile pre-term foetus, and the caesarean delivery should be performed in the least traumatic fashion. An entrapped head can still occur during

caesarean delivery if the uterus contracts downward after delivery of the body, even with an adequate-appearing lower uterine segment. Entrapped heads occur more commonly with pre-term breeches, especially with a low transverse uterine incision. Many authors advocate low vertical uterine incisions for pre-term breeches. Low vertical incisions usually require extension into the corpus, resulting in caesarean delivery for all future deliveries. The membranes should be kept intact as long as possible and once the breech is extracted the head should be delivered quickly before the uterus begins to contract downward. The transverse incision can be extended vertically upward (T incision) if any difficulty occurs with delivery of the foetal head. Alternatively, the transverse incision can be extended laterally while curving upward, avoiding trauma to the uterine arteries.[4,5,9]

External cephalic version (ECV) is an alternative to caesarean section for breech delivery. ECV is the transabdominal manual manipulation of the foetus into a cephalic presentation. ECV should be performed in or near a delivery suite because in the unlikely event of foetal compromise during or following the procedure, emergent delivery may be necessary. One should be prepared for the possibility of caesarean delivery. An ultrasound to confirm breech, check growth and amniotic fluid volume, and rule out anomalies associated with breech is important. Foetal well-being should be confirmed prior to the exercise.[5,10,11]

Following an ECV attempt, whether successful or not, the nonstress test (biophysical profile if needed) is repeated prior to discharge. Rhesus immune globulin should be administered to women who are Rh-negative. Some physicians induce labour following successful ECV, while others discharge the patients and wait for spontaneous labour. Some foetus may revert to breech presentation after successful ECV.[4,5]

Uncommon risks of ECV include fractured bones, ruptured viscera, labour or premature rupture of membranes, abruptio placentae, foetomaternal haemorrhage (0-5%), and cord entanglement (<1.5%). A common risk of ECV is transient slowing of the foetal heart rate (in as many as 40% of patients). This risk is believed to be a vagal response to head compression with ECV. It usually resolves once ECV is discontinued and is not usually associated with adverse sequelae for the fetus.[4,5,10]

ECV is indicated for a term foetus in a mother with no contraindications to vaginal delivery. Absolute contraindications for ECV include multiple gestations with breech

presentation, contraindications to vaginal delivery (eg, placenta praevia), and nonreassuring foetal heart rate tracings. Relative contraindications include polyhydramnios or oligohydramnios, foetal growth restriction, uterine malformation, maternal HIV infection and foetal anomaly. [5,10,11]

References

1. Joseph V C, Malpresentation and Cord Prolase in Current Obstetric & Gynecological Diagnosis and Treatment. 8th Edtion 21:410-421.
2. Mati J K G, Aggarwal V P, Sanghvi H C, Njuki C M, The Nairobi Birth Survey: Labour and delivery J obstet/Gynaecol. East Cent. Afri. 1983:247.
3. Njuki S K Breech presentation at KNH Modes of delivery and outcomes. M med Thesis University of Nairobi 1979.
4. Richard Fischer, Breech presentation, May 10 2004, e medicine
5. Cunningham G R Gant F N et al Breech presentation and delivery in Williams Obstetrics Mc Gray Hill 21st Edition 2001, 22:510-532
6. Ballas S, Toaff R: Hyperextension of the fetal head in breech presentation: radiological evaluation and significance. Br J Obstet Gynaecol 1976 Mar; 83(3): 201-4
7. Hannah ME, Hannah WJ, Hewson SA, et al: Planned caesarean section versus planned vaginal birth for breech presentation at term: a randomised multicentre trial. Term Breech Trial Collaborative Group. Lancet 2000 Oct 21; 356(9239): 1375-83
8. Cheng M, Hannah M, Breech delivery at term; A critical review of the literature. Obstet Gynaecol 82; 605, 1993.
9. Wright RC: Reduction of perinatal mortality and morbidity in breech delivery through routine use of caesarean section. Obstet Gynecol 1959; 14: 758-63.
10. Cook HA: Experience with external cephalic version and selective vaginal breech delivery in private practice. Am J Obstet Gynecol 1993 Jun; 168(6 Pt 1): 1886-9; discussion 1889-90
11. Lau TK, Lo KW, Rogers M: Pregnancy outcome after successful external cephalic version for breech presentation at term. Am J Obstet Gynecol 1997 Jan; 176(1 Pt 1): 218-23

OBSTETRIC CASE 10

HIV IN PREGNANCY - LIVE BABY

Ip No.	0961207	D.O.A.	01.6.04
Name	A M	D.O.D	08.6.04
Age	21	Parity	0 + 0

Presenting complaints

She was admitted from the antenatal clinic for elective caesarean section.

History of pregnancy.

Her last menstrual period was 6.9.04 and her expected date of delivery was 13.6.04. So the gestation by dates was 38 weeks. She had attended the KNH antenatal clinic from 30 weeks gestation. She had made 6 visits. The antenatal period was uneventful except for 2 episodes of vaginal candidiasis for which she was treated. The antenatal profile was; Haemoglobin -11 g/dl ELISA for HIV - positive

VDRL – negative Blood group - O positive

Before ELISA for HIV was done, pre-test counselling had been done and later on, post-test counselling was also done. Among issues discussed was prevention of mother to child transmission and safer sex practices. She was given haematinics and multivitamins. At 34 weeks gestation AZT at the dose of 300mg BD was started. She chose to have an elective caesarean section and breast-feed exclusively for 4 months.

Obstetric and gynaecological history

She attained menarche at 14 years. Her cycle was regular every 28 days with a flow of 3 – 4 days. She did not experience dysmenorrhoea. She had never used any contraception. She had had 4 sexual partners. She did not report having ever suffered from a sexually transmitted infection.

This was her first pregnancy

Past medical history

This was non-contributory

Family and social history

A single lady was cohabiting with her boyfriend but went back to her parents in Dandora when she realised that she was pregnant. She was unemployed. She did not drink alcohol or smoke cigarettes. There was no history if chronic illness in her family.

Physical examination

She was in stable general condition and was not pale or jaundiced. She had no oedema or lymphadenopathy. Blood pressure was 120/70 mmHg, pulse rate of 84/min, respiratory rate of 22/min and temperature was 36.5°C.

Respiratory cardiovascular and the central nervous systems were essentially normal.

Abdominal examination

The abdomen was uniformly distended with fundal height at term. The lie was in longitudinal with a cephalic presentation. The head was $5/5^{th}$ above the pelvic brim. The foetal heart rate was 140 beats / minute. No contractions were palpated.

Vaginal examination

She had normal external genitalia the cervix was closed, soft, long and posterior. No discharge was found on the examining fingers. The patient was admitted and prepared for elective caesarean section.

Investigations

Haemoglobin 11.5 g/dl

W B C – 8.9 x 10g/l

Platelets – 240 x 10g/l

Urea and electrolytes were normal

An informed consent was taken, blood grouped and cross-matched. She was premedicated with 0.6 mg atropine 30 minutes before going to Theatre.

In Theatre she was placed in lithotomy position and vulvovaginal toilet and catheterisation done. In supine position the abdomen was cleaned and draped.

Anaesthesia was administered. The abdomen was opened using a pfannenstiel incision and the uterus opened via lower uterine segment incision. The membranes were not ruptured until the baby's head was partially delivered. This ensured that the mother's blood did not contaminate the baby. The cord was double dumped and ligated. The placenta was delivered and the uterus and the abdomen repaired. The outcome was a female infant birth weight of 3000 grams and apgar score of 8/1 and 10/5. She received crystalline penicillin and gentamycin intravenously for 48 hours, after which she was given amoxil orally. Post operatively the patient did well. The baby received a stat dose of nevirapine and was breast-feed exclusively for 4 months. Both the mother and baby were well and were discharged home on the fourth postoperative day.

Postpartum follow up

She came to the clinic 2 weeks later. Her wound was well healed. Her breasts were active, soft and non-tender. The baby was breast- feeding well. She had no complaints. Advice on contraception and safer sex practices was reinforced. She was booked to come again after one month for further follow up.

Discussion

The patient presented was a 21-year-old primigravida with human immunodeficiency virus infection in pregnancy. She had a caesarean section for prevention of mother to child transmission and was to breast feed exclusively for 4 months.

The acquired immunodeficiency syndrome (AIDS) is marked by profound immunosuppresion principally of cell-mediated immunity. This gives rise to a variety of opportunistic infections and neoplasm's. AIDS is caused by DNA retroviruses referred to as the human immunodeficiency viruses, HIV – 1 and HIV – 2. HIV – 1 is the most prevalent worldwide and HIV – 2 is endemic in West Africa. It is estimated that 40 million people are infected with the virus worldwide. 70% of these are found in the sub-Saharan Africa, where 55% of the infected are women. [1] In Kenya in 2001 it was estimated that the HIV prevalence was 13 % in adults aged 15 – 49 years.[2] The prevalence is reported to have declined and was reported as 9.4% among women participating in the 2003 and 8.3% among women in the Kenya Demographic Health Survey, 2003.[3]

The HIV attacks several cells in the body but the T lymphocytes, CD4 are the principle targets. The virus attaches itself to these cells and is internalised. It then uses reverse transcriptase to transcribe its genome RNA and DNA. The viral DNA is integrated into the cellular DNA. Infection shortens the lifecycle of the cell. This leads to insidious but progressive drop of the CD4 cells eventually leading into profound immuno-suppression.

The incubation period after exposure is usually within days to weeks. The acute viral illness is similar to other viral syndromes lasting less than 10 days. After this the body maintains the antigens at very low levels and the patient remains asymptomatic. The period of remaining asymptomatic varies from a few years to more than 10 years. The early symptoms include lymphadenopathy followed by chronic constitution symptoms such as diarrhoea, oral candidiasis, oral hairy leukoplakia and ophthous ulcers. As the CD4 count reduces further, opportunistic infections such as the pnemocystis carini cryptococcus, toxoplasmosis and kaposis sarcoma set in. A W was asymptomatic.

Several tests can be used to test for the infection. The Enzyme Linked Immunosorbent Assay (ELISA) screen for the antibodies. A positive test can be confirmed with the western blot or immunoflurescence, which also test for antibodies but are more sensitive. These antibody serotesting does not exclude recent infection for the acute HIV infection and the period before antibodies production. Identification of the p 24 core antigen or viral RNA is necessary. Viral cultures can also be seen. [4] These tests are expensive and in our set up the antibody serotesting is used being less costly. The patient presented had a positive ELISA.

The HIV is transmitted principally through unprotected sexual intercourse with an infected person. This accounts for about 90% of transmission. Contaminated blood and blood products and contaminated instruments contribute to less than 1% of transmission. Transmission of mother to child accounts for about 10%, [1,5] A W had been exposed to several sexual partners and most likely got infected through unprotected sex.

The mother to child transmission (MTCT) occurs ante-natally in 5 – 8 % of the exposed, during labour and delivery in 10 – 20 % and during breast- feeding in another 10 – 15% when breast fed for 18 – 24 months [3,5] In Kenya about 50 000 to 60 000 infants are infected with HIV annually due to MTCT. [5] Factors that increase the risk of transmission during pregnancy include high viral load, low CD4 count, HIV infection acquired during pregnancy. In the intrapurm period, rupture of membranes for more than 4 hours and vaginal delivery when compared to elective caesarean section increase the risk of transmission. Post natally there is increased risk with, prematurity, prolonged duration of breastfeeding, mixed feeding and breast disease. [3,5,6,7] Our patient had an elective caesarean section to reduce the risk of transmission to the baby.

All pregnant women should be encouraged to test for HIV infection. Counselling should be done to pass the relevant information to the mothers. Prenatal screening should be universal but voluntarily. In KNH prenatal HIV testing acceptance was found to be 99.4%[8] Those found to have the infection should be offered the anti-retroviral therapy that has been shown to reduce MTCT. [1,9]Several regimes have been

used. In our set up zidovudine (AZT) is used from 34 - 36 weeks till delivery and the baby may get it for one week.

Intrapurtum single dose of nevirapine is given at the onset of labour and to the baby within 72 hours. This lowers the risk of HIV transmission by nearly 50% in a breast feeding population.[10] Other intrapartum interventions include delayed artificial rapture of membranes and avoiding all invasive procedures as far as it is practical. Elective caesarean section has been shown to reduce the risk of MTCT. The benefit is greatest for those with high viral load. [11] The patient presented received AZT antenally and nevirapine before the elective caesarean section.

Breast-feeding is known to transmit the HIV to the infant. Half of this transmission occurs within the first 6 weeks and ¾ by 6 months.[5,7] The mothers should be encouraged to use formula milk and not breast feed at all. However where this is impractical the mother should be encouraged to breastfed exclusively for 4 months then wean the baby [4,12,13] Breast milk may be expressed and boiled just before feeding the baby to get rid of the HIV. [5] Our patient could not afford formula milk she was to breast feed exclusively for 4 months then wean the baby.

In the post partum period, the mothers should be followed for routine care and other specific needs. These needs may include follow up on the mother's health condition, checking on the infant feeding and advice on family planning and safe sex. Our patient was advised on infant feeding, family planning and safe sex.

References

1. Cunningham G. F. Norman F.G leveno K J et al; Sexually transmitted disease in Williams obstetrics 21st edition 2001, McGraw hill publishers, 57: 1498-1504.
2. Clinical guidelines for management of HIV infected pregnant women and prevention at maternal to child transmission Feb: 2001 M O H, Kenya.
3. Kenya Demographic Health Survey 2003, Preliminary Report; CBS, MOH Kenya, KEMRI, CDC.
4. Centre for disease control and prevention: 1998 guidelines for treatment of sexually transmitted diseases. MMMR 47 : 1 1998 b
5. National guidelines, prevention of mother to child HIV/AIDS transmission by ministry of health and NASCOP , second edition 2002
6. Iandersman SH, Kalish etal obstetrical factors and the transmission of HIV – 1 from mother to child N. Engl. J Med. 334: 1617 1996
7. Kiarie J N . The acceptability of prenatal HIV screening. MMED thesis Nov 1996
8. Nduati R. John G. Mbori Ngacha et al effects of breast feeding and formula feeding on transmission of HIV – 1 a randomised clinical trial JAMA 283 : 1167 , 2000
9. Mofenson LM, Lambert SS Stiehmer et al; Risk factors for prenatal transmission of HIV – 1 in women treated with zidovudine . N Engl. S Med. 345 : 385 , 1999
10. Guat L: Musoke P: Fleming I et al Intrapartum and neonatal single dose nevirapine compared with zidovudine for prevention of maternal to child transmission of HIV – 1 lancet 345 (9 181) : 795 – 802 , 1999
11. European mode of delivery collaboration elective caesarean section versus vaginal delivery in prevention of vertical HIV – 1 transmission. A randomised clinical trial lucent 353: 1035, 1999
12. United Nations children fund. The state of the world's children 1998 – breast milk and transmission of HIV– panel 6 –UNICEF home information participation organisation activities. 1998
13. McIntyre J. Transmission of HIV from mother to child, strategies for prevention postgraduate doctor far (1) 4:6 1997

OBSTETRIC CASE 11

CERVICAL INCOMPETENCE MCDONALD STITCH ISERTED – LIVE BABY

Ip No.	0811147	Name	F. N.
Age	33	D.O.A.	23.7.04
Para	1 + 3	D.O.D	29.7.04

Presenting complain

The patient was admitted through the antenatal clinic for cervical cerclage. She had no complaints.

History of present pregnancy

Her last normal menstrual period was on 10.11.03 and the expected date of delivery was 17.8.04 therefore she was at 14 weeks and 3 days gestation. She had attended the antenatal clinic since 10 weeks gestation. In the clinic the need for a McDonald stitch was explained to her since her history was highly suggestive of an incompetent cervix. Antenatal profile was:

Haemogram 13g/dl		Blood group	B rhesus +ve
VDRL	negative	ELISA for HIV	negative

Obstetric and gynaecological history

She attained menarche at 14 yrs of age her menstrual cycle was every 28 – 30 days with a normal flow of 2 – 3 days. She was para 2+2. Her first pregnancy was in 1997. She had a normal delivery to an infant who is alive and well. In 1999 she had a premature delivery at 6 months and the baby died soon after birth. In 2000 she had an abortion at 5 months while in 2003 she had an abortion at 4½ months. She reported to have had spontaneous rapture of membranes followed by brief labour pains and expulsion of the foetus in the last three pregnancies. She had used oral pills for contraception in between her pregnancies.

Past medical history

This was not significant

Family and social history

She was a housewife married to a teacher. She did not take alcohol or smoke cigarettes. There was no chronic illness in the family

Examination

She was in good general condition, not pale or jaundiced. She had no oedema. The vital signs were BP 110/70mmhg PR 82/min Temp 36.

The cardiovascular respiratory and central nervous systems were within normal

Abdominal examination

The abdomen was slightly distended in the suprapubic region. The fundal height corresponded to 14 weeks of gestation. There were no areas of tenderness and no other palpable mass.

Speculum examination

The external genitalia was normal.

The cervix was about 1 cm long and admitted one finger.

Management

An ultrasound was done which confirmed a normal intra uterine gestation at 14 weeks. Her haemogram, serum urea and electrolytes were within normal. An informed consent was taken and patient prepared for Theatre.

In theatre vulvovaginal toilet was done under general anaesthesia and catheterisation was done. Using a speculum the cervix was exposed. The membranes were not bulging and there was no evidence of bleeding or drainage of liquor. A purse string was inserted at the level of internal OS avoiding the cervical canal. The bites were at

1/2, 4/5, 7/8, and 10/11 positions. A silk no 2 suture was used. She did well post operatively and was discharged home.

Follow-up

She then continued the follow-up uneventfully.

At 36 weeks and 3 days, she presented with complains of low abdominal pain which had been for two days. The pain was radiating to the back. There was no associated drainage of liquor or per vaginal bleeding. She was admitted to the labour ward.

Abdominal examination revealed a fundal height of 36 weeks, longitudinal lie, and cephalic presentation. The head was four fifths above the pelvic brim. The foetal heartbeat was 144/min. No contractions were palpated.

Sterile speculum examination was done. The cervix was open with slightly bulging but intact membranes. The McDonald stitch was visualised and was removed.

She then progressed to have established labour within 3 hours. The labour progressed well and she delivered by spontaneous vertex delivery to a live female infant. The birth weight was 2800 gm with an apgar score of 8/1, 10/5 both the mother and the baby did well post natally. They were discharged to be reviewed in the post-natal clinic after six weeks

Discussion

The patient was para 1+3 lady who had had a normal delivery followed by 3 consecutive second trimester abortions. In her fifth pregnancy cervical cerclage was done and she delivered a live baby.

A normal cervix remains firm in pregnancy thus maintaining the foetus in the uterus until the appropriate time for delivery. The function of the cervix during pregnancy depends on the regulations of connective tissue metabolism. Collagen is the principal component in the cervical matrix. In cervical incompetence, painless cervical dilation occurs, followed by bulging of the membranes through the cervical canal. The membranes then rapture and the foetus is expelled. Unless treated this sequence tends to recur with the subsequent pregnancies.[1,2]

Cervical incompetence occurs in 1% of all pregnancies.[3] At KNH the prevalence was reported to be 1 in 90 pregnancies.[4]The cause of cervical incompetence is unknown but several factors are thought to be contributory. Congenital factors include developmental abnormalities associated with abnormal mullerian duct development or exposure to diethylstilbestrol in utero. Connective tissue disorders such as Ehlers-Danlos syndrome have also been implicated. The proportion of muscle in a normal cervix is 10%. A higher concentration of muscle in relation to collagen has been implicated in cervical incompetence. Surgical trauma following conization, forceful dilation and curettage and cauterisation is thought to result to cervical weakness in some patients. Most cases are however idiopathic. [2 5,6,7] The patient discussed had no obvious cause or risk factors.

Several methods of making the diagnosis have been described. A history of repeated painless second trimester or early third trimester pregnancy losses should point to this problem. Outside of pregnancy, hysterography and checking cervical resistance with a dilator or special instrument or an inflated catheter balloon have been tried. [8]

In pregnancy ultrasonic technologies are applied. The trans vaginal ultrasound is more sensitive than the transabdominal. A cervical length of less than 2.5 cm, protrusion of the membranes and funnelling of the cervix are features suggestive of incompetence.[9,10] F N the diagnosis was made from the classic history.

Treatment of cervical incompetence requires surgical reinforcement of the weak cervix by a purse string suture. The procedure should be delayed until 12 – 14 weeks to allow early abortions due to other factors to occur. The optimal period for the

insertion of the stitch is 16-18 weeks of gestation. The later the gestation, the higher the chances of failure and complications. Cerclage is rarely performed after 24-25 weeks. Sonography to confirm foetal viability and to exclude major malformations should be done. Any infection should be treated first. Sexual intercourse should be avoided at least one week before and after the cerclage. In our unit the patients are observed for 48 hours and tocolytics and antibiotics are routine.[2,9] F N had the cerclage done at 14 weeks gestation and she was advised to avoid sexual intercourse for at least one week thereafter. Many investigators have used different antibiotics and tocolytic agents. No clear benefit has been demonstred and these drugs should be used with caution.[11]

The McDonald stitch is the simple procedure involving cerclage of the cervix with a stitch transvaginally. Little bleeding may result. Its success rate is 85 – 90%.[12] At KNH a success rate of 53% for term pregnancy was reported and a survival rate of 64.5%. [4] The shirodkar operation is more complicated and is associated with higher rates of complication, a modification has been done. The modified shirodkar operation is reserved for previous McDonald cerclage failure and cervical structural abnormalities. Tran abdominal cerclage that is placed at the level of uterine isthmus has been recommended in cases of anatomical defects or where transvaginal cerclage has been unsuccessful. [2,13] For our patient had a McDonald stitch was selected.

Contraindications to cerclage include uterine bleeding, uterine contraction, polyhydramnos with known foetal malformations, ruptured membranes and cervical dilatation beyond 4 cm. [2] F N did not have any of these complications.

Complications of the cerclage include infections and membrane rupture. In the event of any of these the stitch should be removed. The complications are less when the procedure is done by the 18th week of gestation. If the stitch is not removed on time, delivery with it in situ can lead to injury to the cervix. The stitch should be removed if labour ensues or after 37 completed weeks. [2,14]

The patient presented did not develop any complications. The stitch was removed at the 37th week when she came complaining of low abdominal pains. Soon after the stitch was removed she had a normal delivery.

References

1. Rock J A, Thomson D T, Management of abortion in Te Linde's Operative gynaecology 8th Edition Lippincott Williams 1997, 23: 481-482
2. Cunningham G R Gant F N et al; Abortion, in Williams Obstetrics Mc Gray Hill 21st Edition 2001, 33: 862-865.
3. Callen; ultrasonography in obstetric gynecology, Sanders 4th Edition 577-596.
4. Njagi S, Radiological diagnosis of cervical incompetence. Mmed Thesis university of Nairobi 1980
5. Abramovici H, Faktor J H, Pascal B; Congenital malformations as indications of cervical cerclage in habitual abortion and premature delivery. Int Fertil 1983,28,161
6. Singer M S, Hochman M, Incompetent cervix in hormone-exposed offspring, Obstet gynecol 1978; 51; 625
7. Rudd N L Nimrod C, Holbrook K A et al Pregnancy complications in type 1V Ehlers-Danlos syndrome Lancet, 1983; 8:50
8. Ansari A H, Renolds RA: Cervical incompetence. A Review ,J Repro Med 32:161.1987
9. Zilianti M et al Ultrasound Med, 1995 14:719-724
10. Guzman ER, Pistowsk DM, VItzileos AM, et al. A comparison of ultrasonagraphically detectable cervical changes in response to transfundal pressure, coughing and standing in predicting cervical incompetence. Am J obstet Gynecol 997; 177:660
11. Cervical Insuffiency; ACOG practice bulletin, No 48, Nov 2003.
12. Caspi E, Schneider DF, et al Cervical internal OS cerclage: Description of a new technique and comparison with shirodkar operation. Am J perinatol 7: 347.190
13. Cammarano CL, Herron MA, Parer JT Validity of indications for transabdominal cervicoisthmic cerclage for cervical incompetence. Am J Obstet Gynecol 172; 187, 1995
14. Charles D, Edward WR, Infections complications of cervical cerclage. Am J Obstet Gynecol 141: 1065 1981

OBSTETRIC CASE 12

PREMATURE RAPTURE OF MEMBRANES --LIVE BABY

Ip No.	0824692	Sex	Female
Name	M W	D.O.A.	5.8.04
Age	32	D.O.D	8.8.04
Parity	2 + 0		

Presenting Complains

The patient presented with drainage of liquor for 9 hours.

History Of Presenting Illness

The patient was admitted through the casualty with history of having drained liquor early that day at 34 weeks 4 days gestation. She reported to have noted bed wetness at around 4.00 am in the night when she woke up to pass urine. Colourless, warm fluid trickled down her legs to the floor when she stood up. There was no history of abdominal trauma, pain or straining prior to the episode. She had not experienced any per vagina discharge or bleeding. Her passage of urine had been normal.

History Of Current Pregnancy

Her last menstrual period was on 7.12.03, her expected date of delivery was 14.9.04 she was therefore at 34 weeks and 4 days at admission. She had attended antenatal clinic in a peripheral clinic since 20 weeks of gestation, antenatal profile done was as follows.

HB – 10.5 g/dl	Blood Group – B +Ve
VDRL – negative	ELISA for HIV – negative

Past Obstetric And Gynaecological History

She attained menarche at 14 years. She had a regular cycle of 25 days with a normal flow of 2 –3 days. She was para 2 + 0. Her first pregnancy was in 1998 at term birth weight of 3200 gm. The second delivery was in 2000 at term birth weight of 3400 gm. Both deliveries were vaginal and both babies were alive and well. She had not used any contraception prior to this pregnancy.

Past Medical History

This was not significant

Family and social history

She was married sold vegetables in the market. She lived in Kawangware with her husband. There was no history of chronic illness in her family. She did not drink alcohol or smoke cigarettes.

Physical Examination

She was in good general condition. She was not pale or jaundiced and had no oedema or lymphadenopathy. Her vital signs were BP – 110/70mhg PR – 84/min PR-22/min temp – 36.8oC

Abdominal Examination

The abdomen was uniformly distended. The fundal height was corresponding to 36 weeks gestation, with a longitudinal lie and cephalic presentation. The head was 4/5 above the pelvic brim. There were no palpable contractions. The foetal heart rate was regular at144 beats per minute.

Pelvic Examination

A sterile speculum exam was done. Her external genitalia was normal. Clear liquid had pooled in the posterior fornix. The cervix was partially open with slightly bulging membranes. There was slow active drainage of the liquor from the OS. A digital examination was done which confirmed the cervix to be 3 cm dilated. It was soft and central, about 70 % effaced.

Diagnosis

A diagnosis of preterm premature of membranes at 34 weeks gestation was made.

Management

The patient was admitted into the labour ward. A decision to deliver her was made since she was draining liquor actively and was at 34 weeks and 4 days gestation. An intravenous infusion of 5 units of syntocinon in 500 ml of 5% dextrose was started at

10 drops per minute. The flow rate was increased by 10 drops per minute every half hour until she attained 3 contractions in 10 minutes, each lasting 40 seconds or more. She was also started on intravenous augmentin 1.2 gm 8 hourly. An examination done 4 hours later revealed a cervical dilation of 7 cm. 2 hours later she had the urge to bear down and was found to be fully dilated. She had a spontaneous vertex delivery to a live female infant who weighed 2200gms and score 8/1 and 10/5. The baby was stable and joined the mother in the postnatal ward. On the 2nd postnatal day she was discharged on oral antibiotics to come for review in the postnatal clinic after 6 weeks.

Follow up

The patient did not turn up for postpartum follow up.

DISCUSSION

The patient presented was a para 2+0 who presented with preterm premature rupture of membranes at 34 weeks gestation. She was managed actively with good outcome.

Premature rupture of membranes (PROM) is rupture of the membranes for more than 8 hours prior to the onset of labour in a patient who is beyond 37 weeks of gestation. It is also referred to as prelabour rupture of membranes. Preterm premature rupture of membranes (PPROM) is rupture of the membranes prior to the onset of labour in a patient who is at less than 37 weeks of gestation. When the rupture of membranes persists for more than 24 hours, it is termed as prolonged. [1,2] The patient discussed presented with Preterm premature rupture of membranes at 34 weeks and 3 days gestation.

The incidence of PROM is approximately 3-10% of pregnancies.[1] In KNH the incidence was reported as 9.3% by Otieno and 8.2% by Wanjala.[3,4] Preterm premature rupture of the membranes (PPROM) is associated with 30-40% of preterm deliveries and is the leading identifiable cause of preterm delivery. Prematurity contributes to up to 85% of neonatal morbidity and mortality.[2]

The cause of PROM is not known but several risk factors have been identified. They include smoking, previous preterm delivery, occult amniotic fluid infection, multiple foetuses, polyhydramnios, cervical incompetence and abruptio placenta. Nutritional deficiencies of zinc and cooper have also been implicated.[6,7] Bacteria implicated with chorioamnionitis are known to bind invade and cross the chorioamnion membranes. The chorion, amnion, decidua and placental macrophages contain peroxidases. Some bacterias are known to produce hydrogen peroxide. The presence of the bacteria in these membranes activates the peroxidase-hydrogen system. The resulting free radicals lead to local tissue destruction, necrosis and cleavage of peptide bonds in collagen.[1,5] The patient discussed did not have any obvious risk factors for PROM.

Patients with PROM may present with complaints of leaking fluid, vaginal discharge, vaginal bleeding, and pelvic pressure. They may have symptoms of infections such as fever and abdominal tenderness if already infected. The fundal height may be smaller

than dates and the foetal parts readily palpable if significant amount of liquor has escaped. This patient complained of leaking fluid.

The diagnosis of PROM can be made by doing a sterile speculum exam during which pooling of fluid in the vagina or leakage of clear fluid from the cervix may be demonstrated. This vaginal fluid (liquor) when allowed to dry on a glass slide and viewed with a light microscope shows ferning or arborisation. It turns Nitrazine paper blue. Blood contamination of the Nitrazine paper can invalidate results.
The amniotic fluid index (AFI) can be helpful in demonstrating reduction of the amniotic fluid. If doubt still exists, transabdominal instillation of indigo carmine dye into the amniotic cavity under ultrasound guidance and observation of staining on a sanitary pad should confirm the diagnosis. This is rarely necessary.[1,2,5]

Oligohydramnios resulting from PROM may lead to several complications. They include pulmonary hypoplasia, facial and limb compression defects, and intrapartum foetal distress due to cord compression. [1, 2,5]

The natural history of PROM progresses in such a way that 90% of patients go into spontaneous labour within 24 hours. Evidence supports that induction of labour, as opposed to expectant management, decreases the risk of chorioamnionitis without increasing the cesarean delivery rate. [7] The management of preterm premature of membranes is either expectant or active, depends on the gestational age and the presence or absence of infection. The degree of oligohydramnios should also be considered. [1,6] Our patient was induced using syntocinon.

For patients who are at 34 weeks gestation and above labour is induced with intravenous oxytocin. This is the routine in our unit. The patient discussed was beyond 34 weeks and labour was induced with good outcome. For gestation age below 34 weeks conservative management may be employed. Complete bed rest with continuous or intermittent foetal heart rate monitoring and close observations for signs and symptoms of maternal infection is important. Some authorities advocate the use of antibiotics prophylaxis for prevention of chorioamnionitis. In some studies the antibiotics have been associated with prolongation of pregnancy and reduction in infant and maternal morbidity. Those against it argue that the antibiotics increase the

risk of vaginal colonization by resistant species and subsequent ascending infection. Sexual intercourse should be avoided. Digital cervical examination should be avoided unless prompt or immediate delivery is expected. [1,6,8]

If a patient develops evidence of intra-amniotic infection (maternal temperature >37.2°C, foetal tachycardia, fundal tenderness, foul or purulent vaginal discharge, maternal tachycardia, elevated C-reactive protein level) she ought to be delivered regardless of gestational age. Some authorities recommend corticosteroid use for women with PPROM prior to 30-32 weeks of gestation in the absence of chorioamnionitis. Other investigators have reported increased rate of infection with steroid use. Tocolytics may be used to prolong gestation long enough to complete a course of corticosteroids. [1,2,9,10]

Second-trimester PPROM has a dismal prognosis and risks of infection and oligohydramnios in conservative management seem to out weigh the benefit.[2]

References

1. Martin L, Pernoil M D,
   Late pregnancy complications in Current Obstetric & Gynecologic Diagnosis and Management. Appleton & Lange 8th Edition 1994 1: 336-338 .
2. Paul TW, Galan H
   Premature Rupture of Membranes; E medicine March 14, 2002
3. Otieno J A
   Preterm premature rupture of membranes Mmed Thesis University of Nairobi 1993
4. Wanjala S
   Preterm premature rupture of membranes, Mmed Thesis university of Nairobi 1983.
5. Cunningham G R Gant F N et al
   Preterm birth in Williams Obstetrics Mc Gray Hill 21st Edition.
6. Howl Y et al
   Aggressive tocolysis versus expectant management
   J. Maternal-fetal, med 1198; 7:8
7. Hannah ME, Ohlsson A, Farine D: Induction of labour compared with expectant management for prelabour rupture of the membranes at term. Term PROM Study Group. N Engl J Med 1996 Apr 18; 334(16): 1005-10
8. Mercer BM, Miodovnik M, Thurnau GR: Antibiotic therapy for reduction of infant morbidity after preterm premature rupture of the membranes. A randomised controlled trial. National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. JAMA 1997 Sep 24; 278(12): 989-95
9. NIH Consensus Development Panel: Effect of corticosteroids for fetal maturation on perinatal outcomes. NIH Consensus Development Panel on the Effect of Corticosteroids for Fetal Maturation on Perinatal Outcomes. JAMA 1995 Feb 1; 273(5): 413-8
10. National guidelines for quality obstetrics and perinatal care, MOH Division of Reproductive Health; Nov 2004

OBSTETRIC CASE 13

MALARIA IN PREGNANCY – LIVE BABY

Ip No.	09664883	D.O.A.	I5.10.04
Name	J M	D.O.D	20.10.04
Age	22	Parity	1 + 0

Presenting complain

J M was admitted with complains of hotness of body and chills for 1week.

History of Presenting Illness

The patient had been well till 1 week prior to admission when she developed hotness of body that alternated with chills. This was associated with joint pains, malaise and headache. She had nausea but had not vomited. She was passing urine normally and did not have diarrhoea. She had not travelled out of Nairobi for 6 months.

History of current pregnancy

Her last monthly period was on 19.10.03 and the expected date of delivery was 26.7.04. She was at the gestation of 38 weeks and 3 days. She attended clinic in Kenyatta National Hospital since 20 weeks gestation. The antenatal period was uneventful. The antenatal profile was as follows.

HB – 12.1 g/dl	VDRL – negative
Blood group – A +ve	Elisa for HIV – negative

Obstetric and Gynaecological history

She was a para 1 + 0. Her last delivery was in 2001 to a female infant, birth weight 3500 gm. The baby was alive and well.

J M attained menarche at 14 years. Her cycle was regular every 28 – 30 days with a normal flow of 3 – 4 days. She was not on any contraception prior to the pregnancy.

Past medical history

This was not significant

Family and social history

She was a housewife who lived with her husband in Dandora. Her rural home was in Kangundo. She had been in Nairobi for 2 years. She did not take alcohol or smoke cigarettes. She had no history of chronic illness in her family.

Examination

She was sick looking, mildly pale but not cyanosed or jaundiced. She had no lymphadenopathy or oedema. Her vital signs were: BP – 100 / 60 mmHg, PR – 115/min PR 24/min and temp 38.2°C

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

Abdominal examination

The abdomen was uniformly distended with fundal height at term. The foetus was in longitudinal lie, cephalic presentation and the head was 5/5 above the pelvic brim. There were no contractions palpable. The foetal heart rate was 132/min regular.

Pelvic examination

The external genitalia was normal. The cervix was long, central and was closed. The pelvis was clinically adequate.

Diagnosis

A clinical diagnosis of malaria was made and the patient was admitted.

Investigations

Blood slide for malaria parasites showed moderate p. falciparum parasitaemia.

Hb	9.5 g/dl	blood sugar	3.8 mmol/l
WBC	9×10^9	urinalysis	normal
Platelets	300×10^9	blood slide on 3rd day	no parasites

Management

The patient was started on intravenous 5% dextrose, intramuscular artenum 300mg stat and oral paracetamol. She then received 100 mg of artenum once daily for 4 days. Vital signs (blood pressure, temperature, pulse rate and respiratory rate) were

observed every 4 hours. She was encouraged to take small frequent feeds, which she successfully did. She improved remarkably. On the third day of treatment she complained of low abdominal pain that was radiating to the back. A vaginal examination revealed a dilated cervix at 4 cm. she was transferred to the labour ward for labour monitoring. The labour progressed well and she has a spontaneous vaginal delivery to a live female infant who weighed 3300 gm and scored 9/I, and 10/5. The baby remained well and did not have any complications. The mother remained stable after the delivery. She completed her artenum dose and a stat dose of sulphadoxine/ pyremethamine was added. On the 2nd post partum day she was discharged home on haematinics to be seen in the postnatal clinic after two weeks.

Follow up

The mother did not turn up for follow up.

Discussion

The patient discussed was para + 0 who had malaria at term. She was treated and responded well. In the course of treatment she went into spontaneous labour and delivered a healthy baby.

Malaria is caused by the parasite known as plasmodium. Four species can cause disease: Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, and Plasmodium malariae. Each species has a defined area of endemicity, although geographic overlap is common Malaria is predominantly observed in the tropics and is a potentially life-threatening disease.

In Kenya malaria accounts for 30% of outpatient hospital attendance. [1] Plasmodium falciparum is the commonest species in Kenya accounting for about 98% of the infections.[2] JM had falciparum infection.

The typical clinical symptomatology and severe disease pathology associated with malaria is caused by the asexual erythrocytic or blood stage parasites. Severe disease typically is secondary to infection with *P falciparum.* This species is more virulent because of the high parasitaemia that can occur and the property of sequestration, which may contribute to end organ damage. After an infected mosquito bites a human being, the malarial sporozoites enter hepatocytes and then emerge into the bloodstream after a few weeks. These merozoites rapidly enter erythrocytes and develop into trophozoites. As parasites develop in the erythrocyte numerous waste substances such as hemozoin pigment and other toxic factors accumulate in the infected red blood cell. These are dumped into the bloodstream when the infected cells lyse. These toxic factors stimulate macrophages and other cells to produce cytokines and other soluble factors which act to produce fever, rigors and probably influence other severe pathophysiology associated with malaria. Symptoms of malaria infection are non-specific and may manifest as flu like illness with fever, headache, malaise, fatigue, and muscle aches. Some patients may present with diarrhoea and other GI symptoms. Our patient presented with fever, headache, malaise, joint pains and nausea.

Malaria infection during pregnancy can have adverse effects on both mother and foetus, including maternal anaemia, foetal loss, premature delivery, intrauterine

growth retardation, and delivery of low birth-weight infants. It is a particular problem for women in their first and second pregnancies and for women who are HIV positive.[3] Our patient was having a second pregnancy but she was HIV negative. She developed mild anaemia, which was treated with oral haematinics.

The problems that malaria infection causes differ by the type of malaria transmission area. In high transmission areas, women have gained a level of immunity to malaria that wanes somewhat during pregnancy. Malaria infection is more likely to result in maternal anaemia, intrauterine growth restriction (IUGR) and delivery of low birth-weight infants. In low transmission areas, women generally have developed no immunity to malaria. Malaria infection is more likely to result in severe malaria disease, maternal anaemia, premature delivery, or foetal loss.[3,5] JM had not travelled to a malaria endemic zone. She had lived in areas of low transmission and so she was at the risk of developing severe disease. However she responded well to treatment. Though she went into labour she was at term and the baby as mature and of normal weight. This is probably because she got the infection late in pregnancy.

IUGR appears to result from placental insufficiency in the antenatal period and may be caused by acute induction of prostaglandins and arachidonate lipogenase metabolites in late pregnancy. Plasmodium falciparum-infected erythrocytes adhere to the vascular endothelium of venous blood vessel of the placenta and other organs. The placenta appears to be a site of preferential parasite sequestration and development. The density of placental parasitation is associated with delivery of low birth weight babies.[3,4] Low birth weight is a great risk factor for neonatal mortality and a major contributor to infant mortality. Intra uterine foetal death and congenital malaria are other possible complications of malaria.[2]

Anaemia associated with malaria infections is due to macrophages clearing the infected erythrocytes as well as phagocytizing and destroying uninfected red blood cells during the infections. Active malaria infections induce bone marrow suppression. Intravascular haemolysis does not appear to be a major contributor to malarial anaemia except in the pathological state known as blackwater fever. [3,4]

Treatment of malaria depends on the severity of the disease, the complications and the sensitivity of the parasite in the area where the disease was contracted. The safety of the baby should be taken into account but generally the benefits of treating severe malaria outweigh the risk to the baby.[6] Combination of drugs is advocated to deal with the serious challenge of malaria parasite resistance to drugs. Mild malaria is treated with oral preparations of sulphadoxine/ pyremethamine and amodiaquine. Oral quinine may be used in case of allergy to sulphur. For moderate to severe disease and resistant malaria parenteral quinine is used. Analgesics and anti pyretics should be used to relieve the symptoms. Resistance to the sulphadoxine/ pyremethamine has been noted locally. The artemesinin derivatives are being used widely. Their advantage includes fast response and convenient administration by intra muscularly once daily. Addition of the sulphadoxine/ pyremethamine is advocated to encourage parasites clearance. Oral preparations of artemesinin derivatives are available.[1,2,5] J M was treated with artenum, which is an artemesinin derivative. She responded very fast and the sulphadoxine/ pyremethamine was added. Complications of malaria such as anaemia, hypoglycaemia and dehydration should be sought and treated accordingly. J M had mild anaemia, which was treated with oral haematinics.

Malaria is a major factor and one of the few, along with poor nutrition, anaemia, and other infections, that is amenable to intervention once a woman becomes pregnant. Intermittent preventive treatment has is an effective approach to preventing malaria in pregnancy.[5] The ministry of health guidelines direct the use of sulphadoxine/ pyremethamine to pregnant women in malaria endemic areas twice during each pregnancy. This is given during the second trimester and the second dose in third trimester with an interval of at least 4 weeks between.[6] Pregnant women should sleep under insecticide treated nets to avoid mosquito bites. Other preventive measures include used of repellants, wearing clothes that cover arms and legs and environment management to clear bushes and drain stagnant water.[5] Our was counselled on these issues.

Malaria is common in puerperium and prophylaxis should be continued till 6 weeks postpartum. J M was given a dose of sulphadoxine/ pyremethamine at the end of her treatment with artenum. This would serve to enhance the treatment as well as serve as prophylaxis in the puerperium. Unfortunately she never came back for follow up.

References

1. Ministry of Health, Medical conditions in pregnancy in Essential Obstetric care; Manual for health service providers in Kenya. Pg 148-153, 2002
2. National guidelines for quality Obstetric and Perinatal Care, Ministry of Health, Department of Reproductive Health November 2004
3. National Centre for infectious Diseases, Division of Parasitic Diseases1600 Clifton Rd, Atlanta, GA 30333, U.S.A April 23, 2004.
4. Ibhanesebhar SE, Okolo A, Placenta and malaria. *Inter j Gynaecol. Obstetet* 37(4) 247 1192
5. Focused Antenatal Care and Malaria in Pregnancy, Orientation Package; Ministry of Health Kenya. MOH DRH/DOM/JHPIEGO August 2002
6. National guidelines for diagnosis. Treatment and prevention of malaria for heath workers. Ministry of Health. 1998

OBSTETRIC CASE 14

ABRUPTIO PLACENTA – LIVE BABY

Ip No.	0983493	D.O.A.	25.10.04
Name	J M	D.O.D	29.10.04
Age	38	Parity	3 + 0

Presenting complain

J M was admitted with complains of per vaginal bleeding for 2 hours and low abdominal pains for 4 hours.

History of Presenting Illness

The patient had been well till that morning when she noted mild abdominal pains. The pain persisted necessitating her to seek medical intervention. While travelling to the hospital she noted minimal per vaginal bleeding. She had not experienced any trauma or strain.

History of current pregnancy

Her last monthly period was on 14.2.04 and the expected date of delivery was 21. 11.04. She was at the gestation of 36 weeks. She attended clinic in Kenyatta National hospital from 20 weeks gestation. The antenatal period was uneventful. The antenatal profile was as follows.

HB – 13.1 g/dl	VDRL – negative
Blood group – A +ve	Elisa for HIV – negative

An ultra sound evaluation done on 25.6.04 had revealed a normal intra uterine pregnancy with a fundal placenta.

Obstetric and Gynaecological history

She was a para 3 + 0. Her first delivery was in 1990 to a female birth weight 2800 gm. In 1993 she had another delivery to a male infant birth weight 3300 gm. Her last delivery was in 1998 to female infant birth weight 3200 gm. All the deliveries were vaginal at term and the children were alive and well.

She attained menarche at 14 years. Her cycle was regular every 28 – 30 days with a normal flow of 3 – 4 days. She was not on any contraception prior to the pregnancy.

Past medical history

This was not significant

Family and social history

She was a housewife married to a businessman. She did not drink alcohol or smoke cigarettes. She had no history of chronic illness in her family.

Examination

She was in good general condition, not pale or jaundice. She had no lymphadenopathy or oedema. Her vital signs were: BP – 100 / 60 mmHg, PR – 88/min PR 22/min and temp 36.6°C

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

Abdominal examination

The abdomen was uniformly distended with fundal height at 36 weeks gestation. The foetus was in longitudinal lie, cephalic presentation and the head was 5/5 above the pelvic brim. There were no contractions palpable but there was tenderness in the supra pubic region. The foetal heart rate was 132/min regular.

Pelvic examination

A sterile speculum examination was done. The external genitalia was normal. The cervix was long, central and was closed. There was a clot of blood in the posterior fornix but no active bleeding.

Diagnosis

A diagnosis of antepartum haemorrhage was made

Management

The patient was admitted and an urgent ultra sound ordered. Blood for grouping and cross matching was taken. The ultra sound was done and revealed an intra uterine gestation at 35 weeks and 4 days. The placenta was fundal with no features of

separation. The foetal movements were reduced with no breathing movements and the tone was reduced while in the ultra sound room the patient started bleeding actively. A written consent was taken and a normal saline drip started immediately. The patient was then wheeled to theatre and the paediatrician was called. In theatre an emergency caesarean delivery was accomplished. The amniotic fluid was found to be blood stained. The outcome was live male infant who scored 9/I, 5/5and 9/10. The baby was resuscitated and admitted to the newborn unit for further observation. The placenta was found to be normal grossly but there were 2 small retro placental clots.

The mother remained stable post operatively and recovered well. The baby improved and stabilized within the first 24 hours. On the 4th post partum day both the mother and the baby were then discharged home to be seen in the postnatal clinic after two weeks.

Follow up

She came for review after 2 weeks. She had no complaints. The wound had healed well. Her breasts were active and soft. The uterus had involuted. She came again for review after one month. She raised no complaints. Advise on family planning was given and she was discharged through the family planning clinic.

Discussion

The patient discussed was admitted with per vagina bleeding for 2 hours and low abdominal pain. A diagnosis of placenta abruption was made. A quick delivery was accomplished and the baby survived.

Placenta abruption refers to the premature separation of a normally implanted placenta before the delivery of the foetus. The separation results in haemorrhage into the decidua basalis near its interface with the placental cytotrophoblastic shelf and the anchoring villi. [1,2,3] The haemorrhage may be external in which case it flows through the cervix and is detectable. In about 20% of the cases the haemorrhage is concealed.

The incidence of placenta abruption varies and on average is 1 in 200 deliveries. [1, 4, 5] At KNH the incidence of ante-partum haemorrhage was reported to be 4.7 – 6.7 % of all deliveries. 15.4% of these resulted from placenta abruption.

The cause of placenta abruption is unknown but several risk factors are recognised. Previous placental separation is a predisposing factor. Following one episode, the incidence of recurrence is 10 – 17 %. With two previous episodes the incidence of occurrence exceeds 20%. [1,2, 4] The commonest associated condition is hypertension in pregnancy. Mothers who are hypertensive are more likely to suffer a more severe abruption but the severity of the hypertension does not correlate with the incidence of abruption. Premature pre term ruptured membranes are associated with increased incidence of abruption. [2, 6, 7] Other factors associated with increased risk of abruption are cigarette smoking, cocaine abuse, thrombophilias, advanced maternal age, multiparity and alcohol consumption. Any condition leading to distension of the uterus (multiple pregnancy and polyhydramnios) increases the risk of abruption. Diabetes and collagen diseases have also been implicated as well as uterine anomalies or tumours. [1,2,5] Other than being advanced in age and multiparity, J M did not have any of these other risk factors. She was 38 years old, para 3 + 0.

Precipitating factors to abruption include abdominal trauma, circumvallate placenta, sudden reduction in uterine volume and abnormally short cord during delivery. The

patient presented was found to have marginal insertion of the cord with a succenturiate lobe but not circumvallate.

Placental abruption is initiated by haemorrhage into the deciduas basalis. The decidua then splits leaving a thin layer adherent to the myometrium. Several mechanisms are thought to the important in the pathophysiology. Local vascular injury with bleeding into the decidua and haematoma formation is one mechanism. Another mechanism is an abrupt rise in uterine venous pressure transmitted into the intervillous space. The resulting venous engorgement leads to separation of the placenta. Mechanical separation is thought to occur following trauma sudden decompression of the uterus or traction by a short cord. Initiation of the coagulation cascade leading to clot formation in the placental pool with some relative stasis is another possible mechanism. [1,2,3] The separation of the placenta may be complete or partial. The blood may gain access into the amniotic fluid. Maternal foetal haemorrhage foetal maternal haemorrhage or amniotic embolism may occur. In the patient discussed, the amniotic fluid was bloody. Blood may infiltrate the uterine wall. When extensive intramyometrial bleeding occurs uteroplacental apoplexy (couvelaire uterus) results.

The clinical presentation varies. About 30% of the separations are small producing few or no symptoms and usually will not be noted until the placenta is inspected. Per vaginal bleeding is present in 80% of the cases and 66% have abdominal or back pain. About 20% of the patients are erroneously diagnosed as having idiopathic premature labour, [8,1,2] Foetal distress is present in more than 50% of the cases, with foetal demise in 15%. When the separation is extensive, uterine tetany, disseminated intravascular coagulation and hypovolemic shock may set in. Following shock oliguria may develop. [1,2,8] The patient discussed presented with abdominal pain, mild per vaginal bleeding and features of foetal distress were demonstrated on ultra sound.

Placental abruption will usually be suspected from the clinical presentation. Ultra sound may help confirm the diagnosis but negative findings with ultrasound examination do not exclude abruption. [9] Laboratory tests should be done to determine the haemoglobin or the packed cell volume, and the coagulation status. Bedside clot observation test is a simple and valuable procedure when serial laboratory tests are not practicable. In this test venous blood is placed in a clear test tube. A clot should

form within 5 – 10 minutes and dissolve in shaking the test tube when the clot system is within normal. Hypofibrinogenaemia of less than 200mg/dl is suggestive of placenta abruption. Our patient was evaluated using an ultra sound but the separation was not visualized.

Placenta abruption is graded on the degree of separation, the status of the foetus and the presence or absence of DIC. [10]

Grade I not recognizable clinically before delivery usually diagnosed by presence of retro placenta clot.

Grade II intermediate: classical signs of abruption are present and the foetus is alive.

Grade III severe: the foetus is dead

III a without coagulopathy.

III b with coagulopathy.

The patient discussed had placenta abruption grade II.

The management of placenta abruption depends on the gestational age and the status of the mother and the foetus. When the foetus is immature, bleeding is not extensive and in absence of uterus irritability or foetal distress expectant management is beneficial [11, 12] The woman should be hospitalised, typed and cross-matched and observed for signs of further separation. Some authorities advocate tocolysis for preterm pregnancy to improve the outcome [11, 12] Other authorities consider abruption as a contraindication for tocolysis because of the danger of masking the symptoms of further separation. [1,8]

Vaginal delivery is indicated for minor degree of separation but the foetus should be monitored for distress. With a dead foetus or non-viable, vaginal delivery is indicated with the exception of severe, uncontrollable haemorrhage. Amniotomy is done to release as much amniotic fluid as possible and induction of labour with ocytocin infusion should be instituted if active labour does not begin shortly after. The labour usually progresses rapidly. Caesarean section is indicated whenever vaginal delivery is not imminent and the foetus has a reasonable chance of survival. When haemorrhage is uncontrollable, a caesarean section is done to save the mothers life. Maternal resuscitation with blood and blood products and crystallised fluids should be

done as indicated. Laboratory tests are important in guiding what needs replacement. Fresh whole blood is superior in treating clotting deficiencies and replacing blood loss. However packed red blood cells, cryoprecipitate packs, platelets and fibrinogen may be given if available and necessary. The patient should be monitored closely in the post partum period for any complications. [1,2,8]The patient discussed had a live baby with features of distress and she begun bleeding profusely. A caesarean section was rapidly accomplished and both the mother and the baby did well.

Several complications may arise with abruption. Maternal mortality rate ranges from 0.5–5 %. Most mothers die of haemorrhage, or cardiac or renal failure. Early diagnosis and appropriate therapy reduce the mortality rate. Foetal mortality ranges from 5.9 – 8.0 % and live births have high rate of morbidity resulting from pre delivery hypoxia, birth trauma or prematurity. Other complications encountered include disseminated intravascular coagulation, acute corpulmonale (as a result of emboli in the pulmonary microcirculation) renal cortical and tubular necrosis and risks associated with transfusion such as hepatitis infection. Uterine apoplexy may lead to uncontrollable bleeding from the placental bed necessitating a hysterectomy.[1,2] J M did not develop any of these complications. The baby was initially distressed but did well on resuscitation.

References

1. Cunningham G R Gant F N et al Obstetric haemorrhage in Williams Obstetrics Mc Gray Hill 21st Edition 2001 25:620-630
2. Permoll M. L; Third trimester haemorrhage in current Obstetric & Gynecological Diagnosis and Treatment. 8th Edition 20:398-404.
3. Nimrod CA, Oppenheimer LW; Third trimester haemorrhage, medicine of the foetus and mother. 2nd edition Lippicott, Philadelphia 1999; 1497 – 1505
3. Caregard M, Gennser G; Incidence and recurrence rate of abruption placenta in Sweden. Obstet Gynaecol 67; 523, 1986
4. Ananth CV smulian JC vintzileos Am; Incidence of placental abruption in relation to cigarette smoking and hypertensive disorders during pregnancy. A meta analysis of observational studies. Obstetric gynaecology 93; 622, 1999
6. Morgan MA Berkowitz KM, Thomas SS et al abruption placenta prenatal outcome in normotensive and hypertensive patients. AM Obstetric Gynaecological 170; 1595, 1994.
7. Witlin AG, Saade G R mother F, Sibai BM; Risk factors for abruption placenta and eclampsia analysis of 445 consecutively managed women with severe preclampsia and eclampsia AM J Obstetric 180; 1322, 1999
8. Hurd W. Misodounick M et al; Selective management of abruption placenta; a prospective study. Obstetric gynaecology 61; 467, 1985
9. Yeo L, Vintzilos AM, Guzman ER, et al. The sonographic diagnostic accuracy of abruptio placenta in patients with premature rupture of the membranes vs intact membranes. Am J. Obstet Gynaecol 180; s166, 1999
10. Barren SL abruptio placenta, Turnbulls obstetrics 2nd edition Churchill Livingston, London 1996, 319 – 325
11. Scholl JS; Abruptio placentae; clinical management in non-acute cases. Am J obstetric gynaecology 156; 40 1987.
12. Bond AL, Enderschiem TG et al. Expectant management of abruption placenta before 35 weeks gestation. Perinatol6; 121, 189

OBSTETRIC CASE 15

ADOLESCENT PREGNANACY – POOR OUTCOME

Name	A. N.	I. P. No.	099090
Age	16	DOA	1.11.04
Parity	0 + 0	DOD	9.1.04

Presenting Complaint

The patient was admitted as a referral from a private clinic with history of labour pains for 2 days.

History Of presenting Complaint

She had developed labour pains and drainage of liquor while at home and went to the private clinic after 6 hours. In the clinic the labour continued for about 14 hours before she was referred to Kenyatta National Hospital.

Antenatal History

Her last menstrual period was on 24.1.04 and her expected date of delivery was 1.11.04. At the time of admission she was at 40 weeks gestation by dates.

She attended antenatal clinic twice in Kawangware. She had not had an antenatal profile.

Obstetric and Gynaecologic History

She was a primigravida. She attained menarche at 14 years. The menstrual period was normal every 30 days and the flow lasted 3 – 4 days. She had never used any form of contraception.

Past medical history

This was not significant

Family and social history

She was a single girl living with her parents in Kawangware. She had dropped out of school in form two due to the pregnancy. She was the first born in a family of 4

siblings. The rest were all in primary school. Her mother was a housewife while the father was a casual labourer. There was no known chronic illness in the family. She did not take alcohol or smoke cigarettes.

Physical examination

She appeared to be in distress. She was moderately dehydrated but not pale or jaundiced. Her vital signs were as follows

BP	120/80mmhg	Temperature	36.6oc
PR	92/min	RR	20/min

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

Abdominal examination

The abdomen was uniformly distended. The fundal corresponded to term. The lie was longitudinal with cephalic presentation and descent of $^2/_5$ above the pelvic brim. The foetal heart was heard at 136 beats per minute. She was noted to have strong contractions, 3 – 4 in 10 minutes lasting 30 – 40 seconds.

Pelvic examination

The external genitalia was oedematous. The cervix was fully effaced, 9 cm dilate. The head was noted to have caput formation and second-degree moulding. The vaginal walls were warm and dry.

Diagnosis

A diagnosis of prolonged labour secondary to cephalopelvic disproportion in teenage primigravida was made.

Management

A decision was made to deliver her immediately by caesarean section. The patient and her mother were explained to the diagnosis and the need for an emergency caesarean section. The mother signed the consent.

The patient was started on intravenous normal saline solution and received 30mls of 50% dextrose while she was being prepared for theatre, she felt the urge to push. She

then progressed to delivery vaginally. The outcome was a live female infant. Birth weight of 3350 gm. The apgar score was $^{2}/_{1}$, $^{3}/_{5}$ and $^{4}/_{10}$. The baby was admitted to the newborn unit due to birth asphyxia. Unfortunately she succumbed after 8 hours.

The placenta was delivered by controlled cord traction. The mother was given ergometrine 0.5mg intramuscularly. A foley catheter was inserted immediately and even though the urine was clear, it was retained for 7 days post delivery. Her perineum was intact.

Post partum management

The mother remained stable post delivery. Rehydration was continued intravenously. Her vital signs remained normal. Intravenous zinacef and flagyl were started. Oral haematics were given. She was taught on breast care and started on bromocriptine for suppression of milk after the baby died. She remained stable. A counsellor from the adolescent clinic was called. A few sessions of counselling were held while in the ward. She was to continue later after discharge.

Investigations done in the ward included;

Hb	-	8mg/dl	VDRL	-	negative
Elisa for HIV	-	negative	Blood group	-	O +ve

On the 7th post partum day the catheter was removed. She remained well and on the 8th post partum day she was discharged home to be seen in the post partum clinic after 6 weeks.

Follow up

She came for review in the clinic and was well with no complaints. Counselling on abstinence and safer sexual practices and was done. Contraception advice was also offered. She was advised on going back to school to continue with her education, preferably in a different school. Her parents were also counselled and encouraged to be supportive.

Discussion

The patient presented was a 16 year old who presented with prolonged labour and delivered vaginally. The outcome was a live baby who scored poorly leading to a perinatal mortality

Adolescence is the period during which an individual progresses from the point of the initial appearance of the secondary sexual characteristics to that of sexual maturity. The individual's psychological process and patterns of identification develop from those of a child to those of an adult and there is a transition from this state of total social economic dependence to relative independence.[1] Adolescents are defined as persons aged 10-19 years. [2]

The adolescent population in Kenya form an important segment of the population. Constituting about 25% of the total population. [2] There is high adolescent fertility rate, which has been increasing over the years. Adolescents indulge themselves in sexual activity freely. They seem to be unaware of the risks to which they expose themselves. These risks include unplanned pregnancies and sexually transmitted infections including HIV. Some studies have shown that up to 23% of adolescents are sexually active with majority having had sexual intercourse within 2 years of attaining menarche. [3] A N had attained menarche at age14 and at 16 years she delivered.

There are major problems facing adolescent health in Kenya. These include lack of formal reproductive health education and information; instead adolescents are exposed to misinformation. Another problem is limited availability of reproductive health care services including contraception and family planning services since they tend to be seen as more suited for the adults or the married. A N was sexually active but she had never used any protection to avoid unwanted pregnancy or sexually transmitted infections.

The rate of adolescent pregnancies had been rising worldwide. In Kenya the rate was given at 1.1% in 1983 and 21% by 1985. [4,5]

When the teenagers conceive, the pregnancies are usually unplanned. Statistics show adolescent pregnancies are at high risk of poor outcome both for the baby and the mother. [3,4,5] Many adolescents come from low social economic background with

having poor education. They may be at risk of poor nutrition, cigarette smoking, and drug abuse. These factors contribute to poor pregnancy outcomes.

Teenage mothers need optimal care to improve the pregnancy out come. In KNH a special adolescent clinic had been established but utilisation remained low. Many teenage mothers do not attend antenatal clinic or when they do they book late. Many seek maternity a services late during delivery. Lack of money to pay for these services contributes significantly to this trend. The patient discussed had attended ANC only twice and did not do any tests. When she went into labour she hid not seek health services immediately. She then ended up in a clinic that did not offer optional labour management for her. She delayed in seeking health services due to financial constraints. She was a form 2 drop out and her father was a casual labourer with little income and a fairly large family to cater for.

Other hazards associated with teenage pregnancies are iron deficiency anaemia, preeclampsia – eclampsia which is more common in the first pregnancy, prematurity and small for dates infants. Some of these problems may be worsened by the lifestyle of the adolescent such as poor feeding and cigarette smoking.[6] The patient presented did not abuse drugs but she was found to have anaemia.

Complications of labour and delivery are highly dependent on the quality of antenatal care and the quality of the management. A. N. had poor antenatal care and labour management. By the time she got to KNH she had been in labour for 2 days. She had a vaginal delivery before the caesarean section could be done with a poor outcome, resulting in a perinatal mortality.

Adolescent mothers are at an increased risk of repeat pregnancies.[5] It is important to offer counselling before discharging them after delivery. The counselling should include issues of safer sex, family planning, and self-improvement. A.N was advised to go back to school and to practice safer sex.

Contraceptive use is lowest among women aged 15 – 19 years according to KDHS 2003. Only 11.6% of married women in this age group were found to be using any modern methods of contraception.[7] The percentage is most likely lower among the single women in this age bracket yet many of them are sexually active. Contraceptive prevalence increases dramatically with increasing level of education.[7] It is therefore important to encourage the education of the girl child in order to improve their social economic status as well as their utilisation of reproductive health services. This will result in less unplanned pregnancies and their complications. A N was counselled to go back to school and her parents were going to assist her.

References

1. World health organisation (WHO) pregnancy and abortion in adolescence: A report of WHO meeting. World health organisation. Rep, 583: 27 1975
2. Adolescent Reproductive Health & Development Policy; Ministry of Planning and Development -NCPD, Ministry of Health- Division of Reproductive Health. May, 2003
3. Lema V M. Factors associated with adolescent sexuality amongst secondary school girls in Nairobi Kenya. J Obstetric Gynaecology East & central Africa 8(1); 38 – 44 1989
4. Sangui H C G. Outcome of pregnancy in teenage mother in Nairobi Kenya. Mmed Thesis, University of Nairobi 1983
5. Muraya G.N. Teenage pregnancy in rural Kenya. Mmed Thesis university of Nairobi, 1985
6. Muram David, Paediatric and adolescent gynaecology in current obstetric and gynaecologic diagnosis and treatment. 8th edition, Appleton and Lange 3: 659 – 660
7. Kenya demographic and health survey 2003 – preliminary report, CBS, MOH, KEMRI, CDC, Nairobi Kenya.

OBSTETRIC LONG COMMENTARY:

THE QUALITY OF ANTENATAL CARE IN RESPECT TO ASSESSMENT OF ANAEMIA AMONG WOMEN DELIVERING AT PUMWANI MATERNITY HOSPITAL NAIROBI.

Abstract

Objective of the study: To determine the quality of antenatal care with respect to assessment of anaemia, among women delivering at Pumwani maternity hospital in Nairobi, Kenya.

Study Design: This was a Descriptive Cross sectional survey carried out at Pumwani maternity hospital in Nairobi, Kenya. The study was carried out in the months of August and September 2004.

Methodology: Women who came to deliver at Pumwani were recruited into the study. Data from their antenatal records was filled in predetermined questionnaires. The results were entered into a computer and then analysed using the SPSS program.

The Results: Of 312 women recruited into the study, 35% of them did not have their haemoglobin estimation done by the time of delivery. Of the 65% who had their haemoglobin estimation done, 9.4% were found to be anaemic. As many as 23% of the mothers were below 20 years of age and only 6% of these had booked ANC in the first trimester. About half (48%) of those below 20 years did not have their haemoglobin estimation done. Majority of the mothers (62%) booked in the second trimester and 36% booked in the third trimester.
For the women found to have Hb less than 10g/dl, for one fifth (21%) of them no action was taken, for three quarters (74%) haematinics were given and 5% were given antihelminthes empirically but no haematinics. Only one fifth (21%) of these women had a repeat Hb estimation done

Conclusion: the quality of ANC in respect to anaemia is poor and needs improvement.

BACKGROUND AND LITERATURE REVIEW

Importance Of Antenatal Care

The antenatal care is an important part of preventive medicine.[1] Pregnancy is a normal physiologic event but it is complicated by pathologic process dangerous to the health of the mother and foetus in 5 – 20% of cases[2]. This is why special care is needed in order to ensure as much as possible an uncomplicated pregnancy and the delivery of a live healthy infant to a healthy mother. [2] Recognition of the need for maternal and child health care came from article 25-2 of the universal declaration of human rights as proclaimed by the general assembly of the United Nations December 10th 1948. Since then antenatal care has been incorporated into primary health care programmes. In Kenya antenatal care was introduced into comprehensive maternal and child health care and family planning programmes in the second half of the 1970s and it has become an integral part of the primary health programme.

Studies have shown that mothers not attending antenatal clinic (ANC) are likely to have certain social demographic characteristics and are more likely to have poorer pregnancy outcome. They are likely to be young, multiparous and unmarried. Perinatal out come is significantly worse in these patients compared to those who are regular antenatal clinic attendees. They are more likely to deliver prematurely to low birth weight infants and are at greater risk for stillbirths and neonatal death. [3]

Access To ANC/ Utilization Of ANC Services

Studies have shown an increase in seeking antenatal care among pregnant mothers both in Kenya and elsewhere. By the end of the 20th century prenatal care had become one the most frequently used health services in the United States.[4]According to the Nairobi Birth Survey, 96.4% of the mothers had attended antenatal clinic at least once and only 3.6% had not.[5] The Kenya Demographic and Health Survey, 2003 reported that 9 out of 10 of the mothers had reported to a health worker at least once for ANC [6] While this increase in antenatal clinic attendance is commendable it is worth noting that attending the clinic is one thing but getting good quality care is a different thing altogether.

Quality Of ANC & Pregnancy Outcome

Despite the wide spread use of pre-natal care the evidence for its effectiveness remains equivocal and its primary purpose and effects continue to be a subject of debate internationally.[7] Some studies have indicated worsening trends in birth outcome despite increasing trends toward more prenatal resource utilization.[8] This suggests that ANC attendance does not always result in the expected results. Inadequate antenatal care can be dangerous. This is because of the false confidence that since one has attended a clinic regardless of the level of care then all is well.[9] Ante-natal care has been practiced in much the same way in most countries for the past 50 years. Neither the timing of visits nor the procedures done have been rigorously evaluated for their effectiveness. New procedures have been introduced in a similar manner.[10]

Maternal death is the worst out come of pregnancy and is often an indication of poor antenatal care or none at all. Maternal mortality is still unacceptably high in the developing world including Kenya. In northern Europe the figures are reported to be as low as 0-11 deaths for every 100,000 live births. Reduction in maternal and infant morbidity and mortality in England and USA has been attributed to good antenatal care.[11,12] Little is known as to whether or not routine antenatal care is effective in preventing maternal mortality and morbidity in developing countries.[13] In Cameroon 82.4% of women who died had attended ANC indicating that probably poor quality of care was responsible.[14] Makhoha found that in KNH 29.4% of mothers with avoidable deaths had attended ANC. In this study, 9.6% of those with avoidable deaths, died of anaemia.[15] These mothers had attended ANC but early screening and supplementation of haematinics was not done. With better care the level of mortality might have been lower. Obore found maternal mortality rate of 921.5 per 100,000 in Kenyatta National Hospital for the years 1995 – 1999 and 28% of these women had attended antenatal clinic.[16] Makhoha reported a maternal mortality rate of 196 per 100,000 births between 1972 and 1977 and 320 per 100,00 deliveries between 1978 and 1987 at Kenyatta national Hospital.[15] Thus maternal mortality has remained high despite increased ANC attendance.

ANAEMIA

Anaemia And Physiological Changes In Pregnancy

Anaemia in pregnancy is a condition in which the haemoglobin level in the pregnant woman is less than 10g/dl in our set up.[17] In some centers anaemia is taken to be haemoglobin less than 11g/dl.[18] Many physiologic changes occur in pregnancy and among the most striking are in the hematological system. Blood volume increases by up to 100% by term. Plasma increases earlier and faster than the red cells, hence a dilution effect occurs leading to what is known as physiologic anaemia in the second trimester. This means that pregnant mothers have lower haemoglobin levels than the general population. [18]

Iron deficiency anaemia contributes to about 90% of anaemia during pregnancy. In our set up the iron deficiency is contributed to by poor dietary intake and hookworm infestations.[19] In pregnancy the foetus requires iron for production of the foetal haemoglobin. This passes from the mother along with other nutrients. The increase in the red cell mass together with the foetal requirements increases the need for maternal iron intake. Considering that many women of reproductive age lack sufficient iron stores, even outside of pregnancy there is need for supplementation. [20] Failure to supplement may lead to iron deficiency anaemia. This demand is even higher with multiple gestations, making anaemia more common and more severe. Folate requirement also increases since it is used in production of red blood cells and should be supplemented together with iron. A pregnant woman will lose blood at delivery; an anaemic woman is therefore at increased jeopardy, hence the need to correct any anaemia before delivery.

Anaemia is one of the common problems in pregnancy especially in the developing countries. Early screening of anaemia is one of the aims of the antenatal care. Prevention and early correction is possible and this improves pregnancy outcome.

The incidence of Anaemia in pregnancy varies in different geographical areas.
WHO estimates that 58% of pregnant women in developing countries are anaemic.[21]

Gebbie reported that anaemia was the second most common obstetric problem in Kenya. [22] This is despite the effort made to encourage iron supplementation in the antenatal period.

Effects Of Anaemia In Pregnant

Anaemia is associated with increased maternal morbidity and mortality, as well as poor pregnancy outcome. Defective antenatal care and the magnitude of anaemia significantly influence the maternal and foetal out look. The frequency and severity of complications parallel the severity of anaemia and the incidence and severity of complications are worse in developing nations .[23,24] According to WHO, Anaemia has been implicated as contributory in up to 40% of maternal deaths in the third world countries.[25] Kenya is among the10 countries in the world with highest maternal mortality.[26] Ngoka in Pumwani Maternity Hospital found that 69% of mothers who died had attended ANC but no routine investigations were done, yet investigations are major component of preventive care in the ANC. [27] In Coast General Hospital Solomon found that more than 50 of the mothers who died had no routine laboratory tests done though they had attended ANC. [28] He found anaemia to have been the most prevalent complication. It was associated with high perinatal and maternal mortality. Mothers getting quality ANC have higher haemoglobin levels with less of complications associated with anaemia. By reducing anaemia and its complications the maternal mortality may be decreased.

The red blood cells are the main oxygen carriers in the body. With low haemoglobin the oxygen carrying capacity is reduced and this reduces the oxygen available for placental transfer to the baby. Even moderate anaemia results in reduced oxygen tension of amniotic fluid and intra uterine foetal hypoxia.[29] Anaemia may be associated with foetal growth restriction, foetal distress, toxaemia and prematurity. [30,31] The correction of anaemia has been reported to reduce the incidence and severity of complications. Aggarwal reported that the birth weight, crown heel length, head circumference of the neonate and placental weight increased significantly with rise in maternal haemoglobin levels[32]

Evaluation And Intervention

Ideally a woman planning to have a baby should have a medical evaluation before she conceives.[1]When this is done; those found to have low haemoglobin level are identified. The anaemia is then corrected and their iron stores restored before they conceive. In our set up however many women visit the clinics when already pregnant. Anaemia must be sought and corrected during the earliest visit. In the successful management of anaemia, reliable techniques for detection, assessing its severity and monitoring the response to appropriate treatment should be available.

During the first visit a full physical examination should be done. This includes among other things looking for pallor in several different areas, such as the nail beds, tongue, palms, and the conjunctiva. Studies have shown that although imperfect, use of pallor to screen and treat severe anaemia by primary care providers is feasible and worthwhile. Usually, the majority of persons with severe anaemia will be detected during such examination [33] It was shown that in conducting clinical screening of moderate to severe anaemia (Hb <8 g/dl) using pallor for a variety of body sites, sensitivity ranges between 53-100%. [34]

Different methods such as the copper sulphate, cyanmethemoglobin method, the Coulter counter, the haemoglobinometer, and the HemoCue instrument may be used depending on the availability. When the laboratory tests results are available the anaemia if present should be graded and corrected accordingly. Where the laboratory availability is limited then clinical screening should be used.

Initial evaluation of a pregnant woman with anaemia should include measurements of haemoglobin, the haematocrit and red cell indices. A careful examination of the peripheral film is advisable. In moderate and severe anaemia estimations of the serum iron concentration or ferritin are helpful. Other important tests include stool examination for hookworm, blood slide for malaria parasites, as well as sickling test depending on the woman's genetic background. Other more specific tests depend on the results of the initial tests as well as the history and the physical examination findings.

Management depends on whether it is: moderate (70-100 g/l), severe (40-69 g/l) and very severe (<40 g/l). Corresponding haematocrit (PCV) values are 24-35%, 13-23% and <13% respectively.[35]

Mothers with severe anaemia (very pale, reporting symptoms such as easily fatigued, breathless on mild exertion, weakness, dizziness) may need admission. The cause should be treated and corrective measures for the haemoglobin level and iron stores taken. In acute or severe anaemia, transfusion may be necessary. Treatment with total dose infusion of iron (TDI) helps build the depleted iron stores and overcomes the difficulties associated with oral supplementation. This will also depend on the gestation of pregnancy the closer to term the higher the need for blood transfusion.

After the first screening is done in the first visit, ideally in the 1st trimester, a repeat should be done in the eighth month of pregnancy but clinical assessment of anaemia should be done in every visit. If the anaemia is mild or moderate oral haematinics will usually be sufficient.

WHO recommended that as a minimum, the woman attending ANC should expect and demand iron/folic acid tablets. Other recommendations included early screening for anaemia by clinical examination and laboratory testing for pregnant women and treatment for any anemia.[36]

Justification

Anaemia continues to be a health hazard for pregnant women. This is more severe and prevalent in the developing countries of which Kenya is one. Anaemia is reported to be the second most common obstetric problem in Kenya.[22] Anaemia can be detected and corrected during antenatal care, if the services provided are adequate. This study evaluated the quality of ANC with respect to anaemia in a cross section study of pregnant women attending the most populous maternity hospital in Kenya.

BROAD OBJECTIVE

To determine the quality of antenatal care in respect to assessment of anaemia among women delivering at Pumwani maternity hospital in Nairobi, Kenya.

Specific Objectives

1) To document the proportion of women attending ANC who were screened for anaemia.
2) To document the prevalence of anaemia in pregnancy in the study population.
3) Evaluate the proportion of anaemic women treated for anaemia.
4) To document the social demographic characteristics of the mothers with anaemia.

Study Design/Methodology

This was a cross-sectional descriptive survey. The study was carried out in the Pumwani maternity hospital in August and September 2004. The data was sourced from the antenatal cards of the patients who came for delivery in this hospital into a predetermined questionnaire and then analysed. The questionnaire was first pre tested to establish its suitability and reliability. Fifteen questionnaires were used. The questionnaire was then redesigned.

Inclusion Criteria

Those mothers who delivered in Pumwani maternity hospital during the period of the study and had attended antenatal clinic.

Exclusion Criteria

The mothers whose antenatal records were not available, those who had not attended ante-clinic and those who did not consent to avail their records were all excluded from the study.

Procedures

Clients were recruited at the admission desk as they came for admission. Every second mother admitted was provided with an explanation and asked to give written consent for the study. This was to try and eliminate bias. The explanation was done at the convenience of the mother depending on her condition. Those mothers who were below 18 years of age had the consent given by a guardian. Once the consent was obtained, the relevant data was filled in to the questionnaires by the researcher. The principle researcher locked up the data obtained to ensure confidentiality. It was then entered into a computer using the EPI info and analysed using the SPSS program by the researcher with assistance from a data analyst. Data presentation was done in descriptive, tabular and graphic form. Appropriate tests of significance (chi square, trend test) were done.

Study Population/Study Subjects/Setting

Pumwani Maternity hospital is located about 5 Km North East of Nairobi city. The study subjects were antenatal records of women who came to deliverer in Pumwani Maternity hospital. The hospital is a Nairobi city council institution that receives patients mainly from Nairobi and auxiliary Nairobi city council health institutions, private health units and many unbooked patients from the surrounding areas. The average number of deliveries per day is 60.It mainly serves clients of the middle to low social economic status.

Study Period

The study took 2 months. It was carried out in the months of August and September 2004.

Statistical/Analysis Plan

The data obtained was then entered into the computer by the investigator with the help of a statistician. Analysis was then done using the SPSS program. The quality of ANC was assed on the basis of

- Was physical examination for anaemia done?
- At what gestation was the booking done?
- Were laboratory tests for haemoglobin done?
- If the mother was anaemic was she treated appropriately?
- Was routine supplementation done?

Sampling And Sample Size Calculations

The sample size was calculated using the following formula

$n = \frac{(Z\alpha/2)^2 (P)(1-p)}{d^2}$ P* = the prevalence

n = the sample size d = margin of error

A 95% degree confidence corresponds to $\alpha = 0.005$ In the table of the standard normal distribution this corresponds to a z value of 1.96

Therefore $n = \frac{(Z\alpha/2)^2 (P)(1-p)}{d^2} = \frac{(1.96)(0.72)(0.28)}{0.005} = \frac{3.8419 \times 0.2016}{0.0025}$

$n = 309.786$

$n = 310$

*In a study done in Kenyatta National Hospital antenatal clinic, 13 % of the mothers were found to have low haemoglobin levels. Of these 72% had some action taken to correct the anaemia.[42]

Ethical Considerations

This study involved getting data from the records and analysing it. The subject's names were not reflected in the questionnaires. Informed consent was obtained from those mothers whose records were used.

Permission to carry out the study was obtained from the Kenyatta National Hospital ethical committee, and the Pumwani maternity hospital ethical committee.

Limitation

The data was collected from patients' records some of which were not well kept. Some information was missing from the records.

From this study the interaction of the healthcare providers with the clients and the quality of health information on anaemia passed to the clients was not determined. What the health care providers failed to record appropriately could not be assessed either. These aspects are important in ANC and would have added more information on the quality of the ANC.

Expected Application Of Results

This study is part of fulfilment of the requirements for a masters degree in medicine in Obstetrics & Gynaecology at the university of Nairobi.

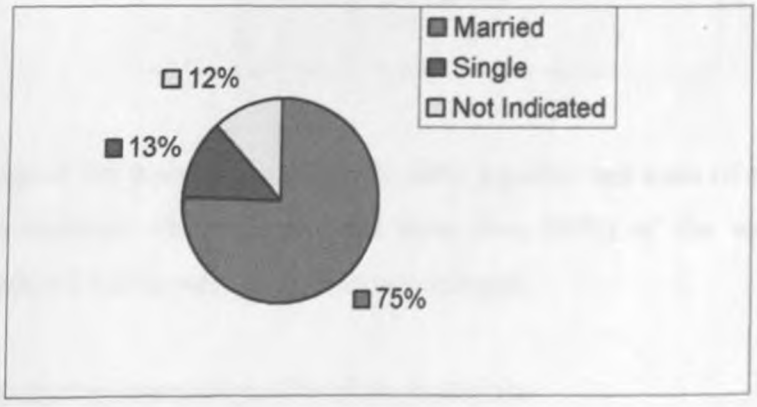
The results may be used to improve the quality of the services offered at the city council clinics and Pumwani Maternity Hospital

THE RESULTS

Social Demographic Profile

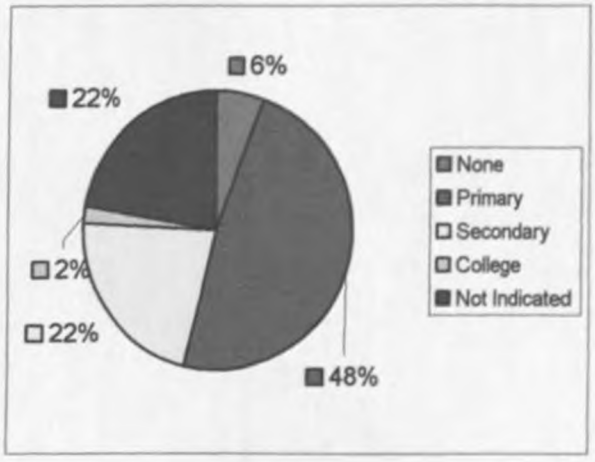
Most of the women requested were very willing to participate in the study. The number of women who participated was 312.The average age of the women in the study population was 23 with a range of 13 to 38 years and the modal age was 25. As many as 23% were below 20 years of age. Only 3 mothers were above 35 years of age. Thirty-seven (12%) of the women did not have their marital status indicated. Of the 275 women whose marital status was indicated 236(86%) married while 39 (14%) were single.

Chart 1: showing the marital status of the population



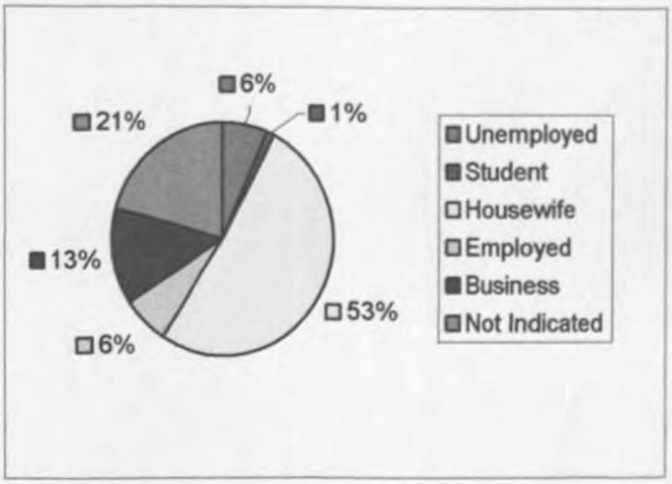
Two hundred and forty three of the women had their education level indicated. The majority of them (150, 62%) had only a primary level education, 69 (28 %) had secondary level education. 6 (3%) had gone up to college while 18 (8%) had no formal education.

Chart 2: showing the Education profile of the population



The occupation of 248 women was indicated. Only a quarter had some of employment or did some business. One hundred and sixty two (65%) of the women were housewives while 20 (8%) were single and unemployed.

Chart 3: showing the economic profile of the population



Prevalence of Anaemia

Of the 312 women in the study 202 (65%) of these women had haemoglobin (Hb) estimation done and 19 (9.4%) of them were found to be anaemic. Fifty-two (26%) of the women had Hb more than 10 g/dl but less than 11g/dl. One hundred and ten (35%) of the mothers did not have Hb estimation during their antenatal clinic. Only 4 (21%) of the 19 anaemic women had a repeat of Hb estimation. Seventy one percent of the mothers did not have examination for pallor.

Table 1: Haemoglobin estimation in the study population

	No of women	%
Done	202	65
Not done	110	35
Total	312	100

Chart 4: Graph showing haemoglobin levels of where estimation was done

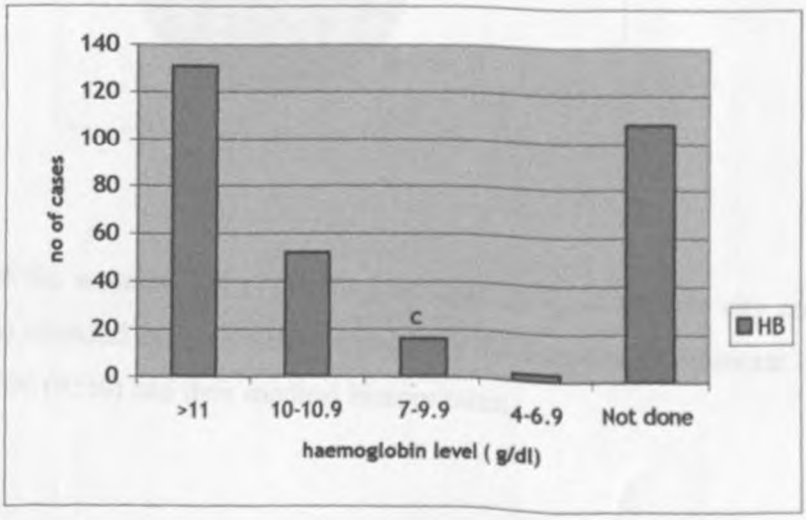
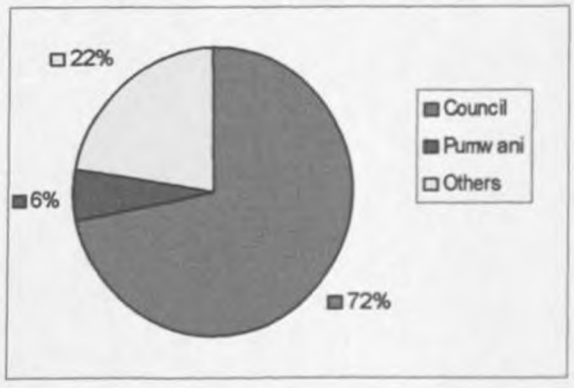


Table 2: Examination for pallor among the study subjects

	No of women	%
Done	90	29
Not done	222	71
Total	312	100

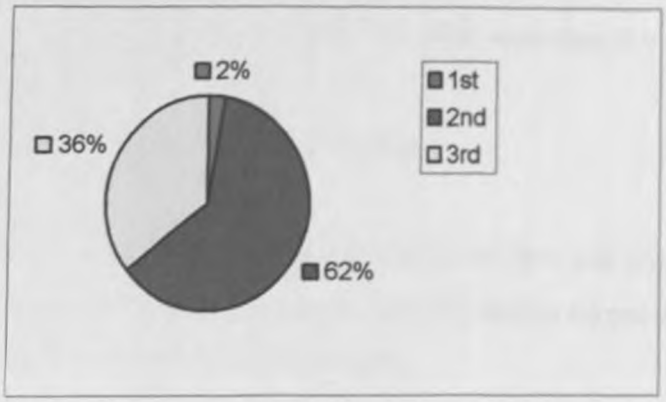
Examination of pallor was not done for most of the women (71%). Fifty-four women (17%) did not have Hb estimation and did not have examination for pallor done.

Chart 5: showing where the women attended ANC



The majority of the women, 224 (72%) had attended ANC in various city council clinics, 70(22%) attended in private clinics and only 6% attended in Pumwani. Most of the women, 296 (95%) had their medical history taken.

Chart 6: showing the gestation at booking by trimester



Only 8 (3%) women booked the ANC in the first trimester. The majority, 193 (62%) booked in the second trimester and 111 (36%) booked in the third trimester. Hundred and sixty-eight (54%) of the women made between 4 and 8 visits while 128 (41%) made less than 4 visits. Only 16 (5%) made 9 visits or more.

Table 3: Showing the number of visits made and respective Hb

No of Visits	No of women		Hb <10		Hb >10		Hb not done	
	n	%	n	%	n	%	n	%
<4	128	41%	6	5	59	46	63	49
4-8	168	54%	11	7	113	67	44	26
>9	16	5%	2	13	13	80	1	7
Total	312	100%	19	6	185	59	108	35

Nearly half (49%) of those who made less than 4 visits did not have Hb estimation compared to 26% of those who made more than 4 visits. Of the 19 with anaemia 58% made 4-8 visits, 32% made less than 4 visits and 10% made more than 9 visits.

Social Demographic Profile of Those With Anaemia

Nineteen women were found to have anaemia. Nine (60%) of them had primary education level, 4 (27%) been to secondary school, 2 (13%) had no formal education. The level of education was not indicated for 4 of them.

Table 4: showing the level of education of the women with anaemia and those without

Level of education	None	Primary	Secondary	College	Not Indicated	Total
Those with anaemia	2	9	4	0	4	19
%	11	47	21	0	21	100
Without anaemia	9	80	47	5	42	183
%	4	44	26	3	23	100

Comparing the level of education of those with anaemia and those without, there was no relationship between anaemia and the level of education ($p=0.43$). The data may have been too small to have enough power to show a difference.

Of the 19 anaemic women 16 (84%) were married and 3 (16%) were single. Thirteen (68%) were housewives while 3 (16%) were single. Of the 110 women whose haemoglobin was not done 82% had no source of income. Only 3 (16%) women had some source of income compared to 28% with source of income among those without anaemia. There was however no association (p=0.563) between having a source of income and anaemia.

Chart 7: Economic profile of those with anaemia

Most, 10 (53%) of the women found to have anaemia were primigravida, 3(16%) were grand multipara while 6(31%) had parity between 2-4.However there was no association between parity and anaemia statistically. (P=0.100)

Table 5: parity of women with anaemia

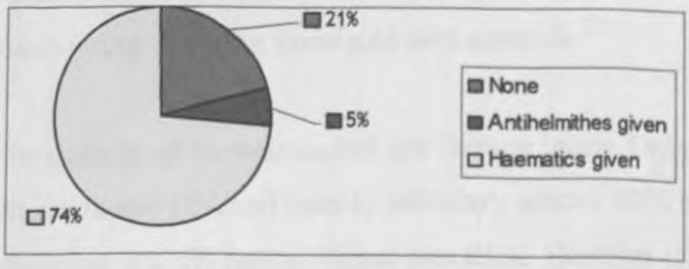
	Primigravida	Para 2-4	Para 5 and above
Cases	10	3	6
%	53%	16%	31%

Of the 19 women with anaemia only 1 (5%) of these women had booked ANC in the first trimester, 12 (63%) booked in the second trimester while the majority, 6 (32%) booked in the third trimester. Of those whose haemoglobin was not done, only 2% booked ANC in the first trimester, 51% booked in the second trimester while 47% booked in the third trimester.

The majority, 14 (74%) had attended ANC in city council clinics, 3 (16%) attended in Pumwani and 2(10%) in private clinics. Six of the women had made less than 4 ANC visits during the pregnancy. Eleven of them made between 4-8 visits while 2 of them made 9 or more visits.

For the women found to have Hb less than 10g/dl, in 4(21%) no action was taken, for 14 (74%) haematinics were given and 1(5%) was given antihelminthes but not haematinics and investigations were not done. Only 4 (21%) of these women had a repeat Hb estimation done. Of the 52 mothers who had Hb between 10 and 11 only 19 of them were on haematinics. In total 85 out of 312 mothers were on haematinics.

Chart 8: Action taken for those with anaemia



Discussion

The prevalence of anaemia in pregnancy among those tested was 9.4%. This is higher than the 5.4% reported by Mati (1971) and 4.3% reported by Mumia (1981).[36,37] However it is lower than the 18.1% prevalence reported among the Maasais in the Western Rift valley by Miyoro .[38] The prevalence of anaemia has thus remained.

The average age of the women in the study population was 23 with a range of 13 to 38 years. Most of the mothers were young with only 3 being above 35 years of age. As many as a fifth (23%) of the mothers were teenagers. These findings are close to those of the Nairobi birth survey and a study in Pumwani where 18.4% and19.4% of the mothers respectively were teenagers. This is unlike the developed world where mothers tend to be more advanced in age. In the British birth survey teenagers were only 9.8%. [5,39]

Teenage pregnancies are known to be associated with increased risks including poor ANC attendance and iron deficiency anaemia.[37] More of the teenage mothers (48% compared to 36% of the older women for the rest) did not have haemoglobin estimation. Of those who had estimation done 12% of them had anaemia compared to 8.9% among the older women. Thus for those who had their Hb done, the prevalence of anaemia appears to be higher among the teenagers than in the whole population. These findings correlate with those of the Nairobi Birth Survey and Ademowore who found young age to be associated with anaemia.[7,25]

The majority of the women had low literacy levels. Only 3% had some college education and 28% had been to secondary school, 62% had only a primary level education, and 8% had no formal education. However the level of education is higher than the findings of the Nairobi birth survey where 12% had no formal education and only 10% had completed secondary school.[5]

Most of the women (86%) were married while 14% were single. This is very close to the findings of the Nairobi birth where 15% of the mothers were single and the Pumwani study in where 85% were married and 15% were single. [5,39] T he marital status of those with anaemia was similar to that of the rest of the population (79% of them were married and 21% were single).

Many women had no independent source of income. Only a quarter had some form of employment or did some business, 65% of the women were housewives while 8% were single and unemployed. Of the women found to have anaemia only 19% women had some source of income. Three quarters were housewives, 6% were single and unemployed. However there was no association between having a source of income and anaemia (p=0.563). This might due to the fact that even the women who are earning have very low in come that does not improve their status significantly.

The primigravida were the majority (53%) in this study, only 3% were para 5 and above. This may be due to the fact that many mothers were young and were therefore beginning their families. The low number of grand multiparas may be due to the reduced ANC attendance as the parity increases.[45] Those with anaemia 53% were primigravida, 35% were para 1-4 were 35% and 18% were para 5 and above. There was no association between parity and anaemia statistically (p=0.10). This is unlike the findings of Fomulu that increasing parity was associated with decreasing incidence of anemia.[41]This might be due to the fact that very few women in this study were grand multiparas. Nine (60%) of them had primary education level, 27% been to secondary school, 13% had no formal education. However there was no relationship between anaemia and the level of education (p=0.43). In this study the education and social economic profile of those with anaemia was not significantly different with that of those without anaemia. This might be due to the fact that the population served by this hospital is very similar. It could also be due to the small number of those with anaemia, the data may have been too small to have enough power to show a difference.

One of the aims of ANC is to look out for anaemia and correct or prevent its development. Screening for anaemia appears not to be effective. A significant proportion (36%)of the mothers had attended the ANC but no haemoglobin estimation was done. Only 27% of those found to have anaemia had a repeat Hb test to check their progress. After the first screening is done in the first visit, ideally in the 1st trimester, a repeat should be done in the eighth month of pregnancy but clinical assessment of anaemia should be done in every visit.

Various factors may have contributed to failure of Hb testing. Financial constraints may have played an important role since it is notable that 82% of those whose Hb was

not done, had no source of income. In the clinic the mothers are required to pay for the various tests including the haemoglobin estimation. This may explain why many mothers attended the clinic but never had the Hb done. Studies have shown that although imperfect, use of pallor to screen and treat severe anaemia by primary care providers is feasible and worthwhile. Usually, the majority of persons with severe anaemia will be detected at practically no cost. Where the laboratory accessibility is limited as it was in this case, then clinical screening should be used. [33] Pallor screening was done in only a quarter of the mothers. Of these about 6% were found to be pale, but in half of them no further action was taken.

Another factor that could have contributed to failure of haemoglobin screening may have been late booking. Many women in Kenya have not appreciated the need to begin ANC early yet ideally care should start before conception. [2] Only 2% of the women booked ANC in the first trimester, majority booked in the second trimester (62%) and as many as 36% booked in the third trimester. This is close to findings of other previous local studies. In KNH only 4.7% had booked ANC in the first trimester and over 68% had initiated ANC at the end of second trimester. In Migori district, only 8% of the mothers initiated ANC visits in the first trimester while at Pumwani maternity hospital only 25% of the mothers booked ANC before the 25th week. [23,42,43] Of those found to have anaemia only 5% had booked the clinic in the first trimester, 32% booked in the third trimester. In case of such late booking, there may not be enough time during the pregnancy to correct anaemia with haematinics. Lack of finances may contribute to late booking since the mothers may lack the fee needed. Late booking may also mean fewer visits and hence reduced opportunity to do the test. Nearly half (49%) of those who made less than 4 visits did not have Hb estimation compared to 26% of those who made more than 4 visits.

It is notable that majority of the women whose Hb was not done (77%) had primary or no formal education, 21% had secondary school education while only 2% of them had been to college. It is possible that the more educated women understand the need for screening tests more than the less educated ones.

Health care providers might not have emphasized the need to do the test. This study did not address the performance of the health workers in counselling the mothers. Some studies suggest there is poor performance of even well trained staff in health facilities. [41] Pumwani receives many patients from other clinics. In this study only 5% of the mothers had attended ANC in Pumwani. This is a challenge to ensuring good ANC. The health care providers in the peripheral clinics would need to be involved.

In some cases Hb is done and found to be low but no action is taken. Among those with anaemia in 21% no action was taken and 5% antihelminthes were given but no haematinics. These findings are close to what Kinuthia found at KNH where of those found to have a low haemoglobin level, 28% had no intervention.[42] This calls for better ANC provision on the part of the health care providers.

Only 27% of all the mothers received supplementation. This is less than the figure reported by the Kenya Demographic and Health Survey which reported that 9 out of 10 of the mothers had reported to a health worker at least once for ANC and 46% received iron supplementation in pregnancy.[19] Merits of routine iron supplementation in pregnancy have been questioned by some in the developed world, [5] however there is there is no evidence against it in areas with high incidence of iron deficiency anaemia. It is therefore recommended in the developing countries since the prevalence of anaemia is still high in order to avoid the hazards of anaemia. Routine iron supplementation results in reduced prevalence of low pre-delivery haemoglobin.[45] Routine folate supplementation is recommended in areas where megaloblastic anaemia is common such as in malaria endemic regions.[46] Though Nairobi may not be a malaria endemic area, malaria is fairly common and some mothers may be from parts of Kenya where malaria is endemic. Folate supplementation is thus desirable. Of the 110 women whose Hb was not done only 21 received routine supplementation. Many women (25%) had haemoglobin more than 10 g/dl but less than 11g/dl and only 37% of these received haematinics. Considering the high demands of pregnancy women with such borderline levels of haemoglobin are at increased risk of developing anaemia.[18,20] These women along with those who did not have the Hb done, may have benefited from supplementation.

Amongst those with anaemia 58% had had made between 4 and 8 visits, 10% had made 9 or more visits while 32% had made only 3 or less visits. This means that despite the women making several visits to the clinic some remained anaemic until delivery. These are very close to other local studies that found that a significant number of women attended ANC but did not have appropriate care provided.[43,41,43] Studies have shown that the numbers of visits made by the mother are not the most important predictors of the pregnancy outcome but rather the care given during each visit. It is argued that fewer visits will avail more time and resources for better ANC. Reduced visits can be successfully introduced without increase in perinatal morbidity and mortality [47,48] The WHO has now recommended 4 visits well distributed during pregnancy in which focused care will be given. [48,49]

Conclusions

Of the women who delivered in Pumwani 65% of mothers attended ANC but did not have haemoglobin estimation by the time of delivery. Only 29% of the women had examination for pallor. Only 5% of the mothers had routine ANC at Pumwani hospital.

Of the mothers found to be anaemic 26% did not have appropriate intervention to correct the anaemia. Only 27% of all the mothers received haematinics.

Many mothers booked ANC late. Only 2% booked in the first trimester while 36% booked in the third trimester.

Record keeping by the health care workers in the ANC was poor, important information such as the age and the marital profile was missing in some antenatal cards.

Recommendations

1. More effort needs to be put in the peripheral clinics for;
    - Good record keeping
    - Clinical examination
    - Laboratory testing
    - Intervention
2. There is need to determine factors affecting the testing for anaemia.
3. There is need for women to book ANC at an early gestation to allow for investigations and management of anaemia.

REFERENCES

1. Lewis T.L.T, chamberlain G.V.P. Obstetrics by Ten Teacher 15th edition,1990 EL BS 2: 39.

2. Martin L.P, Taylor CM. Normal pregnancy and Prenatal Care in Current Obstetric & Gynaecologic diagnosis and treatment. Appleton & Lange 8th edition 1994, 9:191-200.

3. Treacy A.O. Donovan M, et al. Perinatal outcome in unbooked women at Rotinda Hospital, *Irish Medical Journal 15(20; 44-7, 2002 Feb.*

4. Cunningham G, Gant F, et al. Prenatal care in Williams Obstetrics Mc Gray Hill 21st Edition. 2001 10:222

5 Mati S.K.G., Aggarwal VP, Lucas S Et al. The Nairobi Birth Survey design; The Population outline results. *J Obstet, Gynecol. E. Cent Afri 1; 32, 1982.* Mc Gray Hill 21st Edition. 2001

6. Kenya Demographic Health Survey 2003.Preliminary Report; CBS, MOH Kenya, KEMRI, CDC.

7. Alexander G.R Kotelchuck M. Department of maternal and child health University of Alabama, Burmingham, Alabama USA assessing the role and effectiveness of prenatal care. Public health reports 116(4);306-16, Aug. 2001.

8. Koroukian SM, Rimm AA. Department of Epidemiology and Biostatistics School of Medicine, Western Reserve University, Devad, USA. Adequacy of prenatal care utilization. *J clin epid 55(3) 296-305, 2002 Mar*ch.

9. Krueger pm Scholl 10 University of Medicine and Dentistry Stratford USA. Adequacy of prenatal care and pregnancy outcome Journal of American Osteopathic Association 100(8);485-92, 2000 Aug.

10. Questioning effective antenatal care, safe motherhood newsletter, Issue 29 2002(1)

11. Revised estimates 1990 estimates of maternity and infant mortality. A new approach by WHO & UNICEF Geneva, 1999 WHO/RHRO1.9

12. London on Maternal and Infant Mortality Society for the Social history of Medicine *J Social Science and medicine* 1991 *Aug; 10(4): 125-96*

13. Review on effectiveness of antenatal care in preventing maternal mortality and serious morbidity, vol 15, supplement Jan 1 2001

14. Mafany M. Maternal Mortality in Cameroon Mmed Thesis University of Nairobi, 1983.

15. Makhoha A.E. Medicolegal and social demographic factors associated with maternal mortality at Kenyatta National Hospital, Kenya. *J obstet Gynaecol. East and centr Afri vol 9 page 3 1991.*

16. Obore S. A review of maternal in KNH 1995-1991. Mmed Thesis University of Nairobi, 2001.

17. National guidelines for Quality Obstetrics and Perinatal mortality by the Ministry of Health, Division of Reproductive Health, November 2004

18 Manoj K B, Perloff D; Cardiac Pulmonary Renal & Urinary Tract Disorders in Pregnancy in Current Obstetric & Gynecologic Diagnosis and Treatment. Appleton & Lange 8th edition 1994 22:448-452

19. Focussed Antenatal Care and Malaria in Pregnancy, Orientation package by the Ministry of Health. MOH-DRH/DOMC/JHPIEGO August 2002

20 Cunningham G, Leveno G, et al, Hematological disorders in Williams Obstetrics 21st edition Mc Gray Hill 2001 pg 1308

21 Galloway R, Dusch E, Elder L, et al. *J Social Science and medicine. 2002 Aug;55(4):529-44.*

22. Gebbie DMA obstetrics and Gynaecology in Health and disease in Kenya PP 485- 498 Vogel L.C(Eds) E.A.L.B, Nairobi, 1974.

23. Ademowore D, Wale T. Anaemia in pregnancy – A ten year review J. Obst. Gynaecogy East. Central Africa, 7;59;1988.

24. Guidotti RJ; Anaemia in pregnancy in developing countries. *British Journal Of Obstetrics And Gynaecology. 2000 Apr;107(4):437-8.*

25. Viteri FE; The consequences of iron deficiency and anaemia in pregnancy. Adv. Exp. *Med. Biol 325;127, 1994.*

26. Maternal mortality in 1995. Estimates developed by WHO, UNICEF, UNFPA 2001WHO/RHRO1.9

27. Ngoka W. M Bansal YP maternal mortality, Pumwani Hospital 1975-1984. *E.A med journ 64 No. 4 Pg. 277-285, 1987.*

28 Solomon M. quality of antenatal care and outcome of pregnancy at Coast Provincial Hospital. Mmed thesis University of Nairobi, 1992

29 Johnson S.W.L, Jo O.A Amniotic fluid oxygen tensions in severe maternal anaemia *AM Journ obstet Gynaecol. 97; 499-506, 1967.*

30 MC fee J.G. Anaemia in pregnancy; A reappraisal Obstetric Gynaelogy Survey 1973, 28(11); 769.

31 Scanlon KS, YIPR, Schieve CA. High and low haemoglobin levels during pregnancy; Differential risk for preterm birth and small for gestation age *J Obstet. Gynae. 96;741, 2000.*

32 Gomber S, Aggarwal K N, Mahajan C, Aggarwal N Impact of daily versus weekly haematic supplementation on anaemia in pregnant women. *Indian Paediatrics. 2002 Apr, 39 (4):339-46.*

33 Stoltzfus RJ; Edward-Raj A; Dreyfuss ML; Albonico M; Montresor A Clinical pallor is useful to detect severe anaemia in populations where anaemia is Prevalent and severe. *Journal of Nutrition. 1999 Sep;129(9):1675-81.*

34 Dusch E; Galloway R et al. Clinical screening may be a cost-effective way to screen for severe anemia. [Unpublished] [1999]. 13 p. USAID Contract No. HRN-5966-C-3038-00

35 Prevention and management of severe anaemia in pregnancy: report of a technical working group. 20-22 May 1991. Geneva, World Health Organization, 1994, WHL/FHE/MSM/93.3

36 Mati S.K.G Hatimy A. The importance of anaemia in pregnancy in Nairobi and the role of malaria in the aetiology of Megaloblastic anaemia. *J.TNP med. Hyg.64;1-8, 1971*

37 Mumia anaemia in pregnancy. Mmed Thesis university of Nairobi, 1981

38 Miyoro Assessment of the magnitude and possible causes of anaemia in pregnancy among the Maasai women of western Rift Valley. Mmed Thesis UON, 2002

39 Kirwa P.K , Management of labour and delivery at Pumwani Maternity Hospital.

40 Muram D. Paediatric and adolescent Gynaecology. Current obstetric & Gynecologic Diagnosis and treatment. 31;659-660; 8th edition 1994. 31;659-660

41 Fomulu J; A 2 year Retrospective controlled Survey of maternal and foetal prognosis in anaemia of pregnancy at KNH. Mmed thesis, University of Nairobi, 1981

42 Kinuthia S. Assessment of quality and quantity of antenatal care services at KNH Mmed thesis, University of Nairobi, 2002.

43 Agullo D.O , Quality of antenatal care among women delivery in a Rural mission hospital in Kenya. Mmed thesis University of NRB. 1999.

44 Le Roux E, Pattison RC et al Does successful completion of the perinatal Education Programme result in improved obstetric care? SAfri Med J 1194; 88: 180-182.

45 Gonzalez- cossio T. Routine iron supplementation in pregnancy. The WHO Reproductive health library no. 7 update of software ltd, Oxford, 2004

31 Scanlon KS, YIPR, Schieve CA. High and low haemoglobin levels during pregnancy; Differential risk for preterm birth and small for gestation age *J Obstet. Gynae. 96;741, 2000.*

32 Gomber S, Aggarwal K N, Mahajan C, Aggarwal N Impact of daily versus weekly haematic supplementation on anaemia in pregnant women. *Indian Paediatrics. 2002 Apr, 39 (4):339-46.*

33 Stoltzfus RJ; Edward-Raj A; Dreyfuss ML; Albonico M; Montresor A Clinical pallor is useful to detect severe anaemia in populations where anaemia is Prevalent and severe. *Journal of Nutrition. 1999 Sep;129(9):1675-81.*

34 Dusch E; Galloway R et al. Clinical screening may be a cost-effective way to screen for severe anemia. [Unpublished] [1999]. 13 p. USAID Contract No. HRN-5966-C-3038-00

35 Prevention and management of severe anaemia in pregnancy: report of a technical working group. 20-22 May 1991. Geneva, World Health Organization, 1994, WHL/FHE/MSM/93.3

36 Mati S.K.G Hatimy A. The importance of anaemia in pregnancy in Nairobi and the role of malaria in the aetiology of Megaloblastic anaemia. *J.TNP med. Hyg.64;1-8, 1971*

37 Mumia anaemia in pregnancy. Mmed Thesis university of Nairobi, 1981

38 Miyoro Assessment of the magnitude and possible causes of anaemia in pregnancy among the Maasai women of western Rift Valley. Mmed Thesis UON, 2002

39 Kirwa P.K , Management of labour and delivery at Pumwani Maternity Hospital.

40 Muram D. Paediatric and adolescent Gynaecology. Current obstetric & Gynecologic Diagnosis and treatment. 31;659-660; 8th edition 1994. 31;659-660

41 Fomulu J; A 2 year Retrospective controlled Survey of maternal and foetal prognosis in anaemia of pregnancy at KNH. Mmed thesis, University of Nairobi, 1981

42 Kinuthia S. Assessment of quality and quantity of antenatal care services at KNH Mmed thesis, University of Nairobi, 2002.

43 Agullo D.O , Quality of antenatal care among women delivery in a Rural mission hospital in Kenya. Mmed thesis University of NRB. 1999.

44 Le Roux E, Pattison RC et al Does successful completion of the perinatal Education Programme result in improved obstetric care? SAfri Med J 1194; 88: 180-182.

45 Gonzalez- cossio T. Routine iron supplementation in pregnancy. The WHO Reproductive health library no. 7 update of software ltd, Oxford, 2004

46 Fikree FF. Routine folate supplementation in pregnancy. The WHO Reproductive health library no. 7 update of software ltd, Oxford ,2004

47. Villar J. Carroli G et el. Patterns of antenatal care for low risk pregnancy, (Cochran review) In Cochrane library Issue 4,2003. Chichester, UK: John Wiley $ sons, ltd.

48. Munjanja P, Ginilla L, Nystrom. Randomised controlled trial of a reduced-visits programme of antenatal care in Zimbabwe. The Lancet 348; 9024 :364-9 1996

49. WHO Programme to map Best Reproductive HealthPractices. WHO Antenatal care ; Randomised Trial: Manual for the implementation of the New model: The WHO RHL no 4 Geneva, 2001

Gynaecology Case 1

INCOMPLETE ABORTION – VACUUM ASPIRATION DONE

Name	M. N.	I. P. No.	0989332
Age	23	DOA	22.3.02
Parity	1 + 0	DOD	23.3.02

Presenting complain

The patient was admitted with complains of lower abdominal pain and per vagina bleeding for two days.

History of presenting complains

She had been well until two days prior to admission when she developed the above complains. She noticed bleeding, which was initially mild and not associated with any pain. Later in the day she developed lower abdominal pain that was colicky in nature. The bleeding increased coming out in clots. She had changed eight soaked pads the day prior to admission.

Obstetric and gynaecological history

She had attained menarche at 14 years. The flow lasted 2 – 3 days in a cycle of 28 – 30 days. Her last menstrual period was on 4.1.02 giving an amenorrhoea of eleven weeks. She had not attended any antenatal clinic. She had done a pregnancy test that was positive 2 weeks prior to the admission. She was para 1+0. Her last delivery was in 1999 to a live male infant who was alive and well. She had used combine oral pills for contraception and had stopped about 4 months earlier since she desired another baby.

Past medical history

She had never been hospitalised again. She did not have any known clinic illness.

Family and social history

She was married since 1998 and was a nursery school teacher. She was living with her husband in Umoja. She did not smoke cigarettes or drink alcohol. Her mother was asthmatic but there was no other known chronic illness in the family.

Physical examination

She was in good general condition was not pale, had no oedema or lymphadenopathy. Her vital signs were normal; BP 120/80mhg, PR – 84/min, RR 18/min, temp 36.2°C.

Abdominal examination

Abdomen was scaphoid, moving with respiration. She had no areas of tenderness and no palpable masses.

Pelvic examination

The eternal genitalia was blood stained. The cervix was 2 cm dilated. The uterus was bulky and anterverted. The adrexae and the pouch of Douglas were non-tender and had no masses. There were blood clots in the vaginal canal.

Diagnosis

A diagnosis of an incomplete abortion was made.

Investigations

Packed cell volume – 30% Blood group – A +ve

Management

The findings were explained to her and the need for manual vacuum aspiration. She consented. In lithotomy position vulval vaginal toilet was done and a digital examination done that confirmed the earlier findings. A cuscos speculum was used to expose the cervix. The cervix was stabilized with a teneculum and clots evacuated using a sponge holding forceps. Using a Karmans cannula size 12 manual vacuum aspiration was done. About 100 millilitres of products of conception were obtained. Ergometrine 0.5mg was given intramuscularly. The uterus was contracted and haemostasis was achieved. When she was stable a counsellor talked with her for psychological support and she was advised on contraception. She chose the combined pill. The patient was then discharged home on doxycycline, flagyl and haematinics.

Follow up

The patient was reviewed in the GOPC after 2 weeks. She had done well and had no complains.

Discussion

M N was admitted with incomplete abortion, manual vacuum extraction was done with good results.

Spontaneous abortion is the most common complication of pregnancy and accounts for a high proportion of gynaecology consultations and hospital admissions. It can be traumatic and highly emotional for the woman and her partner. Current recommendations are that the term abortion be replaced with miscarriage [1]. The discussed had desired a baby but when she conceived she had a spontaneous abortion. Counselling was done to help her cope with the misfortune.

The world health organisation defines abortion as the expulsion of a foetus or embryo weighing 500g or less or a gestational limit of less than 22 completed weeks. The abortion may be complete or incomplete. The other forms of spontaneous abortion include threatened, inevitable, missed, septic abortion or blighted ovum.

Threatened abortion is when there is uterine bleeding in early pregnancy but the cervical os is closed. It may progress to inevitable abortion. Inevitable abortion refers to the situation where the cervical os is open but the products of conception are not yet expelled. In complete abortion all the products of conception have been expelled. Incomplete abortion means part of the product of conception are not yet expelled. Heavy bleeding and abdominal pain usually with a dilated cervix accompany it. In such circumstances it is usually important to evacuate the uterus to arrest the bleeding and minimize the chances of infection developing. Septic abortion is one where infection has already complicated the abortion. Missed abortion refers to the demise of the embryo or foetus that is retained in the uterus without symptoms.

The true incidence of abortion is not known. Post implantation and biochemical pregnancy loss rates are estimated to be 30% while the clinically recognized pregnancy loses are 10 – 15%. [2] Total pregnancy loss rates have been estimated at 78%. [3] In KNH abortions constitute about 51% of all emergency gynaecological admissions.[4] About 80% of all the abortions occur before 12 weeks gestation. [5] The patient discussed had a spontaneous abortion at 11 weeks gestation.

The leading cause of spontaneous abortion in the first trimester is foetal chromosomal abnormalities. Trisomies are the leading foetal chromosomal abnormalities being found in 30% of all abortions and 60% of the chromosomal abnormal abortuses. [1] Other foetal malformations other than those caused by chromosomal abnormalities are also associated with increased risk of abortions. Other causes of abortions include uterine anomalies, infections, endocrine factors, autoimmune diseases and idiopathic factors. Increasing maternal age and history of previous pregnancy loss are risk factor. The patient presented had no obvious cause or risk factors for abortion.

The diagnosis of spontaneous incomplete abortion can be made from the history and physical examination. Heavy bleeding and abdominal cramps usually accompany it. The cervical os will be open and products of conception may be felt. A pelvic ultrasound reveals retained products of conception and rules out other abnormalities such as ectopic pregnancy. A complete blood count is important to ascertain the haemoglobin level. The white blood cell count maybe raised in case of associated sepsis. The patient discussed had the diagnosis made clinically. A packed cell volume was obtained since in the set up it was readily available.

One the diagnosis is made, it is important to evacuate the uterus in order to arrest bleeding and to reduce the risk of infection. Manual vacuum aspiration using a syringe and a cannula or sharp curettage can be done. The patient discussed was done evacuation by use of karma's syringe and cannula. The patient was then put on oral antibiotics for prophylaxis. Patients whose blood group is rhesus negative should receive anti D prophylaxis. Our patient did not need anti D.

Contraception should be provided to women after abortion. This gives them time to recover physically and emotionally before attempting another pregnancy. N M was counselled on the need for contraception and chose the combined pill since she had used it previously.

References

1. Buckett W, Regan L, Sporadic and recurrent miscarriage. Gynaecology by Shaw 3rd edition. Churchill Livingstone 2003, 24: 343- 355.
2. Hertig AT, Livingstone RG, Spontaneous threatened and habitual abortion; their pathogenesis and treatment, new England journal of medicine 230; 797 – 806 1944
3. Roberts CJ, Lowe DB where have all the conceptions gone? Lancet; 498 1975
4. Mutungi A.K contraceptive acceptance and continuation in women managed for incomplete abortion in KNH Mmed thesis, university of Nairobi, 1990.
5. Wilcox A J, Weinberg CR O'connor, Incidence of early pregnancy loss. New England journal of medicine 1988; 519; 189.

Gynaecology Case 2

SECOND DEGREE UTERINE PROLAPSE – TOTAL VAGINAL HYSTERECTOMY AND COLPORRPHY

Name	P. M.	I. P. No.	0920347
Age	65	DOA	13.04.02
Parity	9 + 0	DOD	20.04.02

Presenting complaint

The patient was admitted through the GOPC with complains of feeling of something coming down the birth canal for 8 months.

History of presenting complaint

She had been well until 8 months prior to admission when she started feeling something coming down in the birth canal. Initially the feeling came when she coughed or strained to pass stool. Later she started having incomplete emptying of the bladder. She was forced to use her fingers to push the mass up for complete emptying. She did not have associated urgency or dysuria. She had no per vagina discharge or bleeding. She sometimes strained when passing stool but had no constipation.

Past medical history

She had only been admitted in hospital during deliveries. She had no known chronic illness or allergy.

Obstetric and Gynaecological History

She was Para 9 + 0. All the deliveries were vaginal, the last one being in 1980. She had been postmenopausal for 20 years. She had never used any contraception and she was not on hormone replacement therapy.

Family and social history

She was married a housewife living with her husband in Murang'a. They both worked in the farm. She did not drink alcohol or smoke cigarettes. There was no family history of any chronic illness.

Physical examination

She was an elderly lady in good general condition, not pale, jaundiced, or dehydrated. She had no lymphadenopathy. Her vital signs were as follows: -

BP	140/90mmgh	RR	20/minute
PR	80/minute	Temp	36.7°c

The cardiovascular, respiratory and the central nervous systems were all within normal.

Abdominal examination

The abdomen was scaphoid moving with respiration. It was soft, with no areas of tenderness or palpable masses.

Pelvic examination

The external genitalia was atrophic with scanty greyish pubic hair. There was a mass bulging in the vaginal canal. On coughing it protruded further to reach the hymenal ring. There was a bulge in the anterior vaginal wall that protruded further on coughing. The surface of the protruding mass looked healthy grossly and was not ulcerated. There was no associated stress incontinence of urine. Bimanual examination was done; the uterus was small but mobile. The pouch of Douglas and adnexae felt normal. There was no tenderness.

Diagnosis

A diagnosis of second-degree uterine prolapse with a cystocoele was made. This is equivalent to Pelvic Organ Prolapse Quantative scoring system (POP Q) class II.

Investigations

HB	13.7g/dl	WBC	$8.2 \times 10^9/_L$
Na^+	142 mmol/L	K^+	3.8 mmol/L
Platelets	$340 \times 10^9/_L$	Urine M/C/S	no abnormality

Management

The nature of her illness was explained to the patient. Since she was in good general health and had completed her family, a total vaginal hysterectomy accompanied by repair of the cystocoele was chosen. She gave an informed written consent. Blood was taken for grouping and cross matching and units of blood made available. In the eve of the operation she was done an enema with a repeat in the morning of the operation. She was fasted from midnight. Half an hour before going to theatre, she was premeditated with 0.6mg of atropine intramuscularly.

In theatre, she was put under general anaesthesia. In lithotomy position vulvo vaginal toilet was done and painting with iodine solution done. Draping was done followed by aseptic catheterisation. Clear urine was drained. Examination under anaesthesia confirmed the earlier findings.

To enhance exposure, the labia minora were stitched to the upper medial aspect of the thigh on either side. An auvard speculum was then inserted into the vagina posteriorly. The cervix was grasped using a volsellum forceps. A uterine sound was introduced into the uterine cavity, which was found to be 6 cm long. Jungle juice (solution of adrenaline, lignocaine and saline) was injected beneath the vaginal mucosa around the cervix. This would assist in defining the facial layers as well as enhance a non-vascular field of operation due to vasoconstriction.

The gentle traction was applied on the cervix as 2 assistants maintained exposure using vaginal retractors and an anterior retractor to protect the bladder as well. The cervix was circumferentially incised at its junction with the vaginal mucosa down to the level of the pubovesico cervical fascia. The bladder was advanced off the cervix by blunt dissection along the fascia plane. The posterior vaginal mucosa was dissected of the cervix similarly. The blunt dissection was done up to the level of the uterosacral ligaments. The posterior cul-de-sac was identified and incision to open it made using scissors, thus exposing the uterosacral ligament. The uterosacral ligament were grasped and clamped, cut and ligated bilaterally. Next the cardinal ligaments followed by the uterine vessels were systematically clamped cut and ligated bilaterally.

The anterior cul-de-sac was then entered by a combination of blunt and sharp dissection. The remaining portion of the broad ligament was clamped, cut, and ligated

in pedicles bilaterally. Next the round ligament, utero-ovarian ligament and the fallopian tubes were identified clumped and ligated. The uterus was then removed.
The ovaries and the tubes were carefully inspected and did not appear suspicious. All the pedicles were inspected to confirm haemostasis had been achieved. The peritoneal cavity was closed with a running stitch, incorporating the cardinal and uterosacral ligament pedicles to offer support to the vaginal vault. The broad ligament pedicles were approximated at the midline. Anterior colporrhaphy was then completed. Making a midline incision on the anterior vaginal wall. The vaginal mucosa was dissected away leaving the fascia on the bladder. Placation sutures were then applied. The normal urethrovesical anatomy was restored as much as possible. Redundant vaginal mucosa was excised and the edges approximated.
A vaginal pack was inserted into the vagina and was for removal after 6 hours. A urethra catheter was left in the bladder to be removed on the third day. Estimated blood loss was 600mls and transfusion was not necessary. The uterus was sent for histopathological examination.

Postoperative care

The patient was observed till she was awake. She received intravenous fluids and intravenous crystalline penicillin, gentamycin and flagyl for 48 hours. The following day after surgery, the vaginal pack was removed. She was started on oral sips and graduated to light diet. She received pethidine 50mg 6 hourly for pain control in the first 36 hours. She was started on oral medication, Amoxicillin, metronidazole and mefanamic acid on the second postoperative day.

She recovered well. On the fourth postoperative day she was allowed home. She was to come again for review in the GOPC after 4 weeks.

Follow up

The patient came for review after 4 weeks. She did not have any complains. The vaginal vault had healed well. The histology specimen did not reveal abnormalities except for features of cervicitis. She was released from the clinic to come again when necessary.

Discussion

The patient presented was a 65 years old lady who presented with symptoms of genital prolapse. Total vaginal hysterectomy with anterior colporrhaphy was done.

A prolapse refers to the protrusion of an organ or structure outside its normal anatomical boundaries. Prolapse of the pelvic organs can be classified according to the anatomical position. The pelvis can be divided into three compartments: -

Anterior – including the urethra and bladder (urethrocoele, cystocoele)

Middle – including the uterus or vault and the bowel or omentum (enterocoele)

Posterior – including the rectum (rectocoele), perineal hernia or rectal prolapse

There are several grading systems. The Baden & walker classification[1] is as follows:

Grade I descent of any organ to the vaginal mid plane

Grade II descent to the hymenal ring

Grade III descent to half way through the introitus

Grade IV complete eversion

The international continence society committee for standardization published the pelvic organ prolapse (POP) quantitative scouring system [2] as follows

0 no descent of pelvic organs during straining

I leading surface of the prolapse does not descend below 1 cm above the hymenal ring.

II leading edge of the prolapse extends from 1 cm above to 1cm below the hymenal ring.

III leading edge of the prolapse extends from 1cm beyond the hymenal ring but without complete vaginal eversion

IV the vaginal is completely everted.

P M had had genital prolapse grade II by the Baden & walker classification, which corresponded to POPQ class II.

It is estimated that about 50% parous women have prolapse, which is symptomatic in 20%.[3] Up to 51% of the women will have anterior vaginal wall prolapse. 27% posterior wall prolapse and 20% uterine or vault prolapse. Genital prolapse is relatively uncommon in east Africa as compared to Europe and United States. At Kenyatta National Hospital an incidence of 0.1% was reported.[4] PM had prolapse involving the anterior and middle compartments.

Multiple factors are involved in the aetiology of pelvic organ prolapse. The most significant factor is pregnancy, labour and vaginal deliveries. Over 90% of patients with prolapse are porous.[5] The pregnancy and delivery result in various degrees of damage to the pelvic support structures including the ligaments, fascia and muscles and their nerve supply. Electrophysiological studies confirm denervation changes in the pelvic floor and urinary and anal sphincters.[6] More damage occurs with prolonged labour, large foetal head and/or shoulders and difficult instrument deliveries. Significant individual variation in strength and recovery of pelvic tissues exists and some women experience no prolapse despite repeated vaginal deliveries. Our patient had 9 normal vaginal deliveries. The repeated deliveries might have caused damage to the pelvic support structures

Pelvic organ prolapse may result from inherent weakness in laxity of the corrective tissue support structures. This occurs in conditions such as type IV Ehlers – Danlos and marfans syndrome in which there is a lesser amount of type I collagen and greater amounts of type III collagen that is more elastic.[7] The tendency to prolapse is more likely to manifest after the menopause when ovarian steroid genesis ceases thus removing the oestrogen support of the genital support tissues. However no consistent trend has been noted between prolapse and menopausal age.[8] Raised intra abdominal pressures increase the likelihood of prolapse. Conditions such as chronic cough, obesity, pelvic tumour ascites, hard physical exertion heavy lifting, tight fitting corsets, and straining increase the intra abdominal pressure. Our patient worked in the farm, which meant heavy manual work. She reported occasional straining on passing stool.

Prolapse symptoms are very variable and do not necessarily reflect the degree of the prolapse. A large prolapse may be asymptomatic. The patient may feel a lump or a dragging below. An annoying protrusion at the vaginal introitus may be present. Discomfort and aching in the lower back is common. Intercourse may be uncomfortable. The symptoms maybe aggravated by standing, vigorous activity coughing, sneezing, or straining, and are eased by lying down. Anterior compartment prolapse maybe associated with urinary symptoms such as stress incontinence, incomplete emptying of the bladder and frequency. The middle compartment prolapse usually presents with blood strained purulent discharge from a protruding ulcerated cervix. Significant displacement of the uterus may lead to pelvic pain, back ache menstrual aberrations and infertility. Vague symptoms of vaginal discomfort may indicate the presence of an enterocoele or vault descent. [3,9] Rarely dehiscence of the vault can occur with acute pain and protrusion of small bowel at the vulva. The small bowel can strangulate leading to an acute abdominal emergency.[3] Posterior compartment prolapse may have bowel symptoms, such a as feeling of rectal fullness and incomplete evacuation. Tenesmus and constipation may be present with a rectocoele or an enterocoele. Mwalali at KNH found that most of the patients (97%) had presented with the feeling of something coming down the vagina. Urinary symptoms were present in 12% of the patients.[4] Our patient presented with feeling of something coming down in the birth canal and incomplete emptying of the bladder. She was forced to use her fingers to push the mass up for complete emptying and digitation.

Correct management of a patient with pelvic organ prolapse depends on a careful evaluation, her age, desire for preservation of reproductive function, desire for preservation of coital function, general medical status, previous attempts at surgical correction, the symptomatology and physical examination. Certain predisposing conditions such as chronic cough and constipation may be present may be present and will need evaluation. The patient's general health and the operative risk should be assessed. PM was in good general health and had completed her family.

The patient is examined in lithotomy or left lateral position using a Sims speculum. Stress incontinence is best demonstrated with a full bladder. The patient is asked to cough or bear down, any anterior vaginal wall prolapse or uterine descent is demonstrated by retracting the posterior vaginal wall. To demonstrate uterine descent, gentle traction maybe applied to the cervix after an appropriate explanation. To demonstrate enterocoele and rectocoele the anterior vaginal wall should be retracted. The examination is best done with the index finger in the rectum. A full bimanual pelvic examination is done to exclude pelvic masses. Sometimes the patient may need to stand up and strain to demonstrate the prolapse or incontinence. PM had obvious prolapse that increased when asked to cough.

Confirmation of the clinical assessment of prolapse can be made using techniques such as the ultrasound, pelvic floor fluoroscopy, magnetic resonance imaging and isotope defaecography. [3, 10, 11] Hysterography can reveal the position of the uterus. [9] When urinary symptoms are present urine microscopy, culture and sensitivity should be done. Aerodynamic studies may be indicated. Renal function evaluation including an ultrasound should be done where damage is suspected. This tends to occur in grade III and IV prolapse due to exteriorisation of the ureters leading to ureteric dilation and hydronephrosis. Our patient did not have renal complications.

Treatment options for pelvic organ prolapse depend on the degree of the prolapse and symptoms. A mild degree of uterovaginal prolapse is often associated with no symptoms or with mild symptoms. In such a case expectant management is appropriate. The patient should be informed of the conditions that should worsen the prolapse and advised to seek prompt management if any occurred. Postmenopausal women should be advised on the benefit of hormone replacement therapy. Perineal muscle exercises should be encouraged. Periodic examinations are indicated for evaluation of the prolapse and the symptoms.

For patients unfit for or who wish to defer surgery, vaginal pessaries can be used, they come in a variety of sizes and shapes and are individually fitted to each patient. Their complications include urinary incontinence vaginal discharge and vaginal ulceration. If neglected they can occasionally lead to vesicle or rectal fistula. The pessaries should fit comfortably and not cause pain. They should be changed every 6 – 12 months. Local oestrogen may be helpful in preventing the complications.

Prolapse surgery is directed towards restoring the anatomy, vaginal function and correcting the symptoms such as incontinence. Most patients with uterovaginal prolapse have a composite lesion including different degrees of uterine prolapse, cystocoele, stress incontinence, enterocoele, rectocoele and perineal relaxation. The best surgical management consists of a composite operation. Commonly a total vaginal hysterectomy accompanied by repair of actual or potential enterocoele, anterior colporrhaphy for the cystocoele and a posterior colpoperineorrhaphy for posterior compartment prolapse if present. If there is accompanying stress incontinence, a continence procedure should be done. It is best to defer surgical repair till the patient completes the family size to allow for definitive repair. [12] Our patient had uterine prolapse and cystocoele; a total vaginal hysterectomy with anterior coporrhaphy was done.

The surgeon should attempt to repair all the relaxations. Attempt should be made to recreate normal anatomy leaving a vagina of normal calibre and length. The vaginal vault should be suspended posteriorly over the elevator plate and not anteriorly. [13]
Uterine prolapse maybe managed by abdominal approach thus total abdominal hysterectomy. For success this would need to be combined with the vaginal repair of cystocoele and rectocoele.
In elderly patients who are not in very good general health, colpocleisis or colpoctomy may be chosen. These procedures are faster. [13]
PM was in good general health surgery was successfully done.

References

1. Baden W. Walker T, Genesis of the vaginal profile: a correlated classification of vaginal relaxation. Clinical obstetric and gynaecology 15;1048 –1054 1972
2. Bump R. Mattiasan A et al, The standardisation of terminology of female pelvic organ prolapse and pelvic floor dysfunction, American Journal of Obstetrics and Gynaecology 175: 10 – 17, 1996
3. Stuart L.S Vaginal prolapse in gynaecology by Robert Shaw, Patrick soutter stuart stantonton 3rd Edition Churchill Livingstone, 2003 55: 813 – 823
4. Mwalali P N A Retrospective study of genital prolapse at Kenyatta National Hospital. Mmed thesis, University of Nairobi 1982.
5. Carley M. Turner, Scott D et al obstetric history in women with surgically corrected adult urinary incontinence and pelvic organ prolapse. Journal of the American association of gynaecologic laporoscopists 6: 39-44 1999
6. Weidner A Barber M, Visco A et al; pelvic muscle electromyography of levator ani and external anal sphincter in nulliparous women and women with pelvic floor dysfunction. American journal of obstetrics and gynaecology 2000, 183: 1390 – 1401
7. Carley M. Schaffer J urinary incontinence and pelvic organ prolapse in women with Morfan or Ehlers Danlos Syndrome. American Journal of Obstetrics and gynaecology2000 182: 1021 – 1023
8. Versi E. Harvey M. Cardozol Urogenital Prolapse and atrophy at menopause: a prevalence study. International urogynecolocy journal 2001 12 ; 107 – 110
9. Clyde HD, Relaxation of pelvic supports in current obstetric & gynaecologic Diagnosis & Treatment. Allan Decherrey & Martin Perno; 8th Edition 1994 Appleton's Lange 41: 809 – 829
10. Stockers J, Halligans Bartram C Pelvic Floor imaging radiology 2000 218: 621 – 641
11. Lienemann A, Anthuber C, Baron A et al Diagnosing enterocoeles using dynamic magnetic resonance imaging. Diseases of the colon and rectum 2000 43; 205 – 212
12. Leson E, Stanton SL Sacrohysteropexy with synthetic mesh for the management of uterovaginal prolapse. British journal of obstetrics and gynaecology 2001 108: 629 – 633

13. Surgical correction of defects in pelvic support. Thompson J D in Te Lindes Operative gynaecology. John A Rock and John D. Thompson 8th Edition Lippincott Williams & Wilkins 1997 38:A 951 – 978

Gynaecology Case 3

GESTATIONAL TROPHOBLASTIC NEOPLASIA

Ip No.	0855249	D.O.A.	20.6.02
Name	S W	D.O.D	30.6.02
Age	25	Parity	0 + 1

Presenting complaints

The patient came complaining per vagina bleeding for one week.

History Of Presenting Complain

SW had an evacuation for molar pregnancy in March 2002 in a private clinic but follow up was not done. She later on noted per vagina bleeding which became heavy necessitating her to seek treatment. The bleeding was associated with abdominal pain. She did not have any cough, bowel and urinary habits were normal.

Obstetric / Gynaecology History

SW was a Para 0+1. She attained had menarche at 14 years. Her menstrual cycle was regular in a 26-day cycle with a flow of 4-5 days. Her only pregnancy was the hydaditiform mole in March. She had never used any contraception.

Past medical history

She was not known to suffer from any chronic illness and there was no history of previous admissions or surgery.

Family / social history

S W was a single lady living with her sister in Githurai. She was educated up to form 4 and was unemployed. Her mother was hypertensive. She had never smoked cigarettes or drink alcohol.

Examination findings

She was a young lady in fair general condition. She was not pale, jaundiced or cyanosed. She had no lymphadenopathy. Her vital signs were; temperature 36.8^0 c, blood pressure 110/60, pulse rate 88/minute and respiratory rate 22/min.

Cardiovascular respiratory and central nervous system were essentially normal.

Abdominal Examination

The abdomen was moving with respiration, was not distended. There was supra pubic tenderness but no palpable masses.

Pelvic Examination.

A speculum examination was done. The external genitalia was normal. The vaginal walls looked healthy with no signs of metastasis. The cervix was long closed and posterior. The uterus was bulky corresponding to 12 weeks gestation. The adnexae and Pouch of Douglas were normal. There was some blood on the examining finger..

Diagnosis

An impression of choriocarcinoma was made.

Investigations

ß HCG	72 342 miu/ml	Hb	10.6 g/dl
Urea and electrolytes	normal.	WBC	5.4×10^9
Blood group	0 +ve	Liver function tests	normal

Pelvic ultra sound scan revealed an enlarged uterus with multiple cystic areas suggestive of uterus invasive mole. The ovaries were enlarged with multiple cysts. Chest X ray was normal.

Management

The patient was started on methotrexate 50 mg once daily for 5 days with leukovorin 15 mg once daily on alternate days. After the first course the ß hCG levels went down remarkably to 102 miu/l. She continued with the treatment until the ß hCG levels fell to normal after which she received 2 more courses. Before every course, liver function tests full blood count and renal function tests would be done to monitor the side effects of methotrexate. All these parameters remained within acceptable range.

ß HCG levels during the treatment

23. 06. 02	72 342 miu/ml
12. 07. 02	102 miu/ml
04. 08. 02	73 miu/ml
23.0 8. 02	35 miu/ml
14. 09. 02	5 miu/ml
01. 10. 02	< 2 miu/ml
20. 10. 02	< 2 miu/ml

Follow up

She was advised on necessary follow up and the need to avoid a pregnancy. Since she was single and had already parted with her boyfriend, she chose to abstain. She continued follow up in the GOPC for one year during which period the disease was confirmed cured.

Discussion

The patient presented developed choriocarcinoma following a molar pregnancy. She was successfully treated using methotrexate.

Gestational trophoblastic neoplasia (GTN) encompasses several diseases that arise from the placenta. They include hydatidiform mole, invasive mole (chorioadenoma destruens), choriocarcinoma, and a placental site trophoblastic tumour (PSTT). They show varying propensities of invasion and metastasies.[1] Hydatidiform mole is the most common form of GTN. While invasive mole and choriocarcinoma are malignant, a hydatidiform mole can behave in a malignant or benign fashion.[2] These tumours produce human chorionic gonadotropin (HCG), which is a useful tumour maker. No methods exist to accurately predict the clinical behaviour of a hydatidiform mole by histopathology. The clinical course is defined by the patient's serum human chorionic gonadotropin (HCG) curve after evacuation of the mole.

The incidence varies. In Western countries, 1 per 1000-1500 pregnancies is affected. In Asian countries, the rate is as much as 15 times higher than in the United States.[3,4] In KNH an incidence of I: 1118 was reported.[5] Hydatidiform mole is more frequent in teenagers and in women older than 40 years. The potential for malignant change is higher when a hydatidiform mole occurs in a woman older than 40 years. [6] SW was 25 years old.

Histologically, choriocarcinomas have no villi, but they have sheets of trophoblasts with hoemorrhage. Of all choriocarcinomas, 50% are preceded by a hydatidiform mole, 25% by an abortion, and the other 25% by a full-term pregnancy. [2] but had Hydatidiform mole that was followed by choriocarcinoma. Our patient had choriocarcinoma following a molar pregnancy.

In choriocarcinoma the most frequent symptom is abnormal uterine bleeding .A history of amenorrhoea may exist. Abdominal pain and a pelvic mass may be present. A third of the patients present without pelvic symptoms but have symptoms of distant metastases. Signs and symptoms of preeclampsia occur in up to one third of patients.

Prolonged hyperemesis gravidarum occurs in some. Hyperthyroidism is found in up to 3% of patients. This is due to the production of human molar thyrotropin by the molar tissue and the similarities between HCG and thyroid-stimulating hormone (TSH). If metastases exist, signs and symptoms associated with the metastatic disease, such as haematuria, haemoptysis, abdominal pain, and neurologic symptoms, may be present. Histological proof is rarely made to avoid the risk of fatal haemorrhage caused by biopsy.[2,3,7,8] SW presented with abdominal pain and vaginal bleeding after a period of amenorrhoea.

Laboratory studies should include serum beta-HCG titers. A serum HCG greater than 100,000 mIU/mL should raise the concern of GTN. In 80% of patients with a benign hydatidiform mole, serum HCG titers steadily drop to normal within 8-12 weeks after evacuation of the molar pregnancy. In the other 20% of patients with a malignant hydatidiform mole, serum HCG titers either rise or plateau.[9,10] Other laboratories studies include blood count to detect anaemia secondary to vaginal bleeding. Liver enzymes may become elevated in the presence of metastasis to the liver. Our patient had high ß HCG titers 3 months after the evacuation of the molar pregnancy.

The most frequent sites of metastases of malignant GTN are the lungs, lower genital tract, brain, liver, kidney, and gastrointestinal tract. SW had no metastasis.[10]

The official International Federation of Gynecology and Obstetrics staging of GTN is as follows: [11]

- Stage I – Confined to the uterus
- Stage II – Limited to the genital structures
- Stage III – Lung metastases
- Stage IV – Other metastases

Patients are classified as high-risk or low-risk. The criteria for high-risk metastatic GTN include hepatic or brain metastasis, serum HCG titers greater than 40,000 mIU/mL prior to the initiation of chemotherapy, duration of disease longer than 4 months, prior unsuccessful chemotherapy, and malignant GTN following a term

pregnancy. Patients with malignant non- metastatic or metastatic low-risk GTN have an almost 100% probability of cure with chemotherapy. The probability of cure after chemotherapy for patients with metastatic high-risk GTN is approximately 75%. The probability of a late recurrence after the patient has been in remission (normal serum beta-HCG titers) for 1 year is less than 1%. [10,12]

Chest x-ray is recommended because the lung is the most frequent site of metastasis. CT scan of the head, abdomen, and pelvis is recommended if the patient has choriocarcinoma to search for metastasis. Our patient had a normal chest x ray.

The management depends on the patient's condition and their risk score. Those who have lost a lot of blood need intravenous fluids and sending blood for type and cross matching. Rh-negative patients should receive anti–RhD immune globulin, if not already immunized.

The currently used prognostic scoring index is a modification of the World Health Organization (WHO) classification. [10] It provides points for the presence of a number of prognostic factors, as follows:

Prognostic factors	Score			
	0	1	2	4
Age in years	< 39	> 39		
Antecedent pregnancy	Mole	Abortion	Term	
Interval*	<4	4-6	7-12	> 12
ß HCG serum level iu/litre	10^3	10^3-10^4	10^4-10^5	10^5
Largest tumour in cm	<3	3-5	>5	

Site of metastases		Spleen Kidney	G I tract	Brain Liver
Number of metastases		1-4	5-8	> 8
Prior failed chemotherapy			Single	> 2

Interval – the time between the antecedent pregnancy and the chemotherapy in Months.

Low risk= 0-5 medium risk= 6-8 high risk = 9 and above

ABO blood group contributes little to the overall scoring and has been removed from the current scoring system.

SW was less than 40 years, the disease followed molar pregnancy, she had no metastasis, the antecedent pregnancy was 3 months prior, had no prior chemotherapy and the HCG level was 75 000 m iu/ ml. Therefore her total score was 2 and she was a low risk patient.

Patients with malignant non-metastatic GTN or metastatic low-risk GTN are treated with single-agent chemotherapy. Methotrexate is widely used. Actinomycin D can be used in patients with poor liver function.[13] During treatment, the serum HCG titers are monitored every week. In our set up three additional courses of chemotherapy are administered after a normal serum HCG titer. After 3-4 normal serum HCG titers, the titers are followed once per month for 1 year.[14] A switch from methotrexate to actinomycin D is made if the patient receiving MTX for non-metastatic or metastatic low-risk GTN develops rising or plateauing serum HCG titers. SW received methotrexate with good response.

Patients with high-risk metastatic GTN are subdivided into 2 groups: those with a WHO score of less than 8 and those with a score of 8 or higher and a high risk of therapy failure. In patients with a WHO score of less than 8, the MAC regimen, a combination of methotrexate, actinomycin D, and cyclophosphamide can be used.

This chemotherapeutic regimen is administered every 21 days until the serum HCG titers normalize. Patients with WHO scores of 8 or higher are treated with EMA-CO regimen. This is a combination of etoposide, methotrexate, and actinomycin D administered in the first week of a 2-week cycle and cyclophosphamide and vincristine (Oncovin) administered in the second week. Some substitute cisplatin and etoposide for cyclophosphamide and vincristine during the second week. This is known as the EMA-CE regimen. Some reserve the EMA-CE regimen for patients in whom EMA-CO fails. Two additional courses of EMA-CO or EMA-CE are administered after a normal serum HCG titer in high-risk patients.[15,16]

Patients with metastasis to the brain receive whole brain irradiation. Corticosteroids with systemic effect are administered to reduce brain oedema. Patients with liver metastasis are considered for liver irradiation. Leucovorin; folinic acid is used to prevent toxicity from high doses of methotrexate. It is used in a dose of 15 mg PO/IM q12h for 4 doses starting 24 h after administration of MTX as part of EMA-CO and EMA-CE regimens [15,16]

During the period of follow-up, patients with should use a reliable method of contraception, such as oral contraceptives. The serum HCG titers are critical in monitoring the status of the disease, and a normal intrauterine pregnancy interferes with this critical monitoring tool.[17]

Nonmetastatic GTN has a cure rate with chemotherapy of close to 100%. Metastatic low-risk GTN has a cure rate with chemotherapy of close to 100%. Metastatic high-risk GTN has a cure rate with chemotherapy of approximately 75% .After 12 months of normal HCG titers, less than 1% of patients with malignant GTN have recurrences.

The rate of occurrence of a repeat molar pregnancy is approximately 1-2%. The rate of occurrence of a repeat molar pregnancy in a patient with a history of 2 previous hydatidiform moles is approximately 10-20%. [18] The pregnancy rate after chemotherapy with MTX and cyclophosphamide is 80%. Of women treated with EMA-CO, 46% have had at least 1 live birth after chemotherapy. Patients who become pregnant after treatment for GTN should have a pelvic ultrasound early during the pregnancy to confirm that the pregnancy is normal.[19]

References

1 Berkowitz RS, Goldstein DP; Gestational Trophoblastic Disease in Novaks Gynaecology 12th edition, Williams and Wilkns,335:1261-1282

2 Berkowitz RS, Goldstein DP: Chorionic tumours. N Engl J Med 1996 Dec 5; 335(23): 1740-8.

3 O'Quinn GA, Barnard DE; Gestational Trophoblastic Disease in Current Obstetric & Gynecologic Diagnosis and Treatment,Appleton & Lange, 8th Edition 1994 Pg 967-975

4 Grimes DA: Epidemiology of gestational trophoblastic disease. Am J Obstet Gynecol 1984 Oct 1; 150(3): 309-18.

5 Makokha HE, Mati JKG; Choriocarcinoma at Kenyatta national Hospital 1973-1979 J Obstet Gynecol E. Centr Afri 1982;127

6 Bandy LC, Clarke-Pearson DL, Hammond CB: Malignant potential of gestational trophoblastic disease at the extreme ages of reproductive life. Obstet Gynecol 1984 Sep; 64(3): 395-9.

7 Soto-Wright V, Bernstein M, Goldstein DP, Berkowitz RS: The changing clinical presentation of complete molar pregnancy. Obstet Gynecol 1995 Nov; 86(5): 775-9.

8 Amir SM, Osathanondh R, Berkowitz RS, Goldstein DP: Human chorionic gonadotropin and thyroid function in patients with hydatidiform mole. Am J Obstet Gynecol 1984 Nov 15; 150(6): 723-8.

9 Tidy JA, Gillespie AM, Bright N, et al: Gestational trophoblastic disease: a study of mode of evacuation and subsequent need for treatment with chemotherapy. Gynecol Oncol 2000 Sep; 78(3 Pt 1): 309-12.

10 GY Quinn M, Bleddy J, Dinar Soutter P, Malignant disease of the uterus. Gynaecology by Shaw. 3rd Edition 2003 Churchill Livingstone.

11 FIGO Oncology Committee: FIGO staging for gestational trophoblastic neoplasia 2000. *Int J Gynaecol Obstet* 2002 Jun; 77(3): 285-7.

12 Sebire NJ, Fisher RA, Foskett M: Risk of recurrent hydatidiform mole and subsequent pregnancy outcome following complete or partial hydatidiform molar pregnancy. BJOG 2003 Jan; 110(1): 22-6.

13 Roberts JP, Lurain JR: Treatment of low-risk metastatic gestational trophoblastic tumors with single-agent chemotherapy. Am J Obstet Gynecol 1996 Jun; 174(6): 1917-23; discussion 1923-4.

14 Batorfi J, Vegh G, Szepesi J: How long should patients be followed after molar pregnancy? Analysis of serum hCG follow-up data. Eur J Obstet Gynecol Reprod Biol 2004 Jan 15; 112(1): 95-7.

15 Bower M, Newlands ES, Holden L, et al: EMA/CO for high-risk gestational trophoblastic tumors: results from a cohort of 272 patients. J Clin Oncol 1997 Jul; 15(7): 2636-43

16 Lurain JR: Advances in management of high-risk gestational trophoblastic tumors. J Reprod Med 2002 Jun; 47(6): 451-9.

17 Feltmate CM, Batorfi J, Fulop V: Human chorionic gonadotropin follow-up in patients with molar pregnancy: a time for reevaluation. Obstet Gynecol 2003 Apr; 101(4): 732-6.

18 Sand PK, Lurain JR, Brewer JI: Repeat gestational trophoblastic disease. Obstet Gynecol 1984 Feb; 63(2): 140-4.

19 Garner EI, Lipson E, Bernstein MR: Subsequent pregnancy experience in patients with molar pregnancy and gestational trophoblastic tumor. J Reprod Med 2002 May; 47(5): 380-6.

Gynaecology Case 4

BARTHOLINS ABSCESS – MARSUPIALIZATION DONE

Name	N. M.	I. P. No.	0836212
Age	22	DOA	26.10.02
Parity	0 + 0	DOD	28.10.02

Presenting complaints

She presented with history of vulval pain and swelling for 3 days.

History of presenting illness

She had been well until 3 days prior to admission when she started experiencing some pain in the perineum. She then noticed a swelling that progressively increased in size. By the time of admission the pain was severe and she was walking with difficulty. There was no associated urinary frequency, dysuria or per vaginal discharge. She had been treated for a similar swelling in 2000.

Obstetric and gynaecological history

She was para 0 + 0. She attained menarche at 14 years. Her periods were regular with a flow of 3 – 4 days in a 28 days cycle. Her last menstrual period was on 15.10.02. She sometime used condoms but not consistently.

Past medical history

This was not contributory.

Family and social history

She was a single lady but she had a boyfriend. She worked as a receptionist in town. She did not smoke cigarettes or drink alcohol. There was no history of chronic illness in her family.

Physical examination

She was in stable general condition but appeared to be in a lot of pain. She was walking with a lot of difficulty. She was not pale, jaundiced or cyanosed. Her vital signs were Bp- 110/70 mmHg, PR – 88/min, RR – 22/min and temperature 37°C.

The nervous, cardiovascular and respiratory systems were all within normal. Abdominal exam did not reveal any abnormality.

Pelvic examination

She had a swelling involving the left labia minora. The swelling was very tender, hyperaemic and fluctuant. Digital examination was not done due to the tenderness. There was no discharge around the external genitalia.

Diagnosis

A diagnosis of Bartholins abscess was made.

Management

The patient was explained to the condition she had and the need for marsupialization. An infirmed written consent was obtained.

Investigations

Haemoglobin -	12.5 g/dl	Sodium -	138 mmol/l
Urea -	1.9 mmol/l	Potassium -	3.8 mmol/l
Creatinine -	65 µmol/l		

She was started on intravenous metronindazole 500mg 8 hourly and augmentin 1.2gm 8 hourly. She also received pethidine 50mg intramuscularly. Half hour before theatre atropine 0.6 mg intramuscularly was given.

In theatre she was put under general anaesthesia in lithotomy position. Vulval vaginal toilet was done and aseptic catheterisation done. Examination revealed a swollen, warm and tense left bartholins gland. Aspiration was done and thick pus was obtained. The right labia majora and minora were normal. A longitudinal incision was made along the abscess in the region of the mucocutaneous junction. Foul swelling pus was drained and a sample taken for microscopy, culture and sensitivity. The cavity was cleaned thoroughly with hydrogen peroxide and rinsed with normal saline. The edges of the cervix we reverted using vicryl number 2 – 0. Haemostasis was achieved and the patient was successfully reversed form the general anaesthesia.

The patient did well post operatively. On the second postoperative day she was discharged home on oral metronindazole, augmentin and mefenamic acid.

Follow up

She came for review in the gynaecological outpatient clinic. She had healed well. The culture did not grow any organisms. She was given advice on safer sex and asked to come back if she had any issues.

Discussion

N M was a 22 years old lady who presented with complains of a swollen painful vulval swelling. She was found to have a bartholins abscess and marsupialization was done.

The bartholins gland lies just inferior and lateral to the bulbocavernosus. They are tubular in character with a draining duct measuring about 5 mm in diameter. The duct is 1 – 2 cm long and open on the side of the vestibule just outside the lateral margin of the vaginal orifice. The gland produces clear, viscid and stingy mucoid secretions.

Infection of the gland and its duct is referred to as bartholinitis. It is the commonest bacterial infection of the vulva. It commonly occurs between menarche and menopause. Usually after the age of 30, the glands undergo involution and become atrophic and shrunken.

Obstruction of the duct leads to accumulation of the secretions in the gland and the distal part of the gland. Infection is an important cause of obstruction. If the associated infection is of low grade or recurrent, a cyst may result. With more acute infections an abscess results[1] Other causes of obstruction include inspissated mucus and congenital malformations.[2] Sutures can easily injure or ligate the duct during a mediolateral episiotomy repair or a posterior colporrhaphy. [3]

Causative organisms may be sexually transmitted the commonest being the gonococcus or may be from the vulval flora.[1] The staphylococcal, streptococci and gram negative bacilli have been implicated.[4]

In Kenyatta national hospital the incidence has been reported as 1-7-1-9%. [5, 6] The mean age of infection in KNH was reported as 23 – 25 years. The majority of the patients were from the low social economic class. [5, 6] The patient discussed was 22 years old, single girl.

Most bartholins duct cysts are asymptomatic and are usually found during routine pelvic examinations. However with acute infections, the bartholins abscess presents with pain, erythema, tenderness and dyspareunia. The pain can be very severe leading to difficulty in sitting or walking. The surrounding tissues become oedematous, inflamed with a fluctuant mass. Usually there are few or no systematic symptoms and signs unless the inflammatory process is extensive. Our patient presented with swelling, tenderness and difficulty in walking.

Treatment of bartholinitis requires appropriate swabs for bacteriology, analgesia antibiotics and surgical drainage of the cyst or abscess. Simple incision and drainage provides temporary relief. However the opening tends to get obstructed again leading to a recurrence. Treatment is therefore best accomplished by marsupialization. This procedure enables the preservation of the glands secretory function and avoids excision of the gland with the cyst. The incision should be wide. After the drainage of the contents and draining, the lining of the cyst is everted and approximated to the vaginal mucosa with interrupted sutures. [3] Postoperative sitzs baths beginning on the third or fourth postoperative day are recommended.

10 – 15% of cysts may recur resulting form closure and secondary fibrosis of the orifice.[3]

Bartholins cysts can be treated by insertion of a Word catheter. A small incision about 2cm is made at the area of the ducts orifice. The catheter is inserted and the bulb inflated with 2 – 3 ml of saline solution. The catheter remains for 3 – 4 weeks. The track gets epithelialized. The catheter therefore accomplishes the same result as the surgery with minimal or no trauma. In women aged over 40 years, the edges of the cyst should be sent for histological examination to exclude carcinoma. [1, 7]

References

1. Robert W. S. Soutter W P, Stantolin L S; Benign diseases of the vulva and vagina. Shaws gynaecology, 3rd edition 2003; Churchill Livingstone pg 607

2. Kermit E. K. Anatomy of the female reproductive system. In Current Obstetric & Gynecologic Diagnosis by DeCherney 8th edition. Appleton and Lange. pg 20

3. Rock J A, Thompson J D surgical conditions of the vulva; Te Lindes' operative gynaecology 8th edition 1997 Lippincot & Wilkins, 35; 890-892

4. Gordon M S: Aids to obstetrics and gynaecology 4th edition; Churchill Livingstone 22: 674 – 678

5. Mumia S A, Bartholins abscess at KNH, Mmed thesis 1981, UON

6. Ndede F. O. Review of Bartholins abscess at KNH Mmed thesis, 1990 UON

7. Curry L S, Barclay D L Benign disorders of the vulva & vagina. Current obstetric & gynaecologic diagnosis and treatment 8th edition. Appleton and Lange. Pg 710

Gynaecology Case 5

SYMPTOMATIC UTERINE FIBROIDS – TOTAL ABDOMINAL HYSTERECTOMY

Name	E. W.	DOA	13.11.02
I. P. No.	0989332	DOD	20.11.02
Age	40	Parity	3 + 0

Presenting Complaint

The patient was admitted through the gynaecology outpatient clinic, where she had presented with complains of abdominal swelling and heavy bleeding for six months.

History Of Presenting Illness

She was well until 6 months prior to admission when she started experiencing heavier prolonged bleeding during her menses. The flow increased from 3 days to 6 days and was much heavier than before, with clots. She also noticed a swelling in the lower abdominal region. The swelling was hard and not painful. After 3 months she started experiencing dizziness and body weakness. She therefore sought treatment in a private health facility. She was given haematinics but the problem persisted necessitating her to seek further help.

Obstetric And Gynaecology History

She was a para 3 + 0 and her last delivery was 12 years ago. All her deliveries were vaginal deliveries at term and the children were alive and well. Her last menstrual period was on 1.11.03. Her normal cycle prior to the above complains was 28 days with a flow of 3 days. She had used an intra uterine contraceptive device for five years until 1998. Since then she had not been on contraception.

Past Medical History

This was not significant.

Family And Social History

She was married, living with her husband in Kiambu. She was a housewife, did not smoke or drink alcohol. There was no history of chronic illness in the family.

Physical Examination

She was in good general condition. She was not pale, not jaundiced and had no oedema or lymphadenopathy. Her vital signs were within normal; blood pressure 120/70 mmHg, PR - 80/min, RR 20/min temp 36.6°C.

Respiratory, cardiovascular and the central nervous system were within normal.

Abdominal Examination

The abdomen was distended in the hypo-gastric region. She had a pelvic mass that corresponded to 16 weeks of gestation. The mass was mobile, firm and non-tender. There were no other masses.

Pelvic Examination

The external genitalia was normal. The cervix was long smooth and closed. There was a uterine mass corresponding to 16 weeks gestation. Both adnexae and pouch of Douglas were normal. There was no discharge on the examining finger.

Diagnosis

A clinical diagnosis of uterine fibroids was made.

Management

Since the fibroids were huge and symptomatic and the patient had achieved her desired family size a decision was taken for a total abdominal hysterectomy to be performed.

Investigations

Pap smear- was within normal	Sodium – 138 mmol/L
HB – 10.2g/dl	Potassium – 4.0 mmol/L
WBC – 6.2 x 10^9/L	Creatinine – 78 μmol/L
Platelets – 330 x 10^9/L	Elisa for HIV – negative

Pelvic ultrasound – multiple fibroids were visualized in the uterus.

Management

The nature of the disease and the need for a total abdominal hysterectomy was explained to the patient. A written consent was obtained. On the night prior to surgery she fasted from midnight. An enema was given and 2 grams of oral metronidazole. Half an hour before being taken to theatre, she was pre-medicated with atropine 0.6 mg intramuscularly.

In theatre general anaesthesia was administered. With her legs flexed vulvovaginal toilet was done and aseptically catheterised. Clear urine was drained. Pelvic examination confirmed the earlier findings. She was then laid supine abdomen cleared and draped. The abdominal cavity was opened via a sub-umbilical midline incision. A bulky uterus with multiple intramural and subserosal fibroids was found. The uterus corresponded to 16 weeks gestation. Packing away the gut and placing a self-retaining retractor accomplished exposure. The round ligaments we identified bilaterally clumped and ligated between the clumps. The anterior leaf of broad ligament was opened bilaterally. The posterior leaf of the broad ligament was similarly opened and dissected away. The ovarian pedicles and the proximal uterine tubes were clumped bilaterally divided between clumps and ligated. Bladder was pushed away by blunt dissection. The uterine vessels were identified and divided between clumps and ligated. The cardinal ligaments were also clumped and divided and ligated. A stab wound was then made at the cervical – vaginal junction. The vagina was opened circumferentially. The uterus together with the cervix and the tubes were then removed. The vaginal vault was closed and peritonised. It was anchored to the cardinal and round ligaments. Haemostasis was achieved. The pelvic cavity was cleaned with warm normal saline and abdomen repaired in layers. The catheter was noted to be draining clear urine and was removed. The specimen was sent for histo-pathological examination.

The patient was observed in the recovery room till she was fully awake. She received intravenous gentamycin 80mg 8 hourly, crystalline penicillin 2mu 6 hourly and metronidazole 500mg 6 hourly for 48 hours. She received pethidine intramuscularly 8 hourly for the first 24 hours after which mefenamic acid 500mg 8 hourly was given. She did well post operatively and on the fourth day she was discharged home. The wound had healed well.

Follow up

She came to the gynaecology out patient clinic after 2 weeks. She had done well and raised no complains. The wound was completely healed. Histology report showed multiple simple fibroids with normal cervix and this was explained to her.

DISCUSSION

The patient discussed presented with symptomatic fibroids and a total abdominal hysterectomy was done.

Fibroids are the most common tumours in women. They are composed of mainly smooth muscle cells and varying amounts of fibrous tissue. They arise from the smooth muscle cells.

The incidence of fibroids is different since most are asymptomatic. A 50% incidence has been quoted at post mortem examination [1]. They are clinically evident in about 25% of the women [2]. In KNH Wanjala reported that 66.7% of hysterectomies done were due to fibroids [3]. In a different series 77% of patients requiring hysterectomies had fibroids [4]. They are rare below 20 yrs of age and are not uncommon below 30 yrs in the blacks. The incidence is much higher among the blacks [5]. There is usually a positive history in the family.

The cause of fibroids is unknown but their growth is oestrogen dependent. The most important risk factor is thought to be continuous estrogens secretion without interruption by pregnancy and lactation. Nulliparity or a long period after delivery hence is usually associated. The patient discussed had her last delivery 12 yrs ago. The fibroids tend to grow during pregnancy and regress in menopause. The combined pill reduces risk by approximately 17% with each 5-year usage; cigarette smoking reduces the risk by 18 %. Obesity increases the risk by 21% for every 10 kg gain above the appropriate weight for height [6, 7, 8].

Fibroids may be single but most are multiple. They are mostly found in the corpus, less on the cervix and rarely in the round ligament. They are classified into 3 categories depending on their location. They initially they start as intramural and with growth they become either sub mucous or sub serous. The sub mucous ones lie just below the endometrium, with growth they may protrude into the uterus cavity or even through the cervix and rarely through the vagina. The sub serous fibroids with growth may become peduculated. They may attach to other structures, acquire extra uterine blood supply from omental vessels and become parasitic. . The patient presented had multiple intramural and subserosal fibroids.

Typically the fibroid is a firm nodular structure of variable size. They have a pseudo capsule, and are well demarcated and distinct from the myometrium. The cut surface is glistering pinkish white or grey. It is firm with whorl like arrangement of muscle and fibrous tissue. As they grow in size their blood supply diminishes, and tend to undergo secondary usually degenerative changes. The changes are usually benign including: hyaline, cystic calcification, septic, carneous (red) and fatty changes. Carneous degeneration occurs when there is venous congestion and thrombosis leading to interstitial haemorrhages. Reduced blood supply leads to infarction. It is commonest in pregnancy due to rapid growth of the fibroids. Infection of the fibroid may occur and is common with those that protrude into the vagina. Torsion of a peduculated fibroid can occur. Rarely sarcomatous change occurs in 0.1 – 0.5 % of fibroids.[1]

Most leiomatous are asymptomatic. The symptoms when present depend on their location, size and number. Most degenerative changes have no association with symptoms. However red degeneration maybe associated with pain and fever. Torsion of pedunculated one, infarction and infection also result in pain.

Menorrhagia is a common presentation of fibroids. The periods are usually heavy and may be prolonged. The bleeding may be heavy enough to cause anaemia. Any type of fibroids may lead to menorrhagia but tends to be more severe with sub mucous ones. The mechanism is not well established but theories include; haemorrhage of the endometrium overlying sub mucous fibroids, enlargement of the total surface area of the endometrium due to mechanical distortion by sub mucous and intramural fibroids, and stasis and dilation of the venous plexus draining the endometrium due to mechanical compression of the venous drainage by fibroids in any site. Fibroids are not usually associated with irregular or inter-menstrual bleeding and changes in menstrual regularity should not be attributed to fibroids unless other causes are excluded. [9] Our patient had symptoms of anaemia, which were treated prior to admission for surgery.

Attempted expulsion a sub mucous fibroid may cause uterine cramp associated with bleeding. This may be confused for spontaneous abortion. Pressure on the urinary bladder may lead to urgency, frequency or even incontinence. Pressure in the rectum

especially caused by a parasitic fibroid may lead to intestinal obstruction. Tumours in the cervix may lead to per vaginal bleeding and dyspareunia.

The effect of fibroids on fertility is controversial, impaired gamete transport, distortion of the endometrial cavity, impairment of blood supply to the endometrium and atrophy and ulceration are thought to interfere with implantation. Intramural and sub serosal fibroids have been associated with impaired outcome of assisted reproductive technology treatment. [10] Fibroids have been associated with increased risk of premature delivery, ante-partum haemorrhage, abruption placenta and post partum haemorrhage.
Polycythemia is a rare complication associated with fibroids. This may be due to erythropoetin production by some myoma cells or production by the kidneys due to pressure by the fibroids. [11]

The diagnosis of fibroids is usually suspected from the symptoms. Abdominal and bimanual examination usually reveals a pelvic mass. The ultrasound should be used to confirm and clarify the pelvic mass. Other tests may be done to collaborate the diagnosis including a hysterosalpingnography, intravenous urogram, computed tomography and magnetic imaging resonance. Other laboratory studies to evaluate complications that might have arisen or to prepare the patient for therapy. Such include a full blood count, renal and liver functions tests. A Pap smear and endometrial biopsy to rule out any co-existing malignancy that would affect the definitive management should also be done. Heteroscopy and laparoscopy are investigational procedures that are often merged into treatment.

The treatment choices depend on the age, parity, the size and location, the symptoms (the general health) and the fertility desires of the patient. Asymptomatic fibroids need no active treatment as long as the diagnosis has been reliably made, traditionally surgical removal has been advocated when the uterus size exceeds 12 weeks pregnancy but this policy has been challenged. [12]

In patients who have had severe bleeding transfusion with whole blood or packed red cells may be indicated. After stabilizing the patient, surgery is indicated for infected

fibroids, acute torsion or intestinal obstruction. Myomectomy should not be done in pregnancy except for torsion.

For patients on expectant management, physical examination should be done every 6 months to determine if the fibroids are growing and ultrasound should be done whenever the need arises. Medical treatment may be indicated in some cases. The aim is to relief symptoms and to reduce the size of the fibroid, usually as a temporary measure. This is usually done in preparation for surgery or in the peri-menopause awaiting menopause when most fibroids will regress naturally. There have been reports of fibroid shrinkage with continuous administration of anti-progesterone such as mifepristone and leuprolide acetate. [13]

Gonadotrophin releasing hormone (GnRH) analogues significantly reduce the size of uterine fibroids. They should be started in the mid luteal phase of the ovarian cycle for most rapid pituitary gonadotrophin down regulation. This treatment together with iron supplementation usually corrects anaemia without transfusion. GnRH analogues should not be used for more than 6 months because of menopause like signs and symptoms especially loss of trabecular bone. Hormone replacement therapy may be indicated. GnRH antagonists have also been used successfully and are reported to produce fibroid shrinkage more rapidly than the GnRH analogues, with immediate amenorrhoea. [14]

Hysterectomy has been traditionally used for large or symptomatic fibroids. For very large fibroids, the abdominal route will usually be used. The vaginal route is especially suitable for sub mucous pedunculated fibroids. Laparoscopic assisted methods of vaginal hysterectomy are becoming increasingly popular. This allows for ophorectomy if indicated and inspection of the pelvis. The patient discussed had achieved the desired family size and had large fibroids. Total abdominal hysterectomy was done.

Myomectomy is done where the patient desires to preserve her uterus. It is most satisfactory where fibroids are solitary or few. Sub mucous fibroids may be better accessed hysteroscopicaly. Sub serous fibroids are easily removed particularly if peduculated and may be removed laparoscopicaly. Myolysis with laser or bipolar needles have been done. The needle penetrates the fibroid at multiple sites at 90° to

the uterus. The fibroid then atrophies. Uterine artery embolization as a therapy for symptomatic uterine fibroids has been done. Different series have reported variable results of percentage shrinkage of fibroids. [15, 16]

Hysterectomy with removal of all myomas is curative. Myomectomy is associated with recurrence in 15 – 40% of patients. Two thirds of these patients with recurrence will need further surgical interventions. Myomectomy may necessitate delivery by caesarean section if conception occurs.[17]

REFERENCES

1. Rock A. J. Thompson D J; Leiomyomata uteri and Myomectomy in Te Linde's Operative gynaecology 8th edition; Lipincott Williams & Wilkins pg 731 – 765
2. Buttram V. C. Jnr, Reiter R C; Uterine Leiomata Aetiology, Symptomatology and Management: Fertile Steril 1981; 36: 433 – 45
3. Wanjala S M; Uterine Fibroids Kenyatta National Hospital: Mmed Thesis UON 1980.
4. Cramer SF, Patel A 1990; The frequency of Uterine Leiomyomas; American Journal Of Clinical Pathology 94: 435 – 438
5. Torpin R. Pond E Et al. The Etiologic and Pathologic factors in a series of 1741 fibromyomas of uterus. Am J obstet Gynaecol 1942; 44: 509
6. Wine M. Muram D H, Gonesso M G et al uterine myomas in pregnancy. J obstet Gynaecol survey 39; 2: 1994
7. Whitefield C R uterine fibroids; in Dew Hurtst textbook of obstetric and gynaecological for postgraduates. 4th Edition Black Well Scientific Publication Glasgow England
8. Tindal V R. Jeffcoats principles gynaecology 5th edition Butterworth's publication England; 417; 1987
9. Shaw R W, Soutler P. W. Stanton S L Uterine Fibroids Gynaecology 3rd Edition. Churchill Livingstone pg 477 – 491 2003
10. Eldai – Greva T, Meaghers et al 1998 Effect of Intramural, Subserosal and Submucosal uterine fibroids on the outcome of assisted reproductive technology treatment. Fertility and sterility 70: 687 – 691
11. Weiss DB Alder A, Aboutia F; Erythrocytosis due to erythropoietin producing uterine fibromyoma AMJ obstetric Gynaecol 122: 358, 1975
12. Reiter RE, Wagner PLG, Ambne JC 1992 Routine hysterectomy for asymptomatic uterine fibroids; a reappraisal obstetrics and gynaecology 79 481 – 484
13. Reinsch RC, Murphy AA, Morales AJ et al 1994. The Effects of R U 486 and leuprolide acetate on uterine artery blood flow in the fibroid uterus. A prospective, randomised study. American journal of obstetrics and gynaecology 170; 1623 – 28

REFERENCES

1. Rock A. J. Thompson D J; Leiomyomata uteri and Myomectomy in Te Linde's Operative gynaecology 8th edition; Lipincott Williams & Wilkins pg 731 – 765
2. Buttram V. C. Jnr, Reiter R C; Uterine Leiomata Aetiology, Symptomatology and Management: Fertile Steril 1981; 36: 433 – 45
3. Wanjala S M; Uterine Fibroids Kenyatta National Hospital: Mmed Thesis UON 1980.
4. Cramer SF, Patel A 1990; The frequency of Uterine Leiomyomas; American Journal Of Clinical Pathology 94: 435 – 438
5. Torpin R. Pond E Et al. The Etiologic and Pathologic factors in a series of 1741 fibromyomas of uterus. Am J obstet Gynaecol 1942; 44: 509
6. Wine M. Muram D H, Gonesso M G et al uterine myomas in pregnancy. J obstet Gynaecol survey 39; 2: 1994
7. Whitefield C R uterine fibroids; in Dew Hurtst textbook of obstetric and gynaecological for postgraduates. 4th Edition Black Well Scientific Publication Glasgow England
8. Tindal V R. Jeffcoats principles gynaecology 5th edition Butterworth's publication England; 417; 1987
9. Shaw R W, Soutler P. W. Stanton S L Uterine Fibroids Gynaecology 3rd Edition. Churchill Livingstone pg 477 – 491 2003
10. Eldai – Greva T, Meaghers et al 1998 Effect of Intramural, Subserosal and Submucosal uterine fibroids on the outcome of assisted reproductive technology treatment. Fertility and sterility 70: 687 – 691
11. Weiss DB Alder A, Aboutia F; Erythrocytosis due to erythropoietin producing uterine fibromyoma AMJ obstetric Gynaecol 122: 358, 1975
12. Reiter RE, Wagner PLG, Ambne JC 1992 Routine hysterectomy for asymptomatic uterine fibroids; a reappraisal obstetrics and gynaecology 79 481 – 484
13. Reinsch RC, Murphy AA, Morales AJ et al 1994. The Effects of R U 486 and leuprolide acetate on uterine artery blood flow in the fibroid uterus. A prospective, randomised study. American journal of obstetrics and gynaecology 170; 1623 – 28

14. Kerttek LM, Murphy AA et al 1993. Rapid regression of leiomyomas in response to acute administration of gonadotorophin releasing hormone antagonist. Fertility and sterility 60; 642-646
15. Goodwin SC, McLucas B et al 199 uterine artery embolization for the treatment of uterine leimyomata-mid term results. Journal of vascular interventional radiology 10; 1159 – 65
16. Spies JB et al; 1999 Initial Results Form Uterine Fibroid Embolization For Symptomatic Leiomyoma. Journal of Vascular Interventional Radiology 10 1149 – 57
17. Weider AG Pernoll ML; Benign disorders of the uterine corpus, current obstetric & gynaecological diagnosis & treatment. Appleton & Lange 8th edition.

Gynaecology case 6

POLYCYSTIC OVARY SYNDROME

Ip No.	396040	D.O.A.	20.6.03
Name	N M	D.O.D	24.6.03
Age	32	Parity	0 + 0

Presenting complaints

The patient came complaining of inability to conceive for 3 years.

History Of Presenting Complain

The patient reported failure to conceive despite having regular unprotected coitus, having been married for 3 years. She did not have any associated abdominal pain, per vaginal discharge or galactorrhoea. She had been treated with clomid 50 mgs for 3 menstrual cycles and another 100 mgs for 3 cycles unsuccessfully.

Obstetric / Gynaecology History

N M was a para 0+0. She attained had menarche at 14 years. Her menstrual cycle was regular until about 4 years prior to the presentation when it became irregular. She had prolonged periods of amenorrhoea followed by prolonged heavy flow. The cycle length was 3-4 months. She had no associated dysmenorrhoea. The couple enjoyed what they considered satisfactory sex 3 to 4 times a week with no dyspareunia. There was no history of sexually transmitted diseases.

Past medical history

She was not known to suffer from any chronic illness and there was no history of previous admissions or surgery.

Family / social history

N M was a married lady living with her husband in Nairobi as his only wife. She was an accountant by profession. Her mother was hypertensive. She had never smoked cigarettes or taken alcohol or drugs of addiction. Her husband worked for a bank. He did not smoke cigarettes or consume alcohol. He had never fathered a child.

Examination findings

She was a middle aged lady in stable general condition and obese. She was not pale, jaundiced or cyanosed. She had no lymphadenopathy. She had a normal female body habitus and hair distribution. Her neck was normal with no masses.

Her vital signs were within normal, temperature 36.5^0 c, blood pressure 120/70, pulse rate 80/minute and respiratory rate 22/min.

Her breasts were normally developed, Tanner stage 4. The nipples were not discharging.

Cardiovascular respiratory and central nervous system were essentially normal.

Abdominal Examination

The abdomen was moving with respiration, was not distended and there were no scars. It was soft, non-tender and there were no masses.

Pelvic Examination.

The hair distribution was of the normal female pattern. The external genitalia was normal. The cervix was long closed and pointing posterior. The uterus was normal size and anteverted. The adnexae and Pouch of Douglas were normal. There was no tenderness.

Investigations

Hb – 12.6 g/dl WBC 7.4×10^9

Urea and electrolytes – normal.

Seminanalysis was normal. Pap smear was normal.

Elisa for HIV was negative. Blood sugar was 4.7 mmol/l

Hysterosalpingogram (HSG) – the uterine cavity was normal. Both uterine tubes were demonstrated with bilateral spill of dye.

Transvaginal ultra sound scan (TVS) revealed normal uterus. The ovaries were enlarged with multiple cysts.

Hormone assay;

Day 2	Follicle Stimulating Hormone	4.4	iu / l	(4-13)
	Lutenizing Hormone	23.5	iu / l	(1-18)
	Progesterone (D 21)	10	iu / l	(> 25)
	Prolactin	324	miu/l	(265-490)

Diagnosis

A diagnosis of primary infertility secondary to polycystic ovary syndrome was made.

Management

The patient was explained to the diagnosis and the need for laparoscopy to confirm diagnosis and to perform ovarian drilling. She consented to the procedure and a written consent was obtained. The patient was prepared for theatre.

In theatre she was put under general anaesthesia and positioned. The abdomen was cleaned, vulvovaginal toilet done and she was draped. A peri-umbilical minilap incision was made. A veress needle was introduced into the abdominal cavity via the incision and 3.5 litres of carbon dioxide used for insufflation. The trocar was then introduced followed by the fibre optic camera. The abdominal and pelvic cavities were inspected and the viscera confirmed intact. Two 5 mm ports were made in both iliac fossa and trocars introduced.

The uterus and both tubes were visualized and appeared normal. The ovaries were enlarged bilaterally and were confirmed to be polycystic. On chromopertubation there was bilateral spill of the dye. Ovarian drilling was done bilaterally. A solution containing 250 mgs of hydrocortisone and 5000 units of heparin was introduced into the abdominal cavity. The gas was then released out of the abdominal cavity. General anaesthesia was successfully reversed.

The patient did well post operatively and was discharged home on metformin 500 mgs twice daily and to continue follow up in the infertility clinic.

Discussion

N M was a 32-year old lady with primary infertility. She was found to have polycystic ovary syndrome and was treated with ovarian drilling and metformin.

The Polycystic ovary syndrome is the commonest endocrine disturbance affecting women.[1] It affects 5-10% of women in the reproductive age group and is the most common cause of anovulatory infertility.[2,3]

Various signs and symptoms including menstrual disturbance, hyperandrogenism, hirsutism, obesity, infertility, insulin resistance and the presence of polycystic ovaries characterize polycystic ovary syndrome. The common menstrual disturbances are oligomenorrhoea or amenorrhoea. Hyperandrogenism expresses itself in hirsutism, alopecia and acne. There is heterogeneity of symptoms and signs amongst those with the syndrome Even in an individual the signs and symptoms may change over time.[4] The patient discussed presented with menstrual disturbance and infertility.

The aetiology is uncertain. It is familial and various aspects of the syndrome may be differentially inherited[4]. Polycystic ovaries may exist without clinical signs of the syndrome, which may then become expressed over time. The prevalence of polycystic ovaries is estimated at 22-33%[5,6,7]. There is positive correlation between the degree of insulin resistance and anovulatory infertility[8].

Basic investigations in couples with infertility, where polycystic ovary syndrome may be a contributory factor should include FSH and LH level (day 2- day 4), progesterone (day 21), and tests for tubal patency and semen analysis. To confirm diagnosis of PCOS, the hormonal profile should also include testosterone, sex hormone-binding globulin, fasting insulin, prolactin, thyroid function tests and 17-hydroxy- progesterone. They are usually elevated, with the exception of the sex hormone- binding globulin, which is depressed. In asymptomatic women, the levels of oestradiol and oestrone may be elevated. A pelvic ultrasound should be taken. The trans-vaginal ultrasound has been shown to be more effective than the trans-abdominal, in the screening of polycystic ovaries.[6] Our patient had a TVS, which revealed polycystic ovaries, and her LH was elevated.

Anovulatory infertility due to PCOS should be diagnosed if two of the following three criteria are present, after exclusion of other causes of androgen excess.[9] The three criteria are;

1) Oligo-ovulation or anovulation.

2) Clinical and /or biochemical signs of hyperandrogenism.

3) Polycystic ovaries morphology on ultrasound scan. This is described as the presence of 12 or more follicles in each ovary, (one ovary is sufficient for diagnosis) measuring 2-9mm in diameter and / or increased ovary volume (greater than 10ml). The patient discussed had anovulation and polycystic ovaries on ultra sound.

The first established treatment for POCS was surgical wedge resection of the ovary. It has been abandoned due to the risk of post surgical adhesion formation and replaced by medical ovulation induction.

The clinical management of PCOS is individualized and is directed mainly at the symptoms. Obesity worsens both symptomatology and the endocrine profile. Obese women should be encouraged to lose weight. Weight loss improves the endocrine profile, the likelihood of ovulation and a healthy pregnancy.[10]

Irregular menstrual cycle may be controlled using the combined oral contraceptive pill or progesterone such as provera or duphaston. Ovulation can be induced by anti-oestrogen clomiphene citrate or tamoxifen. Ultrasound monitoring should be done to minimize the 10% risk of multiple pregnancy and to ensure that ovulation is taking place.[11] Patients with anovulatory infertility that are resistant to anti-oestrogen are treated with either parenteral gonadotrophin therapy or laparoscopic ovarian diathermy. The polycystic ovary is very sensitive to stimulation by exogenous hormones, thus, treatment should begin with very low doses of gonadotrophin, and follicular development be carefully monitored by ultrasound.

Laparoscopic ovarian drilling does not pose the risk of multiple pregnancy and ovarian hyper stimulation. It does not require ultrasound monitoring. It appears to be as effective as routine gonadotrophin therapy.[12] It has been shown to significantly change the insulin and glucose responses in women with hyperinsulinaemia.[13] Some have used as a primary treatment in patients with anovulatory PCOS.[14] L1 found a

cumulative conception rate of 54% 12 months after ovarian drilling.[15] The drilling appeared to result in transient increase followed by a subsequent reduction in ovarian volume.[16] Our patient had been treated with clomiphine unsuccessfully therefore laparoscopic drilling was done.

A number of pharmacological agents have been used to amplify the effects of weight loss. Metformin enhances insulin sensitivity, leading to reduced insulin secretion. It has been shown to ameliorate hyper androgenism and abnormalities of gonadotrophin secretion in women with PCOS[17] and can restore menstrual cyclicity and fertility.[18] The patient discussed was put on metformin after the drilling to enhance response.

Polycystic ovary syndrome has been associated with some long-term consequences. The chronic anovulation and unopposed oestrogen action associated with PCOS lead to endometrial hyperplasia and such women have an increased risk of endometrial carcinoma.[19] Obese women with the syndrome have an increased risk of developing impaired glucose tolerance, frank diabetes mellitus and cardiovascular diseases.[20, 21] Hypertension is likely to develop in women with PCOS. It has been found to be three times higher in women with PCOS, compared to controls.[22] Abnormalities of lipid metabolism have been reported to be higher in women with the syndrome.[23]
It is therefore important to follow up women with PCOS and to treat them depending on the various derangements. Follow up ensures early recognition of any of any of the complications and early interventions. Correcting the endocrinological derangements reduces the occurrence of the complications. The patient discussed was counselled on the possible long term complications and the need for follow up.

References

1. Balen AH 1999 The Pathogenesis of Polycystic ovary syndrome; the enigma unravels Lancet 354: 966 – 967

2. Frank S. Polycystic Ovary Syndrome: N Engl J Med 1995; 333: 853 – 61

3. Homburg R. Polycystic ovary syndrome: From gynaecological curiosity to multisystem endocrinopathy. Hum Reprod 1996; 11: 29 – 39

4. Balen A H, Conway G S, Kaltsas et al 1995 Polycystic ovary syndrome; the spectrum of the disorder in 1741 patients. Human Reproduction 10; 2705 – 2712

5. Clayton R N, Ogden et al 1992 How common are Polycystic ovaries in normal women and what is their significance for the fertility of the population? Clinical Endocrinology 37: 127 – 134

6. Farguhar C M, Birdsall et al 1994 Transabdominal versus transvaginal ultrasound diagnosis of polycystic ovaries on ultrasound scanning in a population of randomly selected women. Ultrasound of obstetrics gynaecology 4: 54 – 59

7. Polson DW, Adams J et al 1988 Polycystic ovaries a common finding in normal women, Lancet 1: 870 – 872

8. Conway G S, Aggrawal R et al. Risk factors for coronary artery disease in lean and obese w men with polycystic ovary syndrome. Clin Endocrinal (Oxf) 1992 ; 37: 119 – 25

9. Tarlatzis R, Fauser B et al revised 2003 Consensus on diagnostic criteria and long term health risks related to polycystic ovary syndrome. The Rotterdam ESHRE / ASRM – sponsored PCOS consensus workshop group. Hum Reprod 2004; 19; 41 – 7

10. Clark A M, ledger W, Galletly c et al 1995, Weights loss results in significant improvement in pregnancy and ovulation rates in anovulatory obese women. Human Reproduction 10: 2705 – 2712

11. Royal College of Obstetricians and Gynaecologists 1998 Guidelines on the initial investigation and management of infertility. RCOG press, London.

12. Abdel Gadir A, MOwafi RS et al 1990 ovarian electrocautery versus human menopausal gonadotrophins and pure follicle stimulating hormone therapy in the treatment of patients with polycystic ovarian disease. Clinical endocrinology 33; 585 – 592

13. Sale A, Morris D, Tulandi Et al: Effects of laporoscopic ovarian drilling on adherent steroids in PCOS patients with and without insulinaemia. Fertility and sterility, unites states, mar 2001: 75 :(3) p 501 – 4

14. Farguhar C. V andekerhove P : laporoscopic drilling by diathermy or laser for ovulation induction. Cochrane database of systemic reviews, England, 2000.

15. L1 TC saravelous et al; factors affecting the outcome of laporoscopic drilling for PCOS in women with anovolatory infertility. Br J Obstetric Gynaecology march 105 (3) P 338 – 44

16. Tulandi T Watkin K. Ian SL; reproductive performance and three dimensional ultra sound volumes determination ovarian drilling int 5 fetl women's med Nov – Dec 1997 42 6 p 436 – 440

17. Nestler J E Jakubowikz DJ 1996 decreases in ovarian cytochrome P450c 17 alpha activity and serum free testosterone after reduction of insulin secretion in Polycystic ovary syndrome, New England Journal of medicine 335: 617 – 623

18. Velazque EM, Acosta A, Mendoza SG – 1997 menstrual cyclinicity after metfurmum therapy in PCOS Obstetrics gynaecology 90: 392 – 395

19. Coulam C B, Janson PO et al 1983 chronic anovulation syndrome and associated neoplasia obstetrics gynaecology 61: 403 – 407

20. Dunaif A 1997 insulin resistance and the polycystic ovary syndrome: Mechanisms and implication for pathogenesis. Endocrine Review 18: 774 – 800

21. Birdsall MA, Farguhar CM, white HD 199 association between polycystic ovaries and extent of coronary artery disease in women having cardiac catheterisation. Annals of internal medicine 126: 32 – 35

22. Dahlgren E, Janson PO Et al at 1992 Polycystic ovary syndrome and risk for myocardial infarction. Evaluated from a risk factor model based on prospective population study of women, Acta obsetrica et Gynaecologica scandinavica 71: 599 – 604

23. Wild R A painter PC coulson PB et al 1995. Lipoprotein lipid concentrations and cardiovascular. Journal of clinical endocrinology and metabolism 61 946 – 951

Gynaecology Case 7

BURST ABDOMEN AFTER CAESERIAN SECTION – SECONDARY REPAIR DONE

Name	A. K.	I. P. No.	099090
Age	20	DOA	20.3.04
Parity	0 + 0	DOD	31.3.04

Presenting complaint

The patient complained of copious drainage of blood stained discharge from the wound.

History of presenting complaint

AK had presented to Kenyatta National Hospital as a referral forma peripheral clinic where she had been in labour for a day. She underwent an emergency caesarean section and the outcome was a live female infant who weighed 3050gms and scored 5 / 1, 6 / 5 and 7 /10. The baby was admitted to the newborn unit. The patient was then started on antibiotics postoperatively and appeared to be recovering well. On the fifth postoperative day she reported excessive draining for the operation site.

Obstetric and gynaecologic history

She was now Para 1 + 0 following the said delivery. She had attended antenatal clinic in Kawangware and when she developed labour she went to the same clinic for delivery.

She attained menarche at 13 years. Her cycle was regular with a flow of 3 – 4 days every 27 days. She experienced mild to moderate dysmenorrhoea. She had never used contraception.

Family and social history

She was single unemployed lady living with her aunt in Kawangware. She was educated up to class 8. She did not drink alcohol or smoke cigarettes. There was no history of chronic illness in the family.

Examination

She was in a stable general condition. She was not pale, not jaundiced and had no lymphadenopathy. Her temperature was 37°c, BP 120/80mmgh, pulse rate 88/mm and respiratory rate of 22/min.

The respiratory, cardiovascular and the central nervous systems were within normal.

Abdominal examination

There was a sub umbilical midline incision scar that was draining copious amounts of blood stained fluid. About 2 cm length of the incision line had separated and the omentum was protruding.

Pelvic examination

The external genitalia was normal. The lochia was rubra, scanty and not foul smelling.

Diagnosis

A diagnosis of wound dehiscence was made.

Management

The condition was explained to the patient and the need for repair in theatre. She gave a written consent for the procedure. She was asked not to take any foods or drinks orally. Blood was drawn for grouping and cross matching and intravenous fluids started. A theatre list was made. Half an hour before being taken theatre premedication with atropine was done.

In theatre the patient was laid supine and general anaesthesia administered. A vulvovaginal toilet was done followed by aseptic catheterisation in semi lithotomy position. Clear urine was drained. In the supine position the abdomen was cleaned using an antiseptic and draped. The stitches in the skin were removed. It was then noted that the stitch used for the rectus sheath repair had given way. Abdominal structures looked normal on inspection. The uterus was involuting well. There were no features of sepsis and the uterine incision was healing well.

A peritoneal wash out with rifocin and warm saline was done and the rectus sheath was stitched using nylon number 1. The edges of the wound were freshened using a surgical blade and further cleaning done. The skin was then closed using nylon interrupted stitches and wound dressed. The general anaesthesia was reversed successfully and the patient taken to recovery ward.

Postoperative care

The patient was started on intravenous zinacef 750mg 8 hourly, flagyl 500mg 8 hourly and pethidine 100mg intramuscularly 8 hourly. On the 3rd postoperative day she was started on oral medication, zinnat 500mg BD, flagyl 400mg TID and Ponstan 500mg TID.

The wound healed well. The stitches were removed on the 8the postoperative day. She was discharged home to come again to the postnatal clinic on the 6th postnatal week.

Follow up

She did not turn up for follow up.

Discussion

The patient presented was a 20-year-old primigravida who developed wound dehiscence following caesarean section. Repair was done by mass closure under general anaesthesia with good results.

Wound dehiscence refers to the separation of layers of the abdominal incision and can be complete or incomplete. The dehiscence is considered complete when the peritoneum is included in the disruption. If the intestines protrude through the wound then this is known as evisceration.[1] The patient discussed had complete dehiscence but not evisceration.

This complication of wound healing may range from a small defect in the skin to the burst abdomen. The reported frequency of fascia dehiscence range between 0.5% to 5% o fall abdominal surgeries but a less frequent rate of 0.3 – 3% had been reported for pelvic surgeries. [1,2] The lower rate associated with gynaecological surgeries has been attributed to use of transverse incisions, healthier patients lower infection rates, increased elasticity after pregnancy and fewer bowel incisions [2,3].

Wound dehiscence with evisceration is a serious complication of abdominal surgery that is associated with prolonged morbidity and high mortality. The mortality range form 10 – 24 %. Coexisting medical conditions increases the morality and morbidity considerably. [4]

Several factors influence the occurrence of wound dehiscence. The type and location of the incision as well as the type of suture used contribute to wound dehiscence. Metabolic factors such as malnutrition, poorly controlled diabetes, corticosteroid use and older age are risk factors. Other risk factors include obesity intraabdominal distension, infection, retching and coughing. Most cases of dehiscence are associated with poor tissue healing rather than suture failure.

Wound healing involves several processes namely inflammation, epithelialization fibroplasia, wound contraction and scar maturation. The processes occur simultaneously. The inflammatory phase is the initial response to injury. Fibroplasia involves production of collagen and is the process by which the wound regains

strength. This process requires proper pH, osmolality and temperature. It takes about 4 – 5 days for enough collagen to give the wound some measurable increase in the tensile strength. Interference with any of these processes leads to poor healing. Presence of a foreign body or infection leads to excessive inflammation and this interferes with epithelialization and fibroplasia. Radiotherapy can also lead to poor wound healing. [1,2]

Proper suture selection is critical in preventing wound dehiscence. The suture should have suitable initial tensile strength have a reasonable absorption time. If a suture is absorbed before the wound has gained enough strength to withstand the associated stresses, then the risk of dehiscence is increased. Proper technique is necessary in knot tying to avoid slipping. Large, loops sutures with secure knots are preferable to avoid strangulating and cutting through the sutures. In the patient presented the suture used for the facial layer had given way probably due to poor tying of the knots. Vicryl suture had been used. It is acceptable for facial closure in patients at low risk of dehiscence but needs very secure knots to avoid slipping.

AK did not have most of the risk factors for dehiscence. However being from low social economic status, it is possible she was in poor nutrition status. Deficiency in proteins minerals and vitamins contribute to poor wound healing.

Infection is commonly associated with delayed healing and dehiscence. It leads to depressed collagen synthesis and increased collagenolysis. Infection causes dehiscence in up to 2.5 – 6.1% of caesarean section patients .[5, 6] Risk of infection is increased in patients with prolonged rupture of membranes and those who undergo more than 3 vaginal examinations in labour. Prolonged operation time and blood loss in excess of 1.5 litres are other risk factors [6,7]. The patient discussed did not have obvious local infection; however she had prolonged labour, meaning several vaginal examinations with prolonged period of rapture of membranes. Her operation time and blood loss were within the acceptable time.

Most eviscerations occur from day 5 – 14 after the operation. 24% occur between day 2 and 5 and 21% after the 10th day.[8] Excessive discharge of serosanguinous fluid for several days before the evisceration occurs. Complete wound dehiscence should be

closed soon after the recognition. An occult haematoma can also present with excessive discharge. Exploration of the wound should be done in the operation theatre under general anaesthesia. As the patient is prepared for theatre, the bowel should be replaced with sterile gauze and packed with pads soaked in povidone – iodine broad-spectrum antibiotics should be initiated. During the exploration, any necrotic tissue, clots, and suture material should be removed. The bowel and omentum should be inspected thoroughly and cleansed with warm normal saline. A K had the dehiscence on the 5th postoperative day and repair was done soon after recognition.

References

1. Donald G. G. Incision for gynaecologic surgery in Te Lindes Operative Gynaecology by Rock J. A., Thompson J D. 8th Edition L W & W, Philadelphia 1997 15: 285 – 317
2. Gary H. L., Frank W L; Wound healing, suture material and surgical instrumentation in Telindes Operative Gynaecology by Rock J.A., Thompson JD. 8th Edition LW & W, Philadelphia 14: 263 – 280
3. Wallace D, Heunanadez W. Sctilaenth JB; Prevention of abdominal would disruption, utilizing the smead – Jones closure technique – obstetric gynaecology, 56 (2): 226, 1980
4. Gornall R. J; Postoperative care in gynaecology by Robert W. Shaw. 3rd edition, Churchill Livingstone, 2003 10: 141 – 150
5. Forrester J L, wounds and their management in Gushieri G.R, Giles AR: Essential surgical practise, 3rd edition. Butter worth Heinemann 1995, pg 177 – 190
6. Wanjohi E. N; Risk factors associated with wound infection after caesarean delivery at Kenyatta National Hospital. MMed Thesis U O N 1982
7. Del Valle combs P. Et al; Does the closure of campers fascia reduce the incidence of post-caesarean superficial wound disruption? Obstet gynaecol 1995 85 (3) : 412 – 416
8. Laufman N. Rube S J; Synthetic absorbable sutures. Surgical gynaecology 145: 592 – 608, 1997

Gynaecology Case 8

CARCINOMONA OF THE CERVIX ATAGE 1 B- WERTHEIMS HYSTEROCTOMY DONE

Name	E. W.	I. P. No.	0988351
Age	32	DOA	6. 5. 04
Parity	2 + 0	DOD	13. 5. 04

Presenting complaint

The patient was admitted through the gynaecology outpatient clinic (GOPC) with a diagnosis of cervical carcinoma.

History of presenting illness

The patient had presented to the family planning clinic for routine follow up. She complained of per vaginal discharge and vulval itchiness. A speculum examination done revealed features suggestive of cervicitis. She was treated for the cervicitis and a pap smear was done. The cytology revealed severe dysplasia (high grade squamous intra epithelial lesion). She was then referred to GOPC for colposcopy. A colposcopic directed biopsy was done. This revealed a well-differentiated non-keratinizing squamous cell carcinoma. Examination under anaesthesia showed a lesion that was confined to the cervix making it stage 1B. She was then admitted in ward 1B for surgery.

Past obstetric and gynaecological history

She was para 2+0, her last delivery being in 1998. Both were spontaneous vertex deliveries. She had her first delivery at 23 years. She attained menarche at 14 years. Her first sexual partner was at 16 years. She had two sexual partners prior to her marriage, she was not sure of her husbands sexual history prior to the marriage. She believed they were both faithful to one another in the marriage. After her last delivery she used oral contraceptive pills up to 2001. From 2001 she used Depo-Provera up to the time of presentation.

Her cycle was regular with a flow of 3 – 4 days every 30 days, initially with the use of Depo-Provera the cycle had become irregular. There was no history of sexually transmitted infections. She had not had another pap smear before.

Past medical history

This was not significant

Family and social history

She was married living with her husband in Riruta. She was a primary school teacher. She did not smoke cigarettes or drink alcohol. Her sister was hypertensive. There was no other chronic illness in the family.

Physical examination

She was in good general condition, not pale or jaundiced. She had no oedema, oral thrush or lymphodenopathy. Her vital signs were as follows;

Blood pressure	:	110/70	PR	:	20/min
PR	:	84/min	temp	:	36.4°C

Abdominal examination

The abdomen was scaphoid moving with respiration. It was soft, with no masses or tenderness. The bowel sounds were normal.

Pelvic examination

The external genitalia was normal. The vaginal walls were grossly normal. The cervix was hyperaemic, bleeding easily on touch but no obvious protruding mass. It felt very firm. The uterus was normal in size. The adnexae and the Pouch of Douglas felt normal and were non-tender. There was no obvious discharge.

Investigations

HB	:	14.2 g/dl	WBC	:	$4.5 \times 10^9/_L$
Platelets	:	$350 \times 10^9/_L$	Na	:	140 mmoL/L
K	:	4.8 mmoL/L	Urea	:	2.6 mmoL/L
Creat	:	82 µmmoL/L	ELISA for HIV:		negative

IVU – normal kidneys and ureters

Diagnosis

A diagnosis of squamous cell carcinoma of the cervix stage 1B was made.

Management

The patient was explained to the nature of the illness and the need for a Wertheim's hysterectomy. She understood and gave a written consent. Blood for grouping and cross matching was taken. Two units of whole blood were made available. The night before surgery she received dulcolax, enema and flagyl as gut preparation. She did not eat or drink from midnight. In the morning 30 minutes before going to theatre, she received atropine and pethidine intramuscularly.

In theatre

Under general anaesthesia, the patient was put in lithotomy position. Vulvo-vaginal toilet was done, aseptic catheterisation done and clear urine was drained. Examination under anaesthesia confirmed the earlier findings. Methylene blue dye was used to pain the vagina. The patient was laid supine; the abdomen was cleaned and draped. The abdomen was opened via a sub umbilical midline incision. The omentum looked healthy grossly. The uterus, both tubes and ovaries were all normal glossy. There were flimsy adhesions in the pouch of Douglas, a few extending to the fallopian tubes. The liver, intestines, the spleen and the kidneys were noted to be normal. On palpation the para aortic nodes felt normal. The flimsy adhesions were realised and the intestines packed away for good exposure of the pelvic organs.

The right round ligament was identified, double clamped cut between clamps and ligated. The infundibulo pelvic ligament on the right was then identified, double clamped cut and ligated. The anterior leaf of the broad ligament was opened on the right lateral side of the uterus and the incision was extended anteriorly towards the uterovesical pouch, to the midline. These procedures were repeated on the left side. The bladder was then pushed away from the uterus, to the level of the anterior vaginal vault, by blunt dissention. The para vesicle space was found to be free of tumour. The posterior leaf of the broad ligament was then opened and deflected downwards. The right ureter was identified, mobilized and suspended with Marceline tape. The right uterine vessels were identified, double clumped, cut and ligated. A similar procedure was done for the left ureter and uterine vessels. The internal, external and common

iliac vessels were identified bilaterally. The lymph nodes along these vessels as lymph nodes long these vessels as well as those along the obuturator arteries were palpated. They were not enlarged. All the identifiable lymph nodes were dissected and removed bilaterally. They were sent for histological examination.

The uterosacral ligaments were cut between clamps and transfixed. The bladder was deflected downwards to expose the upper 1/3 of the vagina. The vagina was opened and cut all around at the level of approximately 3cm from the vault. Thus the lower 2/3 of the vagina was spared. The uterus together with the upper 1/3 of the vagina was all delivered out of the abdomen. The vaginal edges were then approximated using interrupted suture. Haemostasis was achieved and reperitonization done. The Marceline tape anchoring the ureters was released and removed. The abdomen was closed in layers after a correct instrument and swab count. General anaesthesia was successfully reversed.

Post operative care

The patient did well post operatively. The following day she started taking oral sips and graduated to light diet slowly. She was also mobilized. On the 3rd postoperative day, the wound was found to be clean and dry. By the 4th day she was well with no complains and was allowed to go home. She was to come after 2 weeks for review in GOPC.

Follow up

She came back to the clinic after 2 weeks. She had done well, with no complains. The wound had healed well. The histology report confirmed a well-differentiated squamous cell carcinoma of the cervix. The uterus, ovaries and the lymph nodes did not show any features of metastasis. The results and the need for follow up were explained to her. She was to be doing vault smears.

Discussion

The patient discussed was a 32 year old para 2 + 0 who was found to have cancer of the cervix stage 1B. A Wertheim's hysterectomy was done successfully.

Cervical cancer remains a common malignancy world wide being the second most common female cancer after breast cancer. [1, 2] The incidence in the developed world is falling due to effective cervical cancer screening. [2] However in many developing countries, it continues to be a leading cause of death among adult females due to high prevalence rate and inadequate early detection and treatment. [3] There are approximately half a million new cases each year worldwide with 80% occurring in developing countries. [4]

Cancer of the cervix is prevalent in Kenya but the exact incidence is not well known and most women present with late stage disease. . [5, 6, 7] Over 60% of them present in stage 3 or 4.[8] Our patient had early disease. The pick incidence for invasive cancer is 45 – 50 years of age but in recent years there has been a rise in the 25 – 34 year range 4, 9. The pick incidence of CIN is 25 – 40 years. In Kenya Ndavi found the peak prevalence of CIN to be 30 – 40 year age group.[9] The patient discussed was 32 years and she had invasive disease.

Several factors have been associated with development of cervical cancer. Racial and social economic factors have been cited. Cervical cancer appears to occur more on the North African women than in European women. Prostitutes and prison inmates have a higher risk while in celibate women the disease is unusual.[3,10] Other factors associated with increased risk include multiple sexual partners, early age at first intercourse especially within 1 year of menarche, poor personal hygiene, smoking and use of oral contraceptive pills. [3,10] E W had sexual intercourse at 16 years of age, though this was not within her first year of menarche, it was early. She had 2 sexual partners as a teenager before she got married. These factors might have contributed to the development of the cervical cancer. She had used contraceptive pill for some time but had never smoked cigarettes.

Sexually transmitted infections (STIs) such as the human papilloma (HPV) virus and herpes simplex virus have been found to play a role in the development of cervical

cancer. There is overwhelming evidence that HPVs are the main cause of both pre-invasive and invasive squamous cell carcinoma of the cervix. [11] HPV types 6, 11, 16, 18, and 31 are more commonly associated with the carcinoma of the cervix. In some women with HPV infection, the HPV DNA is integrated into the host cell genome leading to changes in morphological appearance. It is thought that other STIs render the cervical cells more susceptible to the HPV infection.[1] Immuno-suppression increases; the frequency of the malignant disease, possibly by increasing the HPV associated pathology. Highly active antiretroviral therapy (HAART) results in a rise in CD4 count and reduce the risk of HPV related pathology. The patient discussed was not immuno-suppressed.

About 70-80% of cervical carcinomas are squamous cell. The remainder include various types of adenocarcinomas, adenosquamous carcinomas and undifferentiated carcinomas. The squamous cell carcinoma is further classified according to the predominant cell type. They include large cell non-keratinizing, large cell keratinizing and small cell carcinomas. The large cell non-keratizing is reported to have the best prognosis. The verrucous carcinoma is a rare subtype of well-differentiated squamous carcinoma. It is often associated with HPV 6.

The well-differentiated carcinoma is classified as grade 1, moderately differentiated as grade II and the poorly differentiated as grade III. The grade III tumour metastasises early but the initial response to radiotherapy is better.

Adenocarcinoma of the cervix is derived form the glandular elements of the cervix. The clear cell variety may be associated with diethylstilbestrol exposure in utero. When the initial growth of the adenocarcinoma is within the endocervical canal, the exocervix appears normal leading to delay in the diagnosis.

The mixed epithelial carcinoma (adenosquamous carcinoma) contains a mixture of squamous and glandular cells. Carcinoid tumour arising from the argyphil cells of the endocervical epithelium also occurs. Direct extension of metastatic tumour may occur arising from the endometrium, rectum or bladder. Lymphatic or vascular metastases into the cervix are rare but may arise form ovarian or endometrial carcinomas. Other forms of malignancies may be encountered in the cervix but quite rarely. Our patient had well-differentiated squamous cell carcinoma of the cervix.

Staging of the carcinoma of the cervix is based on clinical evaluation apart from stages 1a1 and 1a2 where histological diagnosis is needed for the dimensions. The staging should be preferably done under general anaesthesia and should not be altered with subsequent findings. [12]

FIGO staging of cervical cancer [12]

Stage	features
0	carcinoma in situ, CIN 3
I	cervical carcinoma confined to the cervix
I a	microscopic lesion; measured stromal invasion with a maximum depth of 5mm and a horizontal extension not more than 7mm
I a 1	stromal invasion ≤ 3mm, horizontal spread ≤ 7mm
I a 2	stromal invasion 3 – 5mm, horizontal spread ≤ 7mm
I b	preclinical lesions greater than 1a2 or visible lesions confined to the cervix
I b I	clinical lesions ≤ 4cm
I b 2	clinical lesions > 4cm
II	extension beyond cervix but not of the pelvic sidewall or the lower third of vagina
II a	involvement of the upper two thirds of the vagina
II b	parametrial extension but not reaching the pelvic sidewall
III	extension to the pelvic sidewall or lower third of vagina
III a	extension to the lower third of vagina without extension to the pelvic side wall
III b	extension to the pelvic side wall and / or causes hydronephosis or non-functioning kidney
IV	extension beyond the true pelvis or involvement of the bladder mucosa and /or rectum.
IV a	adjacent organs involvement; bladder, rectum
IV b	distant metastasis

Carcinoma of the cervix spreads predominantly by either direct invasion or lymphatic permeation. As the disease advances direct involvement of bone within the pelvis is

common. Blood stream metastasis also occurs usually with the poorly differentiated tumours. The most common sites are the lungs, bone and liver.

Grossly the tumour may be exophytic, endophytitic or barrel shaped (the tumour expands the endocervix in the shape of a barrel). The larger the tumour mass, the higher the chance of spread. Only 5% of lesions involving less than 20% of the cervix have histological evidence of parametrial disease compared to 48% of tumours invading more than 80% of the cervix.[13] Large tumour volume, parametrium involvement and lymph node metastases are associated with a worse prognosis.[1,14] The patient discussed had no evidence of node metastases or parametrial involvement. Her tumour mass was minimal with no fungating tumour.

Stage Ia disease is usually asymptomatic; symptoms develop as the disease advance. The cervix may enlarge, get irregular and firm and eventually become fixed. Exophytic growths appear friable, cauli-flower like and bleeds easily. Infiltrative cancer may present with ulceration. The commonest presentation of invasive cervical cancer is abnormal vaginal bleeding. This may constitute blood stained leucorrhoea discharge, scanty spotting or frank bleeding. The bleeding may be inter-menstrual bleeding or post coital. Vaginal discharge usually sanguineous or purulent, odorous and non-pruritic is common. Pelvic pain usually unilateral radiating to the hip or thigh indicates advanced disease. Urine and / or faecal incontinence may also occur with advanced disease. In the late stages of the disease, weight loss, weakness and anaemia are common.

The physical examination should include careful examination of the supraclavicular and inguinal lymph nodes and palpation of the abdomen for enlarged organs. Speculum examination should be done before the digital examination. In case of an obvious lesion, a deep punch biopsy should be done. In less obvious cases colposcopic directed biopsy or a cone biopsy should be done. The patient discussed was done a colposcopic directed biopsy and this revealed invasive carcinoma.
Examination under anaesthesia should be done, for effective evaluation of the parametria. If there is suspicion of rectal spread protoscopy and symoidoscopy is done. When the disease appears to have spread locally, cystoscopy is indicated. Our

patient had early disease, examination under anaesthesia was done but protoscopy and cytoscopy were not done.

CT scanning and abdominopelvic ultra sound can be used to estimate the size and extent of the primary tumour and to detect nodal disease. The magnetic resonance imaging gives reliable information on tumour volume and extent of uterine and parametrial involvement. The findings of these investigations do not alter the FIGO staging of the disease.[1] Intravenous urography may reveal terminal ureteral obstruction in advanced disease. A chest X-ray is indicated to rule out lung metastases and pleural effusion.

The management of the cervical cancer involves treating both the primary lesion and the potential sites of metastases. The options of treatment include surgery, radiotherapy, chemotherapy or a combination of two or more of these. In women selected for surgery, adjuvant radiotherapy should be avoided if possible due to increased risks of complications. The choice of treatment depends on the stage of the disease, age of the women and the general condition. The treatment goal is either curative or palliative depending on the stage of the disease.

In stage Ia1, the risk of lymph node spread is less than 1%.[14,15] The diagnosis is usually made on cone biopsy or large loop excision of the transformation zone (LLETZ). If the excision margins are clear of the disease, no further treatment is necessary. If the excision margins are involved, a further LLETZ may be performed. A simple hysterectomy may be offered for women who have completed their family. In stage Ia2 the risk of lymph node metastases is approximately 5%. [14] A simple hysterectomy is appropriate. In carefully selected women who desire more children, a LLETZ or cone biopsy may be suitable. Some authorities recommended modified radical hysterectomy and pelvic lymphadectomy.[16] For women desiring fertility preservation a cone biopsy with extra peritoneal or laparoscopic pelvic lymphadectomy or radical trachelectomy with reanastomosis of the vagina and uterine isthmus, with pelvic lympadectomy may be done. Intra cavitory plus external beam radiotherapy is offered for women who are unfit for surgery. 11

Stage Ib and IIa cervical disease is the ideal stage for radical hysterectomy and pelvic lymphadectomy. Primary radiotherapy can be used with similar cure rates as the surgery but surgery has the advantage of possible ovarian conservation and preservation of sexual function.[18] Our patient had stage Ib disease and radical hysterectomy and pelvic lymphadectomy was done.

Cervical cancer stage IIb to IVa is considered advanced disease. Radical external beam radiotherapy with concurrent chemotherapy (cisplatin) plus brachytherapy is the treatment of choice.[19] cisplatin is used as a radio sensitiser. It is associated with reduced disease progression and a longer period of remission.[20] Cisplatin has been used with 5 – fluorouracil in combination, but the combination is tolerated less. In stage IV b, there is no standard treatment and palliation is the goal. Optimal pain control is necessary. Anaemia should be corrected and nutrition improved. Palliative doses of radiotherapy may be given for bleeding control when bleeding is a major issue. Infections should be treated promptly.

Management of recurrent cervical cancer depends on the mode of primary therapy, type of recurrence and the woman's fitness. In local pelvic recurrence following primary treatment with radiotherapy, exenterative pelvic surgery may be suitable. In carefully selected women the exenterative surgery could give a 5-year survival of 40 – 60%. Recurrence after surgery is best treated with radiotherapy with or without platinum based chemotherapy.[22] Radical hysterectomy and exenterative surgery involving partial resection of the bowel, bladder, and or ureter may be performed in carefully selected women with persistent disease following primary radiotherapy.[23]

As part of preoperative care, blood should be cross matched and made available. Rigorous gut preparation is not necessary but a mild laxative and a small enema avoids the filing of the pelvis with distended loops of bowel during the operation. This also helps to make the patient s first few postoperative days more comfortable. A single doe of broad-spectrum antibiotic given prophylactically reduces the risk of infection complications. Low molecular weight heparin should be given 8 – 24 hours prior to surgery for high-risk patients.

Immediate complications of surgery include haemorrhage and damage to the urinary or intestinal tract. One of the most serious complications is pulmonary embolism. Ureteric and bladder fistulae follow 1 – 2% of Wertheim hysterectomie.[1] Atonic bladder with loss of bladder sensation can also occur but usually resolves spontaneously. Complications of radiotherapy include diarrhoea and less commonly acute urinary symptoms. Late complications may include sub acute obstruction, diarrhoea secondary to radiation colitis and haematuria due to radiation cystitis. A Vesicovaginal fistula occasionally occurs.
In patients with massive bleeding, bed rest vaginal packing and use of styptic solutions and tranexamic acid may be helpful in arresting the haemorrhage.

Continuing follow up for treated patients is important to evaluate the results, provide reassurance and to give symptomatic relief to those whose treatment has failed. Suggested follow up should be 3 monthly for 3 years, 6 monthly for 2 years and annual visits thereafter.[1]

The long natural history of the cervical cancer and the relative accessibility of the cervix and the advent of exfoliative cell cytology have enabled a strategy for prevention of invasive disease. This is done by detection and treatment of preinvasive disease. The papanicoalauo technique has been used extensively and significantly reduces death form cervical cancer where population coverage exceeds 80%. In England the incidence of cervical cancer fell from 4467 new cases in 1985 to 2900 in 1995.[24] Improvement of cervical screening is now the subject of intense interest, including liquid based cytology and HPV testing. The liquid based cytology appears to result in fewer inadequate smears and may increase sensitivity. More effort should be put in educating women about the risk factors and the need for screening.

References

1. Roberto D. Bleddyns J et al, Malignant diseases of the cervix in gynaecology by Robert Shaw, 3rd Edition Churchill Livingstone London, 2003, 39: 583 – 596
2. Greenlee R. Murray J et al: Cancer statistics, 2000. CA Cancer J. Clin 2000, 50:7 – 33
3. Shingleton M., Thompson J. Cancer of the cervix. Te Lindes operative Gynaecology 8th Edition 1997, LW&W Philadelphia, 49: 1413 – 1492
4. Parkin D M, Global cancer statistics in the year 2000. Lancet Oncol 2001; 2, 533-43
5. Ojwang SBO, Mati JK; Carcinoma of cervix in Kenya. E Afri Med J 1978; 55:194
6. Mati J K, Past Present, and Future status of cervical cancer research in Kenya human paplilloma virus and cervical cancer in Kenya. Epidemiology, prevention and control 1994
7. Rogo K. O. Human Papilloma virus and human immunodeficiency virus infection in relation to cervical cancer. Umea university Medical sciences dissertations new series 1992
8. Muia Ndavi P. Mwalali PN, Mbugua SE; Cervical cytology in a Kenya rural population J Obstet / gynaecology cent Afri 1984; 3, 167 – 170
9. Goodman, Hill C; Premalignant and Malignant Disorders of the uterine cervix current obstetric and gynaecologic diagnosis and treatment. 8th edition Appleton & Lange 1994 47; 921 -935
10. Safam IA, Nunn's D, Quentin D, David I; The current management of cervical cancer. The obstetrician & gynaecologist 2004; 6; 196-202
11. Benedet JL, Bender H, Jones et al, FIGO staging classifications, and clinical practice guidelines in the management of gynaecologicl cancers. FIGO committee on gynaecologic cancers. FIGO committee on gynacolgic oncology. Int gyanaecol Obstet 2000; 70; 209 – 262
12. Burghardt E, Haas J, Girard F, The significance of the prametrium in the operative treatment of cervical cancer, in Burghardt E, Monoghan JM Operative treatment of cervical cancer – Bailliere Tindall, London 1988
13. Mahmoud I S; Cervical cancer in Shafim lauseley D, Jordan J.A. Editors hand book of gynaecological oncology London, Churchill livingstone; 2000; 210 – 214

14. Creaseman WT. Fetter BF et al. Management of stage Ia carcinoma of the cervix. Am J. Obstet Gynaecology 1985, 153, 164 – 172
15. Hacker NF cervical cancer in Berek J S Hacker NF editors practical gynaecologic oncology Philadelphia; Lippincott Williams & Wilkins 2004- pg 345 – 405
16. Dargent D, Brun J L Roy M Rem I Pregnancies following radical trachelectomy for invasive cervical cancer. Gynaecol oncol 1994; 52- 105
17. Landon F, Maneo A Et al; Randomised study of radical surgery versus radiotherapy for stage Ib – I1 a cervical cancer. Lancet 1997; 350; 535-40
18. Stehman FB, Bundy BN, Thomas G, Keys HM et al; Hydroxyurea versus misonidazole with radiation in cervical carcinoma; long term follow up of a gynaecologic oncology group trial. J chin oncol 1993; 11; 1523 – 8
19. Rose PG, Bundy BN, Watkins EB et al Concurrent cisplatin – based radiotherapy and chemotherapy for locally advanced cancer. N engl J med 199; 340 1144-53.
20. Robertson G, Lopes A, Beynan G, MOnoghan JM; Pelvic exenteration; a review of the gate head experience 1974 – 1992, Bi J obstet gynaeco; 1994; 101; 529 – 31
21. Park RC, Thigpen J I. Chemotherapy in advanced and recurrent cervical cancer. A review cancer 1993; 71; 1446-50
22. Rut Ledge S, Carey MS, Pitchchard H, Allen H; Conservative surgery for recurrent or persistent carcinoma of the cervix following irradiation, is exenteration always necessary? Gynaecology oncology 1994; 52; 353 – 9
23. Quinn M, Babb P, Jones J, Allen E. Effect of screening on incidence of and mortality from cancer of the cervix in England; evaluation based routinely on collected statistics – BMJ 1999; 318; 904 – 908

Gynaecology Case 9

RUPTURED ECTOPIC PREGNANCY – LEFT SALPINGECTOMY

Name	F M	D.O.A.	20.05.04
Ip No.	0964752	D.O.D	23.05.04
Age	30	parity	2 + 0

Presenting complaints

The patient presented with complains of abdominal pains for two days. The pain was of sudden onset. Initially in the left iliac fossae but later became generalised. She had associated nausea and vomiting. She had no vaginal bleeding or discharge. She was passing urine normally.

Past obstetric history

She attained menarche at 15 years of age. Her last normal period was on 25.4.04 giving her an amenorrhoea of 8 weeks. Her cycle was 29 – 30 days and the flow was 3 – 4 days. She had no dysmenorrhoea and the flow was normal. She had never used any form of contraception. She was para 2+0. Her first delivery was in 1994, which was normal, and the baby was alive and well. Her second delivery was in 1998 to a premature infant who succumbed soon after birth. She had normal a puerperium. Since then she had been trying to conceive unsuccessfully. She had not been treated for a sexually transmitted infection.

Past medical history

She had a tuboplasty done in 2003 for secondary infertility. She had no known chronic illness.

Family and social history

She was a housewife living with her husband in Huruma. She did not drink alcohol or smoke cigarettes. There was no history of chronic illness in her family.

Physical examination

She was in fair general condition, moderately pale and afebrile. The vital signs were; blood pressure 110/70 mgHg, pulse rate 98/min respiratory rate 22/min and temperature 36.5°c.

The cardiovascular, respiratory and the central nervous systems were within normal.

Abdominal examination

The abdomen was moving with respiration. There was no obvious distension. On palpation she had marked tenderness in the suprapubic region and both flanks. There was rebound tenderness and guarding. Paracentesis was done and yielded non-clotting blood.

Vaginal examination

The external genitalia was normal the cervix was long, soft posterior and closed. Both adnexae and pouch of Douglas were tender and full. The uterus was bulky. There was no blood on examining finger.

Diagnosis

A diagnosis of ruptured ectopic pregnancy was made and the patient was planned for emergency laporatomy.

Management

Blood samples for grouping and cross matching and packed cell volume (PCV) estimation were taken. An intravenous infusion with normal saline was commenced. The patient was explained to her condition and the need for emergency laparotomy. She signed the consent. Shaving of the pubic region was done and she was premeditated with atropine sulphate 0.6mg intra muscularly.

Operation procedure

In Theatre, under general anaesthesia in semilithotomy position vulvovaginal toilet was done and aseptically catheterised, clear urine was drained.

In supire position the abdomen was cleaned with antiseptic and draped. A sub umbilical midline incision was used to open the abdomen. Moderate adhesions of the

penitoneum and omentum were encountered and released. A haemoperitoneum of about 1000mls and clots were found and evacuated. There was a ruptured gestation sac, in the ampullary region of the left fallopian tube that was actively bleeding. A left partial salpingectomy was done. Both ovaries were found to be grossly normal. The right fallopian tube was buried in adhesions, which were partly released. The abdominal cavity was cleaned with normal saline. The abdomen was repaired after correct instruments and swabs count. The anaesthesia was reversed successfully.

Post operative care

The patient was put on prophylactic antibiotics, crystalline penicillin 2 megaunits, 6 hourly and gentamycin 80mg 8 hourly for 48 hours. Pain relief was by pethidine given intramuscular 100mg 6 hourly for 24 hours. Intravenous fluids were maintained for 24 hours, alternating normal saline with 5% dextrose. She recovered well. The check haemoglobin on day 3 post operatively was 8mg/dl she was discharged on the fourth postoperative day for removal of stitches on the 7th post operative day. She was to come again for review in the gynaecology outpatient clinic after two weeks. She was discharged on oral antibiotics and haematinics.

Follow up

She came to the clinic after two weeks. She raised no complains. The wound had healed well. She was to continue follow up in the infertility clinic.

DISCUSSION

The patient presented had a ruptured ampullary ectopic gestation and a partial salpingectomy was done with good results.

Ectopic pregnancy is derived from the Greek word ektopos, meaning out of place, and it refers to the implantation of a fertilized egg in a location outside of the uterine cavity, including the fallopian tubes, cervix, ovary, cornual region of the uterus, and the abdominal cavity. As the abnormally implanted fertilized egg enlarges, it creates the potential for organ rupture because only the uterine cavity is designed to expand and accommodate foetal development

Since 1970, the frequency of ectopic pregnancy has increased 6-fold in the united states, and it now occurs in 2% of all pregnancies.[1] In Kenyatta National Hospital, Miyoro found an average of 4.4 patients per week while Okumu and Sinei had found 2 patients per week. [2, 3]

Multiple factors contribute to the relative risk of ectopic pregnancy. Pelvic inflammatory disease (PID) has been associated with increased rate of ectopic pregnancy. The incidence of tubal damage increases after successive episodes of PID (13% after 1 episode, 35% after 2 episodes, 75% after 3 episodes).[4] In KNH it was reported that 42-69% of patients had adhesions suggestive of PID.[3] The patient presented had adhesions. With prior ectopic pregnancy, there is 7-13 fold increase in the likelihood of another ectopic pregnancy.[5] Prior tubal surgery increases the risk of ectopic pregnancy depending on the degree of damage and the extent of anatomic alteration. Conception after previous tubal ligation increases a women's risk of developing ectopic pregnancies.[6] The risk of ectopic pregnancy dramatically increases with ovulation induction and use of assisted reproductive techniques to conceive, such as in vitro fertilization (IVF) or gamete intrafallopian transfer (GIFT). [7,8] Conceiving with an IUD in place, increases the risk of ectopic pregnancy 7 times. [9] Our patient had previous tubal surgery but had not used any assisted reproductive techniques.

The highest rate of ectopic pregnancy occurs in women aged 35-44 years. But in KNH 72% were 20-30 years old. [3] The patient discussed was 30 years old. Cigarette smoking has been shown to be a risk factor for developing an ectopic pregnancy. A dose-response effect has been suggested.[15] The patient discussed did not smoke.

Most ectopic pregnancies are located in the fallopian .The commonest being the ampullary portion of the tube (over 80%) followed by the isthmic segment of the tube (12%), the fimbria (5%), and the cornual and interstitial region of the tube (2%). Non tubal sites of ectopic pregnancy are a rare occurrence, with abdominal pregnancies accounting for 1.4% of ectopic pregnancies and ovarian and cervical sites accounting for 0.2% each.[16]Here in Kenya Mwathe reported 34.7% to be ampullary,19.5% isthmal 14.8% fimbrial and 9.6 corneal.[10]The patient presented had an ampullary ectopic gestation.

The classic clinical triad of ectopic pregnancy is pain, amenorrhea, and vaginal bleeding. Unfortunately, only 50% of patients present typically. Patients may present with other symptoms common to early pregnancy, including nausea, breast fullness, fatigue, low abdominal pain, heavy cramping, shoulder pain, and recent dyspareunia. Clinicians should have a high index of suspicion for ectopic pregnancy in any woman who presents with these symptoms and who presents with physical findings of pelvic tenderness, enlarged uterus, adnexal mass, or tenderness. Only 40-50% of patients with an ectopic pregnancy present with vaginal bleeding, 50% have a palpable adnexal mass, and 75% may have abdominal tenderness. Our patient had abdominal tenderness, nausea and vomiting but no vaginal bleeding.

Differential diagnosis include appendicitis, salpingitis, ruptured corpus luteum cyst or ovarian follicle, spontaneous abortion or threatened abortion, ovarian torsion, and urinary tract disease. Intrauterine pregnancies with other abdominal or pelvic problems such as degenerating fibroids must also be excluded. Patients with ruptured gestation sac with intra peritoneal haemorrhage may present with features of acute abdomen and/or shock. The patient discussed had features of acute abdomen and was moderately pale but was not in shock.

Any female patient in the reproductive years presenting with abdominal pain, cramping, or vaginal bleeding should be screened for pregnancy. Serial serum βhCG and progesterone level may be used in differentiating abnormal gestations from healthy intrauterine pregnancies.[11]Ultrasonography (US) is an important tool in diagnosing an extrauterine pregnancy and is able to demonstrate free fluid in the cul-de-sac.[11]Laparoscopy allows assessment of the pelvic structures, size and exact location of ectopic pregnancy, presence of hoemoperitoneum and presence of other conditions. In a patient with a ruptured gestation paracentecis may yield non-clotting

blood. In such a case other investigations need not be done. Immediate surgery is indicated when the ectopic gestation has ruptured and there is haemorrhage. This was the case with our patient. The patient should be stabilized haemodynamically with intravenous fluids and transfused with whole blood if indicated. Our patient was stabilized with fluids but did not require transfusion.

Within the last 2 decades, a more conservative surgical approach to unruptured ectopic pregnancy using minimally invasive surgery has been advocated to preserve tubal function. Laparoscopy has become the recommended approach in most cases. In our set up laparoscopy is not routinely used due to delay in diagnosis since most patients present with an already ruptured gestation.

Linear salpingostomy along the antimesenteric border to remove the products of conception is the procedure of choice for unruptured ectopic pregnancies in the ampullary portion of the tube. Some ampullary pregnancies can be teased out and expressed through the fimbrial end (milking of the tube) by using digital expression, suction, or aqua-dissection. In some cases such as isthmic pregnancies, resection of the tubal segment containing the gestation or a total salpingectomy is preferred over salpingostomy. In a patient who has completed childbearing and no longer desires fertility, in a patient with a history of an ectopic pregnancy in the same tube, or in a patient with severely damaged tubes, total salpingectomy is the procedure of choice. Our patient was done partial salpingectomy since the gestation was ruptured and the tube was badly damaged.

Postoperatively, proper pain control and haemodynamic stability are important considerations. Patients who are Rh negative should receive Rh immune globulin. Medical therapy involving methotrexate may be indicated in patients with unruptured gestation not exceeding 3.5 cm. They must be reliable, compliant, and able to return for follow-up..[13] Expectant management may be done in patients who are asymptomatic have objective evidence of resolution, such as declining β hCG levels. They must be fully compliant and must be willing to accept the potential risks of tubal rupture.

Ectopic pregnancy can lead to massive haemorrhage, infertility, or death. In Kenya it was reported to be the 6th commonest cause of overall maternal mortality.[14] FM did well after the surgery and did not develop any complications.

References

1 Centre for Disease Control and Prevention: Ectopic pregnancy--United States, 1990-1992. JAMA 1995 Feb 15; 273(7): 533.

2 Miyoro: Risk factors and Patterns of Ectopic pregnancy at Kenyatta National Hospital. Mmed Thesis, UON 2002.

3 Sinei SK, Okumu CV Ectopic pregnancy at KNH J obstet gynaec east, Cent Afric 6(1);9 1987

4 Westrom L, Bengtsson LP, Mardh PA: Incidence, trends, and risks of ectopic pregnancy in a population of women. Br Med J (Clin Res Ed) 1981 Jan 3; 282(6257): 15-8

5 Ankum WM, Mol BW, Van der Veen F, Bossuyt PM: Risk factors for ectopic pregnancy: a meta-analysis. Fertil Steril 1996 Jun; 65(6): 1093-9

6 DeStefano F, Peterson HB, Layde PM, Rubin GL: Risk of ectopic pregnancy following tubal sterilization. Obstet Gynecol 1982 Sep; 60(3): 326-30

7 Fernandez H, Coste J, Job-Spira N: Controlled ovarian hyperstimulation as a risk factor for ectopic pregnancy. Obstet Gynecol 1991 Oct; 78(4): 656-9

8 Dor J, Seidman DS, Levran D, et al: The incidence of combined intrauterine and extrauterine pregnancy after in vitro fertilization and embryo transfer. Fertil Steril 1991 Apr; 55(4): 833-4

9 Vessey MP, Johnson B, Doll R et al Outcome of pregnancy in women using an intrauterine device. Lancet 1 (7856): 495-498

10 Mwathe EG Pattern of ectopic pregnancy at KNH Mmed thesis university of Nairobi 1994

11 Stovall TG, Ling FW, Carson SA, Buster JE: Serum progesterone and uterine curettage in differential diagnosis of ectopic pregnancy. Fertil Steril 1992 Feb; 57(2): 456-7

12 Emerson DS, Cartier MS, Altieri LA, et al: Diagnostic efficacy of endovaginal colour Doppler flow imaging in an ectopic pregnancy screening program. Radiology 1992 May; 183(2): 413-20

13 Stovall TG, Ling FW: Single-dose methotrexate: an expanded clinical trial. Am J Obstet Gynecol 1993 Jun; 168(6 Pt 1): 1759-62; discussion 1762-5

14 Makokha A E, Kirumbi L W, Sekadde Kigondu C Nicholas D J,Causes and prevention of maternal mortality in Kenya. A report, 1994 Funded by USAID; FHI

15 Saraiya M, Berg CJ, Kendrick JS: Cigarette smoking as a risk factor for ectopic pregnancy. Am J Obstet Gynecol 1998 Mar; 178(3): 493-

16 Vinken S, Ellen W; Ectopic Pregnancy: April 12, 2004 *www. E medicine*

Gynaecology Case 10

LOST IUCD - RETRIEVAL

Name	H W	Age	22
Ip No.	0943029	D.O.A	20-6-04
Parity	2+0	D.O.D	20-6-04

Presenting complaint

The patient presented herself to the casualty with complains of failure to locate the intra uterine contraceptive device (IUCD) strings for 9 months.

History of presenting complaint

She had the device inserted 2 years prior to her presentation, at a family planning clinic. She never went back to follow up. She had been taught how to examine herself vaginally for the strings. She failed to locate the strings after some time, but did not seek attention immediately. She reported having been treated for vaginal discharge a month prior to her presentation and this had caused her anxiety prompting her to seek further help. At presentation she had no other complain.

Obstetric / gynaecology history

She had attained menarche at 15yrs. Her menstrual cycle was regular coming every 28 days. The flow lasted 3 – 4 days and was normal in volume. She had used Depo-Provera for contraception for 2 years during which period she had irregular menses. Her first pregnancy was in 1999 and delivered a male infant who is alive and well. Her second pregnancy was in 2001 and had a female infant who is alive and well. Both were vaginal deliveries.

Past medical histories

This was non-contributory

Family social history

She was a married lady who was a shopkeeper living with her husband. There was no known history of any chronic illness in her family. She did not smoke cigarettes or drink alcohol.

Examination

She was a young lady in good general condition. Her blood pressure was 120/70 mmhg, PR 22/min, PR 84/min, she was a febrile

Abdominal Examination

The abdomen was soft, non-tender with no organomegally.

Vaginal examination

Her external genitalia was normal. The cervix was posterior firm, long and closed. The IUCD strings were not felt. A speculum examination was done and the strings could not be visualized.

Investigations

An ultrasound of the pelvis was done. It revealed a normal uterus with the IUCD in situ. The adnexae and pouch of Douglas were normal.

Hb – 12.4 g/dl Wbc – 8.2 x 109/l Platelets - adequate

Urea, electrolytes and creatinine were within the normal range.

The findings of the ultra sound and the option of removal under general anaesthesia (GA) were explained to the patient.. An informed written consent was obtained. She was scheduled for removal of the IUCD under general anaesthesia as a day case. HW was counselled on contraception prior to the ICUD removal. She opted to use injection Depo-Provera.

Operation details

In theatre the patient was put under GA. In lithotomy position vulval vaginal toilet was done. She was catheterised and clear urine was drained.

An auvard speculum was used to expose the cervix. An antiseptic was used to clean the cervix. With a teneculum grasping the anterior lip, the cervix was stabilised. Hegars dilators were used to dilate up to size 8. After dilation a small curette was inserted into the uterine cavity and carefully rotated. The IUCD was felt, when the curette was withdrawn, the IUCD was recovered. The strings were noted to be short and had been hidden in the uterine cavity.

Discussion

The patient presented had the IUCD inserted in a family planning clinic more than two years ago. She never went back for examination even after she noticed the IUCD strings could not be felt in the vagina.

The intrauterine contraceptive device (IUCD) is made of plastic, metal or a combination of both materials. It is an effective long acting method of contraception, introduced into the uterine cavity through the cervical canal. The mechanism of action is not well established, but several mechanisms have been suggested. It is thought to exert a local inflammatory reaction within the endometrial cavity interfering with viability of both sperms and eggs. This environment is also hostile to any fertilised ovum, thus inhibiting implantation.[1]

The method is convenient since it is free of daily or coitally related activities. Fertility is restored almost immediately after removal. Some IUCD devices are coated with progesterone, which is released slowly. These are especially useful in women with menorrhagia. The blood loss during menses is significantly reduced.[2] Our patient did not have menorrhagia the IUCD she had did not contain any progesterone.

It is best to insert the device when the woman is having menses. At this time the cervical canal is open and there is less likelihood of pregnancy. Any resulting uterine cramps will be less noticeable. Insertion can be done in the immediate normal puerperium without any added risk of infection, pain or bleeding.[3] The threads extend through the cervix and the patient should be taught to feel for them. Though H W was taught how to examine herself when she noticed the strings were missing, she did not seek medical help until much later.

Failure to locate the threads may mean spontaneous expulsion, breaking or retraction of threads into the cervical canal or uterine cavity, an intrauterine pregnancy or partial or full perforation of the uterus has occurred. The patient should be examined using a speculum to ascertain if the threads are present. On examination of our patient the threads were missing.

It is important to confirm if the IUCD is in the body or not. An ultrasound examination is important in localising the IUCD in the uterine cavity or in the peritoneal cavity. In our patient the IUCD was in the uterine cavity. An x-ray examination after introducing a marker IUCD to locate the uterine cavity can also be used. The hysteroscope can be used to visualise the IUCD in the uterine cavity and the device can be retrieved at the same sitting. [4] For our patient ultra sound examination confirmed the IUCD to be in the uterine cavity.

IUCD use has been associated with pelvic inflammatory disease (PID) over the years. Recent studies suggest that risk after infection has to do with contamination during insertion. Without other risks of PID, the IUCD does not pose increased risk 3 – 4 months after insertion.[5] The patient presented had a vaginal discharge 2 years after the insertion. This is likely to be incidental but it caused her anxiety, provoking her to seek medical intervention. However in patients with current pelvic infection an IUCD should not be inserted.

Common reasons for removal of IUCD include desire for pregnancy, menorrhagia and dysmenorrhoea. Acute infection and displacement of the device are other reasons.

The IUCD is removed by steady traction on the strings. If the strings snap, a pair of artery forceps may be used successfully. Removal of difficult IUCD with no strings can be achieved under general anaesthesia. The patient presented had the IUCD in the uterine cavity but no strings hanging out. Removal was done under general anaesthesia by dilation and curettage. For devices in the peritoneal cavity after perforation, removal can be done by laparotomy or laparoscopically. Patients who are removed the IUCD when not yet ready for a pregnancy need to be counselled on other contraception method. Our patient opted for depo provera.

References

1. Anna F. Glasiers; Fertility control in Gynaecology by Robert Shaw, Patrick Scoutter, Stuart Stanton 3rd Edition, 2003. Churchill Livingstone, 30; 433- 448

2. Anderson J K Rybo G, 1990 levonorgestrel-releasing intrauterine device in the treatment of menorrhagia. British journal of obstetrics and gynaecology 97: 690 – 694

3. Treiman K. Liksin L, IUDs a new look population report series B (5) 1:31 1988

4. De Cherney AH, Polan M; Hysteroscopic management of intrauterine lesions and intractable uterine bleeding. Obstetric gynaecology 1983:61:392

5. Ronald T, Burkman M, Contraception and Family planning in current Obstetric & Gynaecologic Diagnosis & Treatment 8th edition pg 681

Gynaecology Case 11

OVARIAN CYST-UNILATERAL OPHORECTOMY DONE

Name	A K	Sex	Female
Ip No.	0966272	D.O.A.	11.6.04
Age	12	D.O.D	25.6.04

Presenting complaint

A K presented with a 3 months history of progressive abdominal swelling.

History Of Presenting Illness

The patient was well until she noticed the abdominal swelling. It progressively increased in size. Initially it was painless but later there was left sided abdominal pain. Her bowel and urinary habits remained normal. She did not have associated per vaginal discharge.

Obstetric and gynaecology

Had not yet attained menarche and she was a nulliporous.

Family And Social History

A standard 8 pupil, living with her parents in Nairobi. She was the second born in a family of 3 siblings and the others were alive and well. There was no known chronic illness in the family. She did not smoke cigarettes or take alcohol.

Examination

She was a young girl in good general condition. The vital signs were: BP- 100/60 PR – 80/minute

RR – 22/ minute and a temperature of 36.6^0 C

Breasts were noted to be at Tanner stage 2

The respiratory, cardiovascular and central nervous systems were normal.

Abdominal Examination

An obvious asymmetrical distension was noted. The mass was in the suprapubic

region extending more to the left iliac fossa. It was firm, non-tender, mobile and arising from the pelvis. It corresponded to 20 weeks of gestation.

A pelvic examination was not done since the hymen was intact.

Investigations

Abdominal ultrasound revealed normal abdominal structures. The pelvic ultrasound showed a large bilateral multiseptated tubo-ovarian cystic mass, 15 cm in diameter

Hb – 12.7 g/dl Urea and Electrolytes – normal

Liver Function Tests – normal

Management

The diagnosis was explained to the patient and her parents. They were informed of the need for an operation. Informed consent was obtained from the father since the patient was a minor. She was then scheduled for laparotomy. She was starved from midnight the night before the operation. In the morning she was pre-medicated with atropine 0.6 mg intramuscularly half-hour before theatre.

Operation

In theatre she was put under general anaesthesia. She was aseptically catheterised after vulvovaginal toilet was done. The abdomen was cleaned with an antiseptic and draped. The abdomen was opened via a sub-umbilical midline incision.

A large left ovarian cyst with solid cysts elements about 15 cm in diameter was found. The left tube was matted to the cystic ovary. The cyst was removed whole without puncturing it together with the ovary and the left tube. Grossly it appeared like a dermoid cyst. It was sent for histo-pathological examination.

The right ovary and tube and the uterus were grossly normal. Peritoneal lavage with warm normal saline was done. The abdomen was repaired in layers.

She did well post operatively and was discharged on the 4th postoperative day. Histology showed features of a mature benign cystic teratoma. It had numerous glands lined with gastric type epithelium, fibroblasts and cartilaginous tissue.

Follow up

She was seen in the gynaecology clinic after 2 weeks. She had healed well with no complains. The histology results were explained to the mother and the child. They were reassured concerning the future fertility. She was discharged from the clinic.

Discussion

A K was a 12-year-old pre pubertal girl who presented with an abdominal swelling. She was found to have a benign ovarian cyst and a unilateral salpingo-ophorectomy was done.

Benign ovarian cysts are common, frequently asymptomatic and most resolve spontaneously. About 90% of ovarian tumours are benign but this varies with the age.[1] The ovarian tumours are either physiological or pathological arising from any tissue within the ovary. They may fall into any of these classes; physiological, germ cell, epithelial, sex cord, stromal or metastatic.

Physiological cysts are larger versions of the cysts that form on the ovary during the normal ovarian cycle. They commonly occur in young women. They are of two types, the follicular and the luteal cysts. Germ cell tumours are common in women under 30 years of age with 2 – 3 % being malignant, but in women under 20 years the proportion may rise to a third. They arise from totipotent cells and can therefore contain elements from all the three germ layers. The patient discussed had a germ cell cyst. It contained gastric type epithelium, fibro-fatty and cartilaginous elements. She had a dermoid cyst (mature cystic terotoma), which is the commonest benign germ cell tumour.

Epithelial tumours form majority of ovarian neoplasia, both benign and malignant. They may be mucinous, serous, endometroid, brenner or clear cell (mesonephroid) tumours. Sex cord stromal tumours often produce hormones that can lead to post menopausal bleeding in older women and sexual precocity in pre-pubertal girls. The hormones could also cause glandular hyperplasia or occasionally carcinoma of endometrium.[2] Metastatic tumours of the ovary accounts for as many as 25% of all ovarian malignancies. Most simple cysts resolve spontaneously if observed for a period of six of months.[3]

Most benign ovarian tumours are asymptomatic and are found incidentally when investigating for a different problem. However patients may present with acute pain following torsion, rupture, haemorrhage or infection. Other symptoms may be abdominal swelling and chronic low abdominal pain, usually noted when the tumour

is massive. Hormonal effects may be noted in hormone producing tumours. Gastrointestinal or urinary symptoms may result from pressure effects. Rarely oedema of the legs, varicose veins and haemorrhoids may result. Our patient noted a growing abdominal mass but did not seek medical attention until there was pain, probably due to pressure.

Investigations required depend on the circumstances of presentation. Acute symptoms often require emergency surgery but asymptotic patients or those with chronic problems need more detailed assessment.

Ultrasound examination has reasonable sensitivity and specificity in demonstrating ovarian masses. Solid masses are more likely to be malignant than the cystic ones. In our patient ultrasound showed a cystic mass 15cm in diameter. Some have done ultrasound guided ovarian cyst aspiration for cytology. The technique has up to 71% false negative rate and 2% false positive rate for diagnosis of malignancy. Some risk of malignant cell dissemination exists and it is not recommended as a diagnostic tool.[4] A chest x-ray is done to detect metastatic disease and pleural effusion. A plain abdominal x-ray may show calcification in case of a teratoma. An intravenous urogram may be done for huge masses likely to distort the anatomy of the ureters. Ca 124 measurements are used in various malignancy risk indices but the predictive value is rather unsatisfactory.[1] β hCG concentration may be measured to exclude ectopic pregnancy. Trophoblastic tumours and some germ cell tumours secrete βhCG. Other hormones secreted by different ovarian neoplasm include, oestrogen, androgen and α- fetoproteins.

Management depends on severity of the symptoms, malignancy risk and the desire for children. Simple cysts in young women less than 40 yrs of age are likely to be benign. Cysts 3cm in diameter or less are likely to be functional. Those 3 – 8 cm in diameter need follow up for 3 – 6 months. For bigger cysts laparoscopy or laparotomy is indicated. The patient discussed had a cyst larger than 8 cm and laparotomy was done. Older women especially those above 50 yrs are more likely to have a malignancy. Some suggest that simple echo-free unilateral cysts without solid parts or papillary formation and less than 8 cm in diameter are likely to be benign and may be safely

managed conservatively with 3 - 6 monthly ultrasound and Ca 125 estimation[5] However some authors feel that all primary ovarian tumours must be removed due to potential risk of malignant change. [6]

Patients with severe acute pain or signs of intra-abdominal bleeding need emergency laparoscopy or laparotomy. In the pre-pubertal girls, ovarian cysts are uncommon and often benign. When they occur the teratoma and follicular cysts are the most common. Our patient was pre-pubertal and had a teratoma.

For patients who need surgery, it is prudent to do a bimannual examination under anaesthesia to confirm the presence of the mass. If the disease seems benign conservation of the maximum ovarian tissue is done in the young girl. Unilateral salpingo-ophorectomy is the treatment of choice for women under 40 yrs with benign disease. It is essential to explore the whole abdomen thoroughly and to inspect both ovaries to ensure no other pathology exists. Our patient was a young girl it was not possible to conserve any ovarian tissue of the affected side, a unilateral salpingo-ophorectomy was done. Her other ovary was noted to be normal.

References

1. Soutter PW, Girling J. Haidopoulus D; Benign tumours of the ovary, Gynaecology by Robert W. Shaw, 3rd edition 2003, Churchill and Livingstone.44; 665-675

2. Fox H, Lang F A; 1976, Tumours of the Ovary Heinemann, London pg 119 – 137.

3. Sasaki H., Oda M, Ohmura H et al 2001, Preoperative assessment of unilocular cysts by transvaginal ultra sonographic morphologic imaging and pathologic diagnosis. American Journal of Obstet and Gynae 184; 48-54

4. Diernaes E, Rasmusen J. Soergen T, Hasche E 1987, Ovarian cysts: Management by puncture? Lancet: 1084.

5. Ekerrhoud E, Weinerroith H et al, 2001 Preoperative assessment of unilocular cysts by transvaginal ultrasonography: a comparison between ultrasonographic morphologic imaging and histopathological diagnosis. American journal of Obst and Gynae 184: 48 – 54

6. Houkins S. Hudson C N. Surgery of benign conditions of the ovary and broad ligament in Howkins Hudson CN Eds. Show text book of operative gynaecology, 5th edition longman group London pp 215, 1983

Gynaecology Case 12

VESICO – VAGINAL FISTULA; SUCCESSFUL REPAIR

Name	M. M.	I. P. No.	09772375
Age	18	DOA	28.7.04
Parity	1 + 0	DOD	05.8.04

Presenting Complains

M M presented to the gynaecology outpatient clinic as a referral form Mwingi District Hospital with leakage of urine for 4 months.

History of presenting illness

The patient developed the leakage or urine following a difficult vacuum delivery. She had been in labour for 2 days before presenting herself to the district hospital. The outcome of the delivery was a fresh stillbirth male infant weighing 3500gms. The urine leakage started soon after the delivery no catheter was inserted at that time. She did not have associated stool leakage or difficulty in walking. She was then discharged home and did not go back for follow up till 3 months later when she noted the problem was not improving.

Obstetric and gynaecological history

She was a Para 1 + 0. Her last delivery was in March 2004 as described above. During the pregnancy she had not attended antenatal clinic. She attained menarche at 15 years. Her menstrual cycle was 28 days with a normal flow of 3 – 4 days. The cycle was regular. She had never used any contraception.

Past medical history

This was not significant.

Family and social history

She was married and lived in Mwingi with her husband. She had dropped out of school in class 6 due to lack of fees. Both of them were unemployed. There was no known chronic illness in her family. She did not smoke cigarettes or drink alcohol.

Physical examination

She was in stable general condition, not pale or jaundiced. She had no oedema. Her vital signs were normal; blood pressure 110/70 mmHg. Pulse rate 80/min, respiratory rate 18/min and temperature 36.6°C. She was 160cm tall.

The central nervous, cardiovascular and respiratory systems were essentially normal. Abdominal examination findings were within normal.

Pelvic examination

The perineum had excoriations. There was a strong smell of urine. She was wearing a piece of cloth that was wet with urine. Speculum examination revealed a vesico vaginal fistula, 2 cm from the external urethral opening measuring 2 x 1.5cm. The fistula was class II Aa since it was not involving the urethra closing mechanism and was not circumferential.

Management

She was explained to the nature of her problem and the need for further examination under anaesthesia and repair of the fistula.

Investigations done included,

Haemoglobin – 12.5g/dl	Sodium – 142 mmol/l
Creatinine – 65 μmol/l	Potassium - 4.5 mmol/l

The patient was admitted the day before the surgery. She was given fluid diet and an enema in the evening prior to the surgery. She fasted overnight and shaving of the pubic hair was done. An informed consent was taken. 30 minutes before being taken to theatre atropine 0.6mg intramuscularly was given.

In theatre she was put under general anaesthesia and placed in lithotomy position. The perineum was cleaned and draping done. Examination under anaesthesia was carried out. The fistula was noted to be 2 cm was the external urethral meatus, measuring 2 x 1.5cm. It was not circumferential. A left mediolateral episiotomy was done and an auvards speculum inserted into the vagina to expose the vaginal cavity. Jungle juice (solution of adrenaline and hydrocortisone in normal saline) was infiltrated around the

fistula. Circumferential distension was done around the fistula with extension bilaterally. The vaginal mucosa was dissected and the bladder mobilized away. The fistula was then closed using vicryl no 2/0 interrupted mattress sutures starting laterally proceeding medically. Methylene blue dye was introduced into the bladder and no leakage was noted. A vaginal pack soaked in iodine was left in the vagina to be removed after 24 hours. The foley catheter was left insitu. General anaesthesia was reversed successfully, and the patient taken back to the ward after she was fully awake.

Postoperative care

She was given analgesics to relieve pain and advised to take 6 – 8 litres of fluids per day. The catheter was draining urine and there was no leakage. After 24 hours the vaginal pack was removed and no bleeding was noted. On the fifth postoperative day she was discharged home to continue with oral fluids and with the catheter insitu.

She came back for review on the 14th postoperative day. Dye test done was negative. The catheter was removed and she was advised to do pelvic floor exercises. She was advised not to have coitus for 3 months. In case of a pregnancy she was to book antenatal early and delivery by caesarean section.

Discussion

M M was an 18-year-old woman who presented with leakage of urine for 3 months. She was found to have vesico-vaginal fistula that was successfully repaired.

A fistula is an abnormal communication between two epithelial surfaces. Vesico-vaginal fistula (VVF) is an abnormal fistulous tract extending between the bladder and the vagina that allows the continuous involuntary discharge of urine into the vaginal vault. Typically the fistula involves the bladder, urethra, bladder trigone, and the anterior cervix. Less frequently, fistulas may occur (1) between the bladder and cervix or uterus; (2) between the ureter and vagina, uterus, or cervix; and (3) between the urethra and vagina. A ureteric injury is identified in association with 10-15% of VVFs. (4) between the rectum and the vagina (RVF). [1]

The frequency of VVF largely is underreported in developing countries. Margolis cites an incidence of 3-4 cases per 1000 deliveries in West Africa while Kees reports an annual incidence of 100 000 to 150 000 cases of new VVF patients annually in Africa. An estimated 1.5 million patients are awaiting surgery.[2,3]

In developing countries, the predominant cause of VVF is obstetric injuries usually following prolonged obstructed labour. These account for over 90% of the reported fitula.[4] The effect of prolonged impaction of the foetal presenting part in the pelvis is one of widespread tissue oedema, hypoxia, necrosis, and sloughing resulting from prolonged pressure on the soft tissues of the vagina, bladder base, and urethra.

Numerous factors contribute to the development of VVF in developing countries. There are areas where the culture encourages marriage and conception at a young age, often before full pelvic growth has been achieved. Chronic malnutrition further limits pelvic dimensions, increasing the risk of cephalopelvic disproportion and malpresentation. A study done in Nairobi showed the average true conjugate among women sustaining fistula to be 9 centimeters.[5] In addition, few women are attended by qualified health care professionals or have access to medical facilities during childbirth; their obstructed labour may be protracted for days. Other cultural factors that increase the likelihood of obstetrical fistulae include outlet obstruction due to

female circumcision and Gihiri incisions (anterior vaginal walls incisions).[1] M M grew up in a poor family and possibly experienced malnutrition as a child. She dropped out of school and got married as a teenager. She had a delivery at 18 years of age.

Isolated VVF is the commonest accounting for 85% of obstetric fistula. Combinations of VVF and rectovaginal fistula occur in 10 – 15% of the cases. Isolated RVF is very rare except for the case of 4th degree perineal tear. In most cases the baby is born already dead (still birth) or dies soon after birth. The patient discussed had an isolated VVF. She had a stillbirth.

Other complications of prolonged obstructed labour include vaginal stricture, stenosis, shortening or atresia. Secondary amenorrhoea may follow endometrium trauma or Sheehan's syndrome. Peroneal nerve palsy of the sciatic nerve affecting only the efferent motor fibres can occur. Loss of tissue such as the labia minora, posterior urethra / bladder or anterior rectum can occur. Systemic complications may include poor general health, pressure sores over the sacrum trochanter major, heel and scapula. Complex neuropathic bladder dysfunction and urethral sphincteric incompetence often result; even if the fistula can be repaired successfully .The levator ani and perineal muscles together with their nerves are also at risk. MM did not have other complications except the VVF.

VVF classification according to anatomic or physiologic location.[3]

Type I	not involving the bladder closing mechanism and are usually at least 5 cm away from the external urethral meatus.
Type II	involves the bladder closing mechanism. It includes fistulae that lie 5 cm from the external urethral meatus
II A -	without total urethral involvement
A a -	without circumferential defect
A b -	with circumferential defect
II B -	With total urethral involvement
Ba -	without circumferential defect

Bb - with circumferential defect

Type III - all other forms of fistulae e.g. uretero – vaginal fistula.

Classification according to the size;

Small – less than 2 cm

Medium – 2 – 3 cm

Large – 4 – 5 cm

Extensive – over 6 cm

Circumferential involvement means the whole urethral circumference has been involved (urethra has been divided into two portions).

The patient discussed had a medium fistula which was 2 cm form the urethral opening i.e. involving the bladder closing mechanism but was without circumferential involvement. This was class II Aa

Women in early labour should have proper assessment of the pelvis to avoid prolonged labour that leads to fistula and other complications. Any woman noted to be having obstructed labour should be relieved within 3 hours. The bladder should then have continuous drainage by an indwelling catheter for at least 10 days. Prompt relieve of obstruction followed by continuous bladder emptying with high fluid intake improve the healing of the devitalised tissues reducing the risk of fistula formation. Improvement of child nutrition may reduce the incidence of contracted pelvis thus reducing the incidence of obstructed and the sequelae.

Other less common causes of VVFs include to inadvertent bladder injury during pelvic surgery, pelvic infections (tuberculosis, syphilis, lymphogranuloma venereum), vaginal trauma, pelvic irradiation and vaginal erosion with foreign objects (neglected pessary). A congenital urogenital abnormality may exist that includes a VVF.

In general, no absolute contraindications exist for the attempted correction of a VVF in patients who can medically tolerate a surgical procedure. If the fistula is small, less than 1 cm, the fistula has a high spontaneous cure rate with 3-week continuous bladder drainage. [6]

The timing of repair should be dictated by the overall medical condition of the patient and the tissue quality surrounding the fistula. While the emotional status of the patient should not be underestimated, it also should not play the dominant role in the decision process of when to repair a VVF. Many authorities recommend delaying surgery until inflamed and infected tissue has been treated and the infection and inflammation have resolved.[2,7] Some have advocated vaginal oestrogen therapy in the waiting period to improve the health of the tissues. Consideration to adjunctive steroid therapy may be contemplated.[7] By the time MM came to KNH her fistula was 4 months old and inflammation had subsided and she was not infected. Repair was done with good results.

References

1. Rizvi JH: Genital fistulae; A continuing tragedy. J Obstet Gynaecol Res 1999 Feb; 25(1): 1-7
2. Margolis T, Elkins TE, Seffah J: Full-thickness Martius grafts to preserve vaginal depth as an adjunct in the repair of large obstetric fistulas. Obstet Gynecol 1994 Jul; 84(1): 148-52
3. Kees Waaldijk. The obstetric fistula ; VVF and RVF quick reference. P 1-23.
4. Tindal V.R, Injuries; Jeffcoate's principles of Gynaecologic management, 5th Edition Butterworths, 1987, pg 238
5. Gebbie D A Obstetric and gynaecology in Health and Disease in Kenya, Vogel L C (ED): Nairobi E A L B, 1974, pg 497
6. Zimmern PE, Hadley HR, Staskin D: Genitourinary fistulas: vaginal approach for repair of vesicovaginal fistulas. Clin Obstet Gynaecol 1985 Jun; 12(2): 403-13.
7. 7O'Conor VJ Jr, Sokol JK, Bulkley GJ: Suprapubic closure of vesicovaginal fistula. J Urol 1973 Jan; 109(1): 51-4

Gynaecology Case 13

Endometrial Carcinoma Stage 1: Total Abdominal Hysterectomy and Bilateral Salpingo – Ophorectomy

Name	F. W.	I. P. No.	0987330
Age	65	DOA	13.10.04
Parity	0+0	DOD	23.10.04

Presenting complaint

The patient was admitted with complains of postmenopausal bleeding for 1 month.

History of presenting complaint

The patient had been admitted in ward 1 D with per vaginal bleeding for one month. The bleeding was mild coming on and off. While ward 1 D a fractional curettage was done. Histology showed differentiated adenocarcinoma of the endometrium. She was then transferred to ward 1 B for further management. She had no per vagina discharge. Her bowel motions and micturition were within normal. She had been diagnosed to have diabetes mellitus 6 months prior to the development of these complains. The blood sugar was well controlled on diet. She was also known to have hypertension for the past 2 years.

Obstetric and gynaecological history

She was Para 0 + 0. She had attained menarche at 14 years. Her cycle had been 28 days with a flow of 3 – 4 days. She was now 15 years post menopausal. She had never used any form of contraception. She had never had a pap smear done.

Past medical history

She was a known diabetic for 6 months controlled on diet. She was hypertensive for 2 years controlled on aldomet and adalat

Family and social history

She was widowed for 20 years. She was living with her relatives in Eastleigh. She did not smoke cigarettes or take alcohol. Her mother had been hypertensive.

Physical examination

She was elderly obese lady in good general condition. She was not pale jaundiced or cyanosed. She had no oedema or lymphadenopathy. The vital signs were; BP 130/85mmhg, PR 84/min, RR 70/min, temp 36.6°C.

Respiratory, cardiovascular and the central nervous system were within normal.

Abdominal examination

The abdomen was obese but no obvious distension. It was moving with respiration. There were no areas of tenderness. No masses were palpable.

Pelvic examination

The external genitalia was atrophic. The vaginal walls and the cervix appeared normal grossly. Bimanual examination revealed a bulky mobile non-tender uterus. The adrexae and pouch of Douglas were within normal.

Diagnosis

A diagnosis of abdominal uterine bleeding secondary to endometrial carcinoma was made.

Investigations

Haemogram	12.5 g/dl	K	3.0 mmol/l
WBC count	8.2 x 10^9/l	Na	135 mmol/l
FBS	5.6 mmol/l	creatinire	90 mmol/l
Urea	3.0 mmol/l		

Pelvic ultrasound – bulky uterus with an endometrial stripe of 15 mm. The ovaries were normal.

Abdominal ultra sound – normal liver, kidney and spleen, no fluid collection

Chest x-ray normal

Procedure

The patient was explained to the nature of her problem and the need for the operation. She gave a written consent. Blood was taken for grouping and cross matching and 2 units of blood were kept ready. The patient was not fasted overnight but her breakfast on the morning of the surgery was omitted. She was premeditated with pethidine

100mg intramuscularly but atropine was not given. A fasting blood sugar taken that morning was 5.8 mmol/l and the blood pressure was $^{125}/_{85}$mmhg. She was started on a 5% dextrose infusion and 10 IU of soluble insulin was added.

In theatre she was put under general anaesthesia. A vulvovaginal toilet was done and catheterisation done. Pelvic examination was done and earlier findings were confirmed. The abdomen was cleaned and draped, and opened via a sub umbilical midline incision. Intra-operatively, the uterus was found to be bulky corresponding to 12 weeks gestation. Both ovaries were identified and grossly looked normal though atrophic. The omentum and gut looked normal. There was no ascites. The liver and spleen were normal with no lymphodenopathy noted. Also the rectum and bladder were grossly normal.

A total hysterectomy and bilateral salpingo ophorectomy was done. The specimen was taken for histology. The operation took 2 hours. The blood sugar and blood pressure remained within the normal range.

Post operatively the patient did well. The first 24 hours she received soluble insulin 10 IU when the blood sugar rose to 10 mmol/l. By the second day the blood sugar settled and she no longer needed insulin. She continued with her antihypertensive treatment and the blood pressure was well controlled. The healing was not delayed. On the fifth post operative day she was discharged form the hospital.

Follow up

She came to the gynaecological outpatient clinic after two weeks. The wound was well healed. The histology report had shown well-differentiated adenocarcinoma of the endometrial glands that was confined to the endometrium. The patient together with her brothers who had come with her was explained to the results. She was to come again for follow up in this same clinic.

Discussion

F W presented with postmenopausal bleeding. She was found to have carcinoma of the endometrium and total abdominal hysterectomy with bilateral salpingo-ophorectomy was done.

Endometrial cancer is the most common gynaecological malignancy affecting women in the West.[1] The peak incidence of onset is in the sixth and seventh decades but 2 – 5% occur before the age of 40yrs [2]. There is a remarkable geographical and racial variation in incidence; it is highest among the North American whites and lowest among the Asians. Asian women migrating to North America develop similar incidence rates those of the Americans [3].

The precise aetiology is unknown but several factors influence its development. Classically it affects the affluent obese, nulliparous, infertile, hypertensive and diabetic white woman, though can occur in the absence of these factors. States associated with high oestrogen levels such as diabetes, hypertension, obesity and polycystic ovary syndrome are associated with higher incidence of endometrial cancer, [2, 3, 4] Early menarche and delayed menopause are associated with increased risk. [5] Nullipality is associated with a 2 – 3 fold increased risk. The risk falls with increasing number of children. [6] Use of unopposed oestrogen therapy increases risk of endometrial cancer and the risk increases with the duration of exposure. [7] Other factors that appear to increase the risk of endometrial cancer are breast cancer and tamoxifen therapy. [8]The patient discussed was 65 years old, obese, nulliporous, diabetic and hypertensive.

Endometrial malignancy may arise from the glands or stroma of the endometrium. The commonest is the one arising from the endometrial glandular cells. It has been postulated that it may present the end process of a spectrum beginning with hyperplasia, passing through atypical hyperplasia and ending with frank cancer. But only about 25% of patients with endometrial carcinoma have history of pyperplasia.[2] It is therefore likely that it can develop independently of atypical hyperplasia. Important prognostic factors include lymph node involvement, myometrial invasion, histological grade and cell type, positive peritoneal cytology and elevated serum CA 125. Serous papillary and clear cell cancers have the highest risk of lymph node

involvement. Pelvic and aortic lymph node metastasis is directly proportional to the depth of myometrial invasion and the degree of differentiation.

The spread of endometrial carcinoma is by direct extension, lymphatic metastases, peritoneal implants following transtubal spread and haematogenous spread. The tumour remains confined to the body of the uterus for a long time before invading the myometrium and the cervix. Onset of endometrial bleeding facilitates early detection of the disease. Hence it has an overall good prognosis. Un differentiated lesions (grade 3) may spread to the pelvic and aortic nodes while still confined to the superficial myometrium. Our patent had a well-differentiated adenocarcinoma of the endometrial glands that was confined to the endometrium.

There seems to be two different types of endometrial cancer. The most common type is found in younger peri-menopausal women with unopposed oestrogen exposure. The tumour usually begins as hyperplasia and progresses to carcinoma. This type tends to be better differentiated with a better prognosis. [9] The other type of endometrial cancer occurs in the absence of hyper-oestrogenism. It may arise in the background of atrophic endometrium. This type of carcinoma tends to be less well differentiated with a poorer prognosis. It is common in older postmenopausal women.

There are 3 major histological types of endometrial carcinoma; endometriod adenocarcinoma, adenocarcinoma with squamous differentiation and adeno squamous carcinoma. The endometrial adenocarcinoma is the commonest type accounting for over 80% of the carcinomas. They can be well (grade 1), moderately (grade II) or poorly differentiated (grade III). The adenocarcinoma with squamous differentiation tends to be very well differentiated. The adenosquamous carcinoma is often poorly differentiated. Other histological types include the papillary serous carcinoma and clear cell carcinoma. Pure squamous cell carcinomas are extremely rare. The patient discussed had well differentiated endometriod adenocarcinoma.

About 80% of women with endometrial carcinoma present with abnormal uterine bleeding. Any postmenopausal woman who bleeds even slightly has a high probability of having a gynaecological malignancy and should be investigated. Some women may present with an abnormal vaginal discharge. F W presented with vaginal bleeding for which she was investigated.

A small majority may be diagnosed as having abnormal cervical cytology. Presence of pain usually indicates metastatic disease. Dull pain may be due to pyometra or haematometra in postmenopausal women with cervical stenosis

Physical examination is usually unremarkable especially in early disease. With advanced disease, the uterus may be enlarged and vaginal metastasis may be visible. In late disease the uterus is fixed and immobile. A variety of techniques can be used to evaluate a woman with suspected endometrial cancer. Transvaginal scanning is useful in evaluating the endometrial stripe. Endometrial malignancy is unlikely when the endometrial stripe is 5mm or less.[10] Endometrial biopsy can be done as an outpatient procedure. Aspirating or non-aspirating curettes maybe used. Aspirations biopsy using a Novaks curet is 80 – 90% accurate. [2] The pipelle thin plastic cannula may be more acceptable to patient. Aspiration curettage with a vabra aspirator has an overall accuracy of 95 – 98%. [2]

Dilation and fractional curettage under general anaesthesia is the definitive procedure for diagnosis of endometrial carcinoma. It is involves careful and complete curettage of the endocervical canal followed by dilation and curettage of the endometrial cavity. This was done for F. W. Hysteroscopy may be done. It is accurate in detecting endometrial polyps and sub mucous fibroids but visual identification of endometrial carcinoma is unusual. Biopsy must be taken. [12]

A cervical smear should be done. It is useful in detecting cervical cancer. A full blood count, urinalysis, serum creatinine and blood sugar should be done routinely. A chest x-ray to rule out metastases and intravenous urogram to confirm the anatomy of he genitourinary system should be done. Ca 125 may be useful for follow up. MRI is the most accurate radiological examination for assessing local disease spread.

Staging of the carcinoma is done after surgery. Surgical staging by FIGO is as follows. [13]

Stage 1

1 a tumour limited to the endometrium

1 b invasion of less than one-half the myometrium

1 c invasion to more than one-half the myometrium

Stage II

II a endocervical glandular involvement

II b cervical stromal invasion

Stage III

III a tumour invades serosa and or adnexa and or positive peritoneal cytology

III b vaginal metastasis

III c metastasis to pelvic and or para aortic lymph nodes

Stage IV

IV a tumour invades bladder and or bowel mucosa

IV b distant metastases including intra abdominal and /or inguinal lymph Nodes

The treatment offered to the patient depends on the stage of the carcinoma and the medical condition of the patient. The treatment of choice is abdominal hysterectomy and bilateral salpingo ophorectomy especially since clinical staging is inaccurate. [14]

In early disease simple hysterectomy is sufficient done abdominally or vaginally. A midline umbilical incision allows the best access to the abdominal cavity since it can be extended if the para aortic nodes are thought to be involved. Abdominal approach is superior for removal of ovaries and assessment of the peritoneal cavity and retroperitoneal nodes. Once the abdominal cavity is entered, washings should be taken from around the uterus, bladder and pouch of Douglas. The liver and omentum should be inspected for any involvement. Pelvic lymphadectomy may be done and partial omentectomy. This is done for grade 2 and 3 tumours as well as grade 1 tumours that are obviously large and invading the myometrium. In up to 8% of cases the infracolic omentum will contain microscopic disease. [15] The vaginal approach is preferred for patients who are older and unfit, to avoid the more severe morbidity associated with the abdominal approach. In this group of patients the vaginal approach has better outcome. [16] The abdominal approach was done for our patient.

Radiotherapy is indicated after surgery in stage 1 disease in case of poor prognostic factors such as invasion more than half way through the myometrium, high-grade tumours and large tumours. For patients not fit for surgery due to their medical condition or due to more advanced disease radical radiotherapy is given without surgery .[3] Our patient did not require radiotherapy.

For patients with stage II disease found to have only microscopic involvement of the cervix, their prognosis is similar to those with stage I tumours and can be managed as stage I. [17] For patients with macroscopic spread to the cervix radical hysterectomy and bilateral pelvic lymphadectomy is done if fit for surgery. If unfit for surgery radiotherapy alone is used.

For stage III disease if the spread is confined to the pelvis radiotherapy is the treatment of choice. For those found to have macroscopic spread to the ovaries at surgery, abdominal exploration should be done as in ovarian cancer, and omentectomy done.[18]

Few patients have distant metastasises (stage IV) at the time of diagnosis. The lungs are the most common site of metastases followed by peripheral lymph nodes and bladder. The primary aim in this group should be control of symptoms and local control of tumour growth. If metastases are to the lung only, simple hysterectomy and bilateral salpingo ophorectomy or vaginal hysterectomy can be done followed by progestin therapy. If pelvic or para aortic nodes are involved, adjuvant radiation can be offered.

The papillary serous and the clear cell tumours have poorer prognosis. More extensive surgical staging should be undertaken. For those with disease confined to the uterus. No further adjuvant treatment is necessary. If pelvic metastatic disease is found radiotherapy is appropriate. If extra pelvic disease is present intravenous cytotoxic therapy with cisplatin, adriamycin and cyclophosphomide is reasonable.

Follow up after treatment is important in order to detect recurrent disease, detect and treat complications of treatment and manage other related problems.

The common sites of recurrences are the pelvis and the vagina, peritoneal cavity, lungs, liver, bone and inguinal or supraclavicular nodes. Radiotherapy in patients with isolated vaginal disease not previously irradiated can cure 33 – 60% of the cases.[19] Radiotherapy is also helpful for palliation of symptoms especially relief of pain and discomfort due to bony and nodal metastases very few patients will be fit enough for the extensive surgery that may be required. Progesterone therapy has been used with some success. About 80% of progesterone receptor positive and 70% oestrogen receptor positive tumours respond to progesterone treatment .[20] Cytotoxic agents have a role following failure of hormonal therapy. These include adriamycin, cisplatin and carboplatin cyclophosphamide and hexamethylemamine.

About 80% of women with recurrent disease die within 2 years.[21] With the very poor outcome of treatment for recurrent disease it is important to limit the degree of aggressive treatment since it might only serve to increase the morbidity.

References

1. Creasman W. Odicino F, Maisonneavuve P et al Carcinoma of the corpus uteri. Figo annual report on the results of treatment of gynaecological cancer. Journal of Epidemiology Biostatics 3; 35-61, 1998
2. Annekathryn Goodman, Oliver D. pre malignant & malignant disorders of the uterine corpus, current obstetric and Gynaecological diagnosis and treatment, 9th Edition Appleton & Lange.
3. Quinn M, Bleddy J, Dinar Soutter P, Malignant disease of the uterus. Gynaecology by Shaw. 3rd Edition 2003 Churchill Livingstone.
4. Quinn MA, Brown SB, Enms G, circulating gonadotrophins and urinary eostrogens in post menopausal diabetic women. Australian and New Zealand journal of obstetrics and gynaecology. 21; 234 – 236 1981
5. Petterson B. Adami HO, et al menstruation span – a time – limited risk factor for endometrial carcinoma, acta obstetrica & gynaecological. Scandinavia 65; 247 – 255 1986
6. Parazzini F, La Vecchia, Negri E et al, Reproductive risk factors and risk of endometrial cancer. American journal of obstetrics & gynaecology 164; 522 – 527 1991
7. Grady D, Gebretsach T, et al Hormone replacement therapy and endometrial cancer risk; a meta-analysis – obsetrics and gynaecology 85; 304-313 1995
8. Fisher B, Wickerman DL, constantino JL tamoxine for the prevention of breast cancer; Report ofhte NSA BP – P1 study, Journal of the national cancer institute 90; 137 – 1383 1998
9. Bukkhman JV, Two pathogenete types of endometrial carcinoma. Gynaecol oncol 1983, 15; 10- 17
10. Smith – Bind man R, Kerlikowski K, et al, Endovaginal ultrasound to exclude endometrial cancer and other endometrial abnormalities. Journal of American medical association 280; 1510-1517, 1998
11. Eddowes H A, A more acceptable technique for outpatient endometrial biopsy. British journal of obstetrics and gynaecology 97; 961 – 2, 1990
12. Ben-Yehuda om, Kim YB, Leuchter RS does hysterectomy improve upon the sensitology of dilation and curratage in the diagnosis of endometrial hyperplasia

or carcinoma? Gynaecological oncology 68; 4 – 7, 1998

13. International Federation of gynaecology and obstetrics; Annual report on the results of treatment in gynaecologic cancer. Int gynaecol obstet 36 (suppl); 1991
14. Cowles TA, Margina JF et al, Comparison of clinical and surgical staging in patients with endometrial carcinoma obstetrics and gynaecology 66; 413 – 416 1985
15. Saygiliu, Kavaz et al omentectomy, Peritoneal biopsy and appendectomy in patients with clinical stage I endometrial carcinoma international Journal of Gynaecological cancer 11; 471-474 2001
16. Chan JK, Lin Y G et al Vaginal hysterectomy as primary treatment of endometrial cancer in medically compromised women. Obstetrics and gynaecology 97; 707 – 711, 2001
17. Mangionic, de Palo G, Marubini E et al surgical pathological staging in apparent stage I endometrial carcinoma. International Journal of Gynaecological cancer 3; 373 – 384 1993
18. Gersxten K, Faw C, Haung Q Pathological stage III endometrial cancer treated with adjuvant vacliatin therapy. International Journal of Gynaecological cancer 9; 242 – 246
19. Creutzbergc, Van Putten W, Koper P et al surgery and post operative radiotherapy versus surgerry for patients with stage I endometrial carcinoma; multicentre randomised trial. Lancet 355; 1404 – 1411. 2000
20. Quinn MA, Kneale BL, Fortune DW, Endometrial carcinoma in pre-menopausal women; a clinical pathological study. Gynaecological oncology 20; 298 – 306 1985
21. Abeler VM, Kjorstad K E, Berte E carcinoma of the endometrium in Norway; a histopathological and prognostic survey of a total population. International journal of gynaecological cancer 2; 9 – 22, 1992

Gynaecology Case 14

PELVIC ABSCESS LAPAROTOMY DONE

Ip No.	0960082	D.O.A.	20.6.04
Name	J A	D.O.D	24.6.04
Age	23	Parity	2 + 0

Presenting Complaint

The patient came complaining of low abdominal pains for two weeks.

History Of Presenting Complaint

The patient had been well prior to the onset of the abdominal pain. The pain had started gradually but increased with time, and had become severe in the last two days prior to admission. Walking aggravated it. She reported associated hotness of body, chills and nausea. She had vomited two times the previous day. She had no diarrhoea. She was passing urine normally. She had foul smelling per-vaginal discharge. She had been treated twice for vaginal discharge in a private clinic.

Obstetric / Gynaecology History

Her first delivery was in 1998 to a male infant. Her second delivery was in 2001. Her last normal menstrual period was on 5.6.04. She attained menarche at 14 years. Her cycle was regular after every 28 days. The flow was normal lasting 3 – 4 days. She occasionally had unprotected sex and was not on any contraception.

Past medical history

This was not contributory

Family / social history

She was separated from her husband and sold vegetables in the market. There was no history of chronic illness in the family. She did not smoke cigarettes or take alcohol.

Examination findings

She was sick looking, not pale or jaundiced. Her BP was 120/70 mmHg, PR – 94/min, temp 38.5°C, PR – 26/min

Respiratory system

She had tachypnoeia but her chest was clear.

Cardiovascular and central nervous system were essentially normal.

Abdominal Examination

The abdomen was not obviously distended but was tender on palpation. It was rigid with guarding. There was no was no palpable mass.

Pelvic Examination.

This revealed normal external genitalia. The cervix was long, closed and posterior. The uterus was normal size, there was marked tenderness in the adnexae bilaterally, and the pouch of Douglas was full and tender. There was foul smelling discharge on the examining finger.

Diagnosis

A clinical diagnosis of pelvic abscess was made.

Investigations

Hb – 12.6 g/dl WBC 7.4×10^9 N – 74% L – 26%.

Urea and electrolytes – normal.

An urgent pelvic ultrasound was done and showed features of pelvic abscess.

Management

She was admitted and started on intravenous flagyl and zinacef. The patient was explained to her condition. An informed written consent was obtained. She was then prepared for laparotomy.

Intra-operatively about 2 litres of foul smelling pus was drained from the pelvic cavity. Pus was taken for microscopy, culture and sensitivity. The uterus appeared

normal grossly and the tubes were inflamed with right-sided hydrosalphinx. Multiple adhesions were found and those that could be released were released. A peritoneal wash out using rifocin and warm saline was done. A corrugated drain was left intra-abdominally and was externalised through the right iliac fossa. GA was reversed successfully.

Post operatively she continued with intravenous antibiotics for 48 hours. The drain was removed after 24 hours since it had stopped draining.

The specimen taken grew staphylococcus aureus sensitive to zinacef and augmentin. She was on zinacef and flagyl. The patient did well and was discharged home on the 7th postoperative day after removal of stitches to be reviewed in the GOPC after two weeks.

Follow up

At the time of review, she was in good general condition. The wound had healed well and she had no complains.

Discussion

The patient presented had a pelvic abscess and laparotomy was done.

Pelvic abscess is a condition in which pus collects in the pelvic cavity usually in the cul-de-sac. It may follow acute or chronic pelvic infection. The patient presented, had been treated for vaginal discharge twice prior to this admission. It is possible she had a chronic pelvic infection. In some cases it is a complication of post abortal or puerperal sepsis. Chebrot reported of pelvic abscess treated in KNH 13% followed induced abortion. Most patients in his series were reported to be of low parity, unmarried and of low social economic and separated from the husband.[1] The patient presented was of low parity, separated from her husband and of low social economic status.

The condition may present with features of acute or chronic pelvic infection such as low abdominal pain and per vagina discharge associated with a fluctuant mass in the cul-de-sac. The severity of symptoms often is directly proportional to the size of the abscess. However in some patients even huge abscesses may be asymptomatic. Patients treated with antibiotics for pelvic infection who continue to have a fever with abdominal tenderness should be evaluated for pelvic abscess. The ultrasound is a good tool to demonstrate pus collection in the cul-de-sac. Paracentesis or culdocentesis may be positive with purulent pus.[2] For our patient an ultra sound done confirmed the diagnosis and a paracentesis was not done.

Various techniques of draining a pelvic abscess have been described. In our set up laparotomy is favoured. A sub umbilical midline incision is used. This facilitates good exposure for exploration to ensure that all pockets of pus are broken without perforating the gut. Pelvic adhesions should be released. A sample of the pus should be taken for culture and antimicrobial sensitivity studies. If the abscess is on the midline, adherent to cul-de-sac and fluctuant posterior colpotomy may be done for drainage. Percutaneous drainage under ultrasonographic guidance has been reported.[3] Broad-spectrum antibiotics covering for aerobic and anaerobic cover are used. This is because commonly the pelvic abscess is associated with organisms other than the gonococcus and chylamydia especially bactroides.[4] Change of antibiotics may be necessary depending on the culture results. J A had a laparotomy done via the sub

umbilical midline incision and all the pockets of pus were broken. Culture and sensitivity of the specimen taken grew staphylococcus sensitive to zinacef and augmentin. The patient was already on zinacef.

Thorough irrigation of the peritoneal cavity should be done with warm saline and a drain left through a stab wound. For patients treated early with a well-localised abscess, the prognosis is good. Long-term complications include tubal infertility, ectopic pregnancy and chronic pelvic pain. J A had thorough peritoneal irrigation. She healed well.

References

1. Chebrot S C, Pelvic abscess in female genital tract.
Mmed thesis UON 1985

2. Rock A J, Thompson J D; Pelvic Inflammatory Disease In Te Linde's Operative Gynaecology. Lippicott Williams & Wilkins Publishers Philadelphia 8th Edition 30; 664-668

3. Olak J, Christon N V,Stein L A, et al. Operative vs percutaneous drainage of intra-abdominal abscess. Arch Surg 1986; 121: 141.

4. Susan M. R, George D W, Hemsell D. L; Sexually Transmitted Diseases & Pelvic Infections In Current Obstetric & Gynaecologic & Treatment, Appleton & Lange 8th edition, Norwalk 38: 773

Gynaecology Case 15

VULVAL WARTS – CAUTERIZATION DONE

Name	M. M.	I. P. No.	0163521
Age	18	DOA	23.1.05
Parity	0 + 0	DOD	24.1.05

Presenting complaints

The patient presented with complains of vulval growth for 6 months.

History of presenting illness

The patient had been fairly well till 6 months prior to admission when she noted some swellings involving the vulva. Initially there was no associated pain or discomfort and she did not seek medical attention. The growths increased in size and number to involve most of the perineum. The swellings were also associated with itchiness and over time she noted foul smelling whitish discharge. She then decided to seek medical attention. She had normal bowel habits but had dysuria.

Obstetric and gynaecology history

She was para 0 + 0. She attained menarche at 14 years. Her menstrual cycle was regular with a flow of 4 – 5 days in a 28-day cycle. She had a sexual partner and occasionally used condoms for contraception.

Past medical history

She was known to have HIV infection and was on treatment with combivir and indinavir. She was also on septrin for pneumocystis carini pneumonia.

Family and social history

She was a house girl working for a family living in Umoja. She was single but had a boyfriend. She did not smoke cigarettes or drink alcohol. There was no history of any chronic illness in the family.

Physical examination

She was in fair general condition. She was not pale, jaundiced or cyanosed. She had generalized lymphadenopathy and generalized macular skin lesions.

The cardiovascular, central nervous and respiratory systems were within normal. The abdominal examination did not reveal any abnormality.

Pelvic examination

She had extensive exophytic growths covering the labia minora and majora and extending to the peri-anal region. The growths had whitish foul smelling discharge. Speculum examination or digital examination was not possible due to pain.

Diagnosis

A diagnosis of infected genital warts in an immuno-suppressed patient was made.

Management

The condition was explained to the patient and the need for cauterisation. An informed written consent was obtained. She was then started on oral metronidazole and oral augmentin. She was to be cleaning her perineum with a solution of 10% povidone – iodine, twice daily. This was to deal with the infection prior to the surgery.

Investigations

Sodium – 140-mmol/l	potassium -3.8 mmol/l	urea -3.2 µmol/l
creatinire -65mmol/l	haemoglobin -12.0g/dl	

She was then booked for theatre. On the morning of the operation she reported to the ward early in the morning. She had not taken breakfast and had fasted from midnight. She received atropine 0.6mg intramuscularly half an hour before being taken to theatre.

In theatre she was given general anaesthesia. In lithotomy position, the perineum was cleaned with savlon solution and 1% iodine solution applied, and draping done. Examination revealed warty growths involving the labia minora and majora bilaterally, the clitoris, the fourchette and the peri-anal area. The growths were clumped at the base using artery forceps. Excision each at a time was done using diathermy. This was done for the growths that were larger than 1cm.

The vaginal wall and the cervix were noted to have small warts. There was some foul smelling discharge in the vagina. Cleaning of the vagina was done using iodine solution. Haemostasis was achieved and general anaesthesia successfully reversed.

In the ward she continued with the oral medication that had been started a few days before the surgery. She was also given mefenamic acid for analgesia. The following day she was discharged home and was advised to have sitz baths. She was to come back for review after2 weeks.

Follow up

She came back two weeks later to the gynaecological clinic for follow up. She was in good condition. The perineum had healed well. There were still some small warty growths in her perineum, but the perineum was clean and not infected. She was advised to continue with follow up, since repeat cauterisation would be necessary in the future.

Discussion

M M presented with genital warts for which cauterisation was successfully done.

Condylomata accuminata commonly referred to as genital warts, is one morphologic manifestation of the human Papillomavirus (HPV) in the lower genital tract. HPV genome types 6, 11, 16, 18 and 31 account for most genital tract infections [1]. The HPV – 6 and HPV – 11 are associated with most exophytic condylomas, flat cervical condylomas and the low-grade cervical dysplasia.

The virus is sexually transmitted and is the most prevalent sexually transmitted infection [2]. The lesions have an incubation period ranging from several weeks to 8 months but clinical infection is usually apparent in 3 months. Initially the warts may be reddish brown but turn grey or white with exposure to local trauma.

The lesions are usually small and multifocal. Majority are diploid with 10% showing nuclear atypia of various degrees and require differentiation from Vulva Intra Epithelial Neoplasia [3]. Colposcopic examination is essential to determine the extent of the disease. The lesions lead to pruritic discomfort or irritation. In pregnancy they can be a source of massive bleeding due to the increased vascular supply if lacerations occur. Growth potential of warts is enhanced by immuno-suppression and to a less degree, pregnancy. Warts tend to develop in the moist regions of the vulva and the areas affected more by coitus, this includes the posterior fourchette ad lateral areas of the vulva. However they can be found throughout the vulva, on the vagina, on the cervix and in the peri-anal area [4]. In the patient discussed was immuno-suppressed and the warts covered most of the labia majora and minora and part of the peri-anal region.

Several treatment modalities have been used, their limitations being their inability to eliminate the HPV. The choice of treatment should be individualized depending on the volume and location of the disease, the presence or absence of associated dysplasia and prior treatment.

Local application of 25% podophyllin is commonly done. After sustained contact burning discomfort occurs. It is advisable to leave the agent in place for 6 hours before taking a bath. [1] Podophyllin tends to be more effective on the exophytic rather than the flat warts. Use is restricted to the vulva of non-pregnant women.

Halogenated acetic acid, dichloroacetic or trichloroacetic acid (TCA) can also be used. It has advantage over the podophyllin in that it sustains prompt chemical effect, can be used intravaginally and can be used in pregnancy.

Surgical excision, electro surgery, cryosurgery and laser vaporization can be done. Surgery is done for the extensive lesions that are difficult to treat with chemicals, failure of treatment with topical applications, multicentric HPV infections especially when involving the vagina, urethra or anus, significant association with intraepithelial neoplasia and for some immuno-compromised patients. M M had extensive lesions, electro surgery was chosen for her. Warts that are refractory to treatment should be biopsed for evaluation. 5% fluorouracil cream is effective in eradicating the early lesions [5]. It has specific antiviral therapy. Alpha interferon has been used intralesionally for refractory warts.

Before treatment the entire area should be inspected with the colposcope. Other causes of vaginitis should be sought and treated. The area should be kept clean and dry. The virus is present in the normal looking cells making recurrence after treatment common. Where destructive therapy such as the laser vaporisation is done, considerable local care must be given till healing occurs. Sitz baths followed by application of silver sulfadiazine xylocane offers relief and protects against secondary infection by bacteria. M M was explained to the need for follow up in order to deal with any complications or recurrence.

In pregnancy electro coagulation, cryotherapy or laser therapy should be used before 32 weeks gestation to avoid post treatment necrosis which lasts for up to 4 – 6 weeks. Florid lesions may necessitate caesarean section to avoid excessive bleeding and soft tissue dystocia. Laryngeal papillomas and vulval condylomas may follow vaginal birth. Acetic acid treatment in the last 4 weeks may alleviate the need for caesarean section.[5]

References

1. Rock J A, Thompson J D. Surgical conditions of the vulva, Te Lindes Operative Gynaecology 8th edition LW & W Philadelphia 1997 35; 895 – 897
2. Gravet M G, Sampson J E; Other infections conditions; High risk pregnancy; management options 1st edition W B Saunder 1994; 521 – 222
3. Alan M, Dina R; Benign disease of the vulva and the vagina. Gynaecological by Robert Shaw 3rd edition Churchill Livingstone 2003; 40: 607
4. David E S, Genitourinary infections and sexually transmitted diseased Genital Warts Novaks Gynaecology 12th edition 1996; 441
5. Curry L. S Barclay D L; Benign disorders of the vulva and vagina. Current obstetric and gynaecologic diagnosis and treatment. 8th edition Appleton & Lange Norwalk 1994, 34; 97 - 698

GYNAECOLOGY LONG COMMENTARY:

THE PREVALENCE OF DOMESTIC VIOLENCE AMONG FEMALE PATIENTS ATTENDING GYNAECOLOGY CLINIC AT KENYATTA NATIONAL HOSPITAL.

Abstract

Objective of the study: To describe the prevalence and nature of domestic violence among female patients attending gynaecology clinic at Kenyatta National Hospital.

Study Design: This was a Descriptive Cross sectional survey carried out in the Gynaecology clinic in the Kenyatta National Hospital in the months of June, July and August 2004.

Methodology: A total of 198 clients who met the criteria were recruited into the study. The clients were then interviewed in a private room. Their answers were filled into a partially structured questionnaire. The results were entered into a computer and then analysed using the SPSS program.

Results: Of 198 women interviewed 82 (41.4%) had experienced at least one form of domestic violence. Fifty-four (27%) of all women interviewed had experienced physical violence. The most common form of violence in this study was physical. More than half of those who had experienced violence (57%) had ever reported the violence to some authority.

Majority of the women were married with 84 (42.4%) being in customary marriages and 49 (25%) in statutory marriages. Twenty-three (12.5%) were cohabiting. Those cohabiting had the highest rate of violence with 43.5% of them having experienced violence compared to only 6% of those in statutory marriages. Twenty-three (12%)of the women were divorced with 74% of these citing violence as the reason for the divorce. The modal age group was 30-39 but the majority of those who reported violence were in the 50-54 age group. Most women (65%) experiencing violence were unemployed or poorly paid compared to 35% with better income, but this association was not statistically significant (P=. 24). Ten percent had no formal education, 46% had only a primary level education, 30% had secondary level education and 26% had been to college. Violence was not associated with the level of education (P=0.59). Interestingly women with higher education were more likely to report sexual violence (P= 0.003).

The most common diagnosis was uterine fibroids followed by secondary infertility. Physical violence was more prevalent in the secondary infertility group.

Conclusion: The prevalence of domestic violence is high but many victims are reluctant to disclose. There is need to establish structures in the society that are sympathetic to domestic violence victims to facilitate more disclosure.

Introduction & Background

Violence appears to be on the increase not only in Africa but worldwide[1]. Reasons given for this includes delinquency, lack of adequate social amenities in urban areas, poverty and unemployment [1]Theories of aggregation and violence stress the interaction of environmental factors and the individual's intrinsic potential for aggressive behaviour. Theories on the roots of violence and aggression include.[2]

1) Biological Basis

This is usually an adaptive function in situations threatening to the individual; it is a short term coping mechanism that may lead to maladaptive behaviour in the individual. When one faces an obstacle that interrupts their desired goal, he feels the need to remove it by his own action, aiming at restoring control. However not all anger leads to attack other factors come into play.

2) Psychodynamic aspects:

Aggression is considered as primary when it occurs in association with frustration of basic needs and secondary when it is endured and becomes associated with achieving gratification.

3) Environmental Theories

The environment of growing up contributes. Factors such as social learning, frustration and other situations i.e. it is a socially learnt behaviour.

4) Social Conflict

Antagonism between persons is quite common in groups of people in competition for a common goal especially where one group can get and the other does not.

In all cultures, the perpetrators are most commonly men. Women are most often the victims of violence. Such violence when occurring within the home set up is referred to as domestic or intimate partner violence. Domestic violence is one form of gender-based violence.

Violence is an effective method for gaining and keeping control over another person and when the batterer does not suffer adverse consequences as a result of their behaviour, he is likely to continue.

Historically violence against women has not been treated as a "real" crime. This is

evident in the lack of severe consequences, such as incarceration or economic penalties, for men guilty of battering their partners[3]. The legal system in Kenya in theory does not discriminate against women. In reality however the written and unwritten laws in Kenya treat women as subordinate to men especially due to the influence of the customary laws. [4]

Barterers come from all groups and backgrounds, and from all personality profiles. However, some behavioural warning signs of a potential batterer include extreme jealousy, possessiveness, a hot temper, unpredictability, cruelty to animals and verbal abusiveness [3]

Definitions

To abuse, to be violent and to batter refer to very similar treatment and will be used interchangeably.

Strictly, violence is an act carried out with the intention or perceived intention of physically hurting another person, while battering and abuse include other aspects of ill-treatment.

Acts of domestic violence generally fall into one or more of the following categories:

Physical Battering –The abuser's physical attacks or aggressive behaviour can range from bruising to murder. It often begins with what is excused as trivial contact, which escalates into more frequent and serious attacks.

Sexual Abuse – Physical attack by the abuser is often accompanied by, or culminates in, sexual violence where the woman is forced to have sexual intercourse with her abuser or take part in unwanted sexual activity.

Psychological Battering - The abuser's psychological or mental violence can include constant verbal abuse, harassment, excessive possessiveness, isolating the woman from friends and family, deprivation of physical and economic resources, and destruction of personal property. Psychological abuse can also result from various forms of manipulation such as withholding (the silent treatment), countering (refuting or invalidating the spouse's statements or actions), discounting (putting down her emotions, possessions, experiences, hopes, and fears), sadistic and brutal humour, blocking (avoiding a meaningful exchange, diverting the conversation, changing the subject), blaming and accusing, judging and criticizing, undermining and sabotaging,

threatening, name calling, forgetting and denying, ordering around, denial, and abusive anger. [5]

Economic violence -Economic control and progressive social isolation is yet another form of violence. Economic violence against women is real in our society. Patriarchal structures relegate women to an inferior status in political, social, intellectual and economic spheres.[18,19] Many husbands block their wives from becoming financially independent. The continued dependency on the man forces the wife to cling on regardless of the treatment by the man.[24]

The Battery/ Domestic Violence Cycle

Battering escalates. It often begins with behaviours like threats, name calling, violence in her presence (such as punching a fist through a wall), and/or damage to objects or pets. It may escalate to restraining, pushing, slapping, and/or pinching. The battering may include punching, kicking, biting, sexual assault, tripping, throwing. Finally, it may become life-threatening with serious behaviours such as choking, breaking bones, or the use of weapons.[5]

According to Walker[6]abuse is inflicted in a repetitive cycle of three phases:-

- Tension building

In this phase tension builds up. Verbal harassment, criticism, psychological humiliation and minor battering characterize it. The woman notices the changes in the man and she exercises more caution.

- Battering Incidence

The tension building gives way to the actual battering. This involves major physical violence usually accompanied by verbal abuse. It is short lasting 12-24 hours. The man's anger is usually out of control.

- Honey moon

The batterer feels sorry and acts apologetically. He may even buy her gifts, apologize and talk to her well promising not to hurt her again. This enforces the woman's hopes of his change and she therefore stays. At this stage it is difficult for her to leave. Sometimes the loving phase is not there.

Eventually the remorse gives way to minor incidents of abuse and phase one begins all over again.

Prevalence of domestic Violence

Subordination of women as a gender is a worldwide phenomenon defying the confines of race, class, creed social economic groups or nationality [7] The fourth world conference on women held in Beijing, September 4-15 (Beijing Declaration and platform for action) clearly stated that there is no single state in the world where women are safe from violence or are treated as equals with men.

According to United States Natural crime survey, the average yearly rate of assault by spouse or ex spouse was 2.7per1000 women during the period from 1973-1981.[8] In a Yale dispensary room in USA 3.8% of the patients were admitted having been injured by their patners[9]A prevalence of 7-23% among women attending antenatal clinic or obstetric and gynaecology clinics in America has been reported. [10,11,12]Among women seeking termination of pregnancy in America, 40% had self reported history of abuse.[13] Many women who require medical attention for injuries sustained do not report the abuse to their service providers. [14,15]

In Kenya Thenya et al using client samples from four hospitals in Nairobi, found a prevalence of 24% while Mwaliko found a prevalence of 25% among women attending family planning clinic in Kenyatta National Hospital.[16,17] Coalition on violence against women-Kenya (COVAW) in their survey of reported cases in Nairobi, found an average of 93 cases reported in a month[18]. This figure is likely to be less than the actual considering many cases go unreported since the media tends to report severe or sensational cases.

In the hospitals, a social history of domestic violence is rarely sought let alone recognized among women seeking treatment in accidents and emergency room. Unexplained injuries and physical conditions such as depression or hysteria may be as a result of partner abuse.

Cultural / Social Determinants

Some women consider physical abuse as part of marriage life[14] Culturally wife beating is acceptable as a way of the man controlling his wife and ensuring she is disciplined. The paying of the bride price gives him the right to own her.[19] Favourable aspects of our culture, which enhance the well being of women, have been suppressed, while those aspects that diminish women continue to be practiced in various degrees by our societies[20]

Several factors have been associated with domestic violence. Young age, being single, being divorced, recurring medical assistance and a normal family income of less than

us dollars 10,000 to be associated with current domestic violence.[21] Women at greatest risk of domestic violence include those with male partners who abuse alcohol or drugs, are unemployed or intermittently employed, have less than a high school education and are former husbands or former boyfriends. [22] This suggests that risk factors for injury from domestic violence may differ greatly among women of higher social economic status. Protective factors include being older, having access to a confidante and social support from friends. [23]

Why Do Women Stay?

A woman's reasons for staying in abusive relationships tend to be complex. The society has made the women believe it is their responsibility to nurture relationships and hence a broken relationship means a failure on her part. Clergy and secular counsellors are often trained to see only the goal of "saving" the marriage at all costs, rather than the goal of stopping the violence. [24] Women give different reasons for not reporting, including threats by the spouse, fear of husband's imprisonment, shame and guilt or even love, desire to make the relationship work, hoping he will change. Many factors such as her fear, shame, lack of money, and no place to go causes her to stay on. In many cases it is dangerous for a woman to leave her abuser. She realistically fears that the batterer will become more violent if she attempts to leave. If the abuser has all of the economic and social status, leaving can cause additional problems for the woman. Leaving could mean living in fear and losing child custody, losing financial support, and experiencing harassment at work.[25]

Health Effects Of Violence

Domestic violence is a major public health problem and a significant cause of female ill health and mortality. The insidious effects of abuse extend to many facets of a woman's life. It has physical, psychological, and sexual consequences. Torture victims may feel helpless and powerless. This loss of control over one's life and body may be manifested physically in frigidity, attention deficits, and insomnia. This is often exacerbated by the reactions many torture victims encounter from the members of the society. Often these experiences result in psychosomatic illnesses. [26] Various studies have demonstrated an association of domestic violence with depression, anxiety, drugs and alcohol abuse in either or both partners, sexual dysfunction, functional gastrointestinal disorders, headaches, chronic pain, and multiple somatic symptoms [27,28,29] Some battered women develop the Battered woman syndrome.[30]

This describes a pattern of psychological and behavioural symptoms found in women living in battering relationships.

There is significant burden associated with violence against women; domestic violence is the leading cause of disability-adjusted life years (DALYs) loss among women. [31] Recognition of the existing link between violence and HIV infection is important. Women in fear of violence are unable to refuse sex or negotiate for safer sexual practices and this exposes them to higher risk of infection. Women who adopt prevention strategies such as condoms may become vulnerable to abuse.[32]

Gynaecology and Violence

Domestic violence is a problem among the gynaecology patients. Physical and sexual abuse are linked to some of the most serious reproductive health problems, which include teenage pregnancy, high-risk sexual behaviour, sexually transmitted diseases, and chronic pelvic pain[32] Survivors of domestic violence may present in a variety of clinical settings, may report chronic pelvic pain or sexual dysfunction.[33,34] In the face of a negative work up the symptoms may be linked to psychological factors or conflicts.[35]

Infertility is another common gynaecologic disorder that is associated with a lot of stress. Cycles of hope followed by crushing disappointment, along with the high costs and low success rates of medical therapies, can lead to depression, anxiety, and intense frustration for the couple. This kind of frustration can lead to misunderstanding between the couple and can lead to domestic violence. [36,37]

Justification/ Rationale

Domestic violence is common and it is a problem of public health/ reproductive health importance. Kenyatta National Hospital is a referral hospital with a wide clientele that represents women from all walks of life. An estimation of the prevalence of domestic violence in KNH would give some indication of the magnitude of the problem in current Kenya. In the gynaecology clinic even minor forms that otherwise go unreported are represented. This gives some information on the violence cases not captured in hospital (casualty and wards) or police records where the reported cases tend to be the more severe ones.

General Objective

To determine the prevalence of domestic violence among women attending Gynaecology clinic at the Kenyatta National Hospital.

Specific Objectives

- To document the prevalence of domestic violence among women attending Gynaecology clinic in the Kenyatta National Hospital.
- To document the social demographic characteristics of the women with history of having experienced domestic violence.
- To document the pattern of gynaecologic diagnosis among women with history of having experienced domestic violence.

Study Population/Subjects

The study subjects included the women attending gynaecology clinic at the Kenyatta National Hospital. This is the National referral centre as well as a large teaching hospital. It is located about 2.5 km from the Nairobi city centre. The women visiting the gynaecology clinic were either new patients or revisits. They were drawn from all over Nairobi and its environs. Nairobi being the capital city of Kenya has a population drawn from the different ethnic groups from around the whole country. Women from different background were therefore represented. The majority were however from the low to middle social economic class. Some patients were referrals from other health facilities.

The obstetrics and gynaecology department caters for both out patients and in patients. Consultants, senior registrars, senior house officers and interns man it. They fall into 3 units. Each unit runs a gynaecology clinic on a designated day in the afternoon.

Study Design/Methodology

This was a Descriptive Cross sectional survey l.

Inclusion Criteria

Women over 18 years of age, attending the gynaecology clinic and were willing to participate in the study.

Exclusion Criteria

Women who declined to participate.

Procedures

The principle investigator carried out the study with assistance of one nurse who works in the clinic.

As the women came into the clinic, the assisting nurse talked to every third woman to try and eliminate any bias. This was done all the clinic days in the week. The nurse explained to them about the purpose of the study and the procedures. They were then requested to participate. All those who agreed gave written consent and they were directed to the interview room.

The principle investigator carried out the interviews in a private room within the clinic area. These interviews were carried out during the waiting period to avoid inconveniencing the patients. A pre-tested questionnaire was used for the interview. The number of women recruited was 198.

Study Period

The study took 3 months, in the months of June, July and August 2004.

Statistical Analysis

Completed questionnaires were numerically coded. To ensure confidentiality the principle researcher stored the data obtained locked up in a drawer. It was then entered into a computer using the EPI info and analysed using the SPSS program by the researcher with assistance from a data analyst. Data presentation was done in descriptive, tabular and graphic form. Appropriate tests of significance were done. The results were presented in tables and texts and tested for different correlations.

Sampling and Sample Size Calculations

The estimated local prevalence of domestic violence was taken as 24% as documented by Thenya in his study using client sample from four hospitals in Nairobi. The sample size was be calculated using the formula

$$N = \frac{Z^2 X P (1-P)}{c^2} \qquad \frac{N=1.645^2 \ X \ 0.24 \ X \ 0.76}{0.05^2} = 198$$

N- Minimum sample size required

P- Estimated Prevalence of domestic violence in Kenya = 24%

Z-The table value for standard normal deviation that corresponds to the desired level of error.

C- confidence index, 95%

Ethical Considerations

Permission to carry out the study was obtained from the Kenyatta National Hospital ethical committee.

Confidentiality was maintained. The partners were not present in the room during the interview in order to ensure a conducive environment. The interviewer (the principle investigator) maintained a non-judgmental altitude. If someone else interrupted the interview the interviewer stopped the interview. Any women found to be in need of urgent assistance were referred to the hospital counsellor.

Two participants were referred for further help to the coalition of violence against women (COVAW), an organization that offers counselling and legal services to survivors of domestic violence. The COVAW offices are located near the hospital, along Ngong Road.

Study Limitations

The study was exclusive of all women who are not followed up in Kenyatta National Hospital Gynaecology clinic and some women declined to participate. These may have different characteristics not captured in the study.

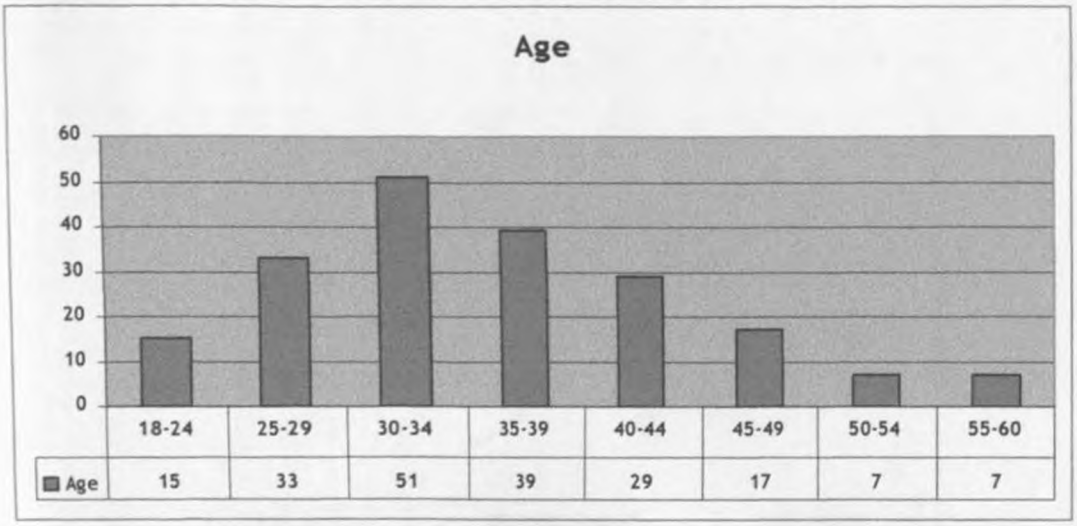
The study relied on the participant's memory, which might not have been not be accurate.

Results

The Social Demographic Characteristics of the participants.

The total number of women recruited was 198. Most of the women in the study were in the age group 30-39 with a range of 18-60 years.

Chart 1: Graph showing the age distribution of the sample group.



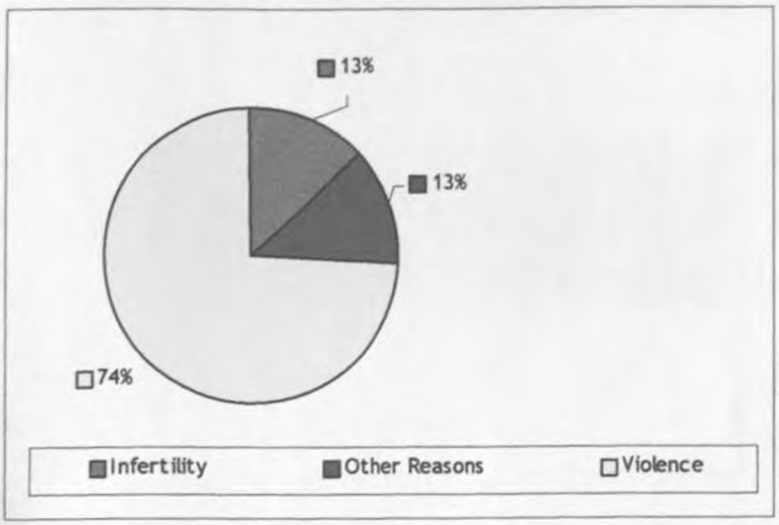
Most of the women were married with 84 (42.4) being in customary marriages and 49 (25%) in statutory marriages.

Table 1: Marital status

Marital status	Total no of women	Percentage
Cohabiting	23	12
Divorced	23	12
Customary	84	43
Statutory	49	25
Single	14	6
Widow	5	2
Total	198	100

Of the 23(12%) of the participants were divorced, 74% of these sited violence as the reason for the divorce.

Chart 2: Reasons for divorce



Forty one per cent of the women did not have an independent source of income, 21.7% were low-income earners and 25% were middle-income earners.

Table 2: Various education levels

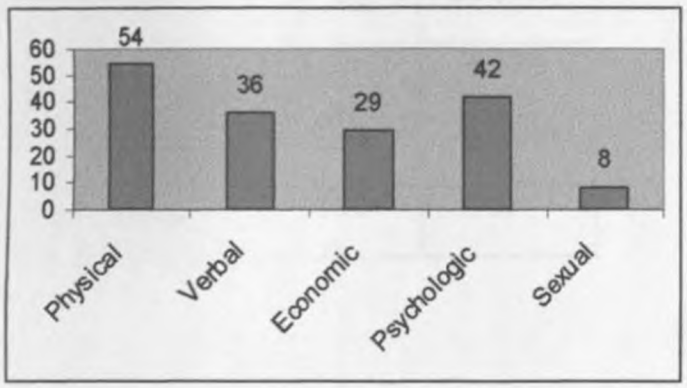
Level of education	No of women	The percentage
None	20	10
Primary	92	47
Secondary	60	30
College	26	13
Total	198	

Of the women interviewed 10% had no formal education, 46% had only a primary level education, 30% had secondary level education and 13% had been to college.

History of violence

Of the 198 women recruited was 82 (41.4%) of these had experienced at least one form of abuse. The most common form of violence was physical with 54 (27% of the participants) having experienced it.

Chart 3: graph showing distribution of various forms of violence



Of the 82 women who had experienced violence 35 (42.6%) had ever reported the violence to some authority or family members while the majority, 47(57.3%), had not.

Table 3: Women who had reported acts of violence.

Where reported	No	%
Family members	30	85
Chief	3	9
Human Rights body	2	6
Total	35	100

Only 8 (5%) of the women gave history of having experienced sexual violence. Sixty five (33%) of the participants felt it was not wrong for a spouse to force the wife to have sex with them. Hundred and thirty-three women considered forced sex by spouse to be criminal or wrong.

Table 4: The women's opinion on forced sex by spouse

Opinion on forced sex		
Criminal	57	28.79%
Wrong (not criminal)	76	38.38%
Not Wrong	65	32.83%
Total	198	100

Forty percent of those who considered forced sex by spouse to be criminal or wrong expressed willingness to report if it happened to them. Of these 75% of them had some idea on whom to report to but 25% of them said they did not know where to report.

Table 5: Where the women would report forced sex

53 out 133		Where would report
10	18.9%	Police
10	18.87%	Chief
12	22.64%	Family members
4	7.55%	Health worker
4	7.55%	Rights body
13	24.53%	Do not know
53	100%	

Of those who considered forced sex by spouse to be criminal or wrong 67(84%) indicated they would not report because of embarrassment. Several other reasons were given for not reporting.

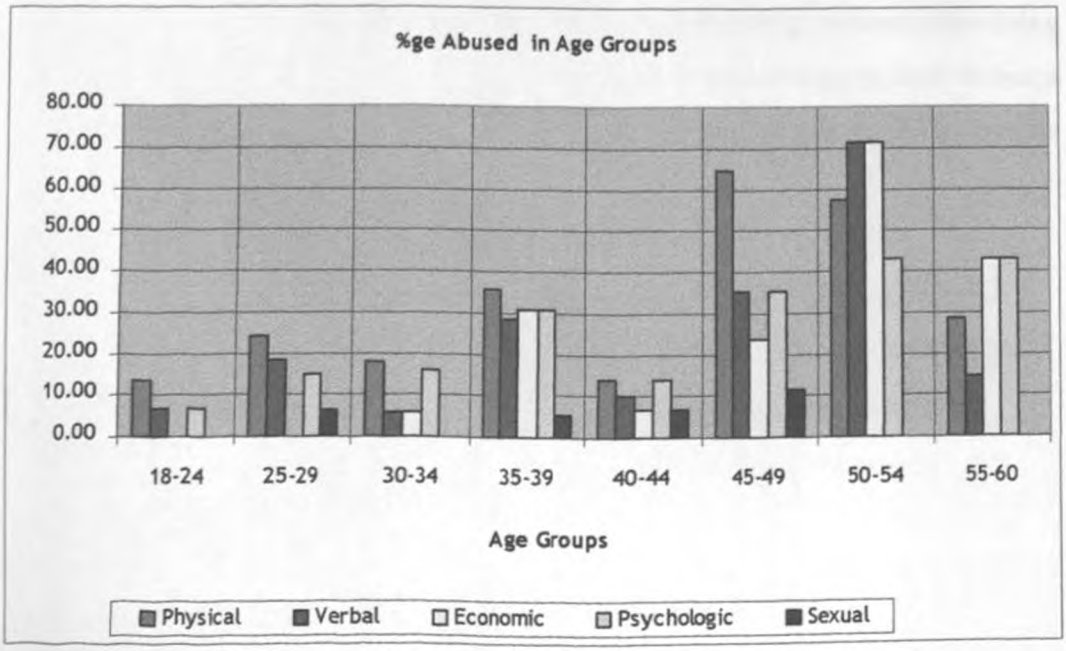
Table 6: Reason for not reporting forced sex

Reasons	80 out 133	
No reason	3	3.75%
Fear	10	12.5%
Embarrassment	67	83.75%
Total	80	100%

The Social Demographic Characteristics and violence of those who experienced violence

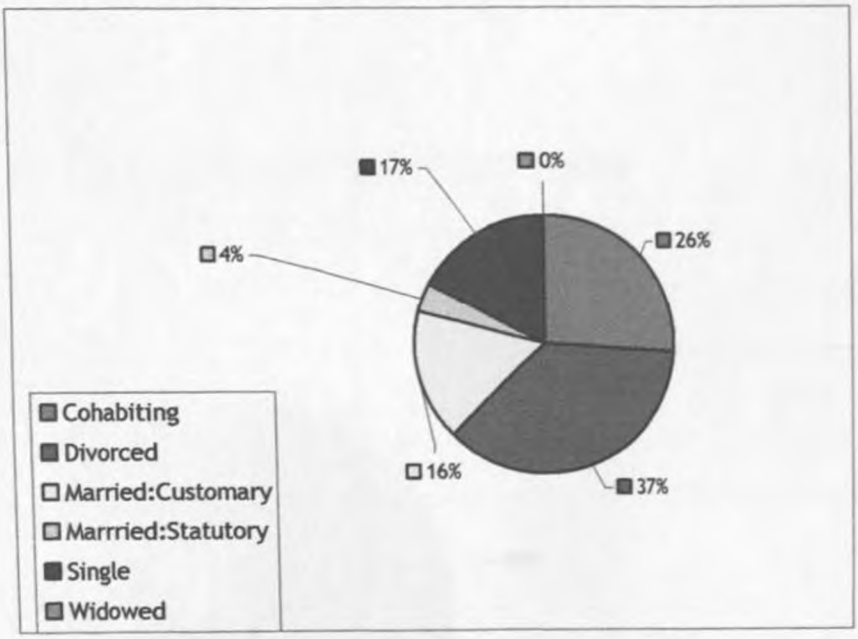
Although most of the women in the study were in the age group 30-39 the majority who reported violence were in the 50-54 age group.

Chart 4: Graph showing the % of those abused in various age groups



Most of the women were married with 84 (42%) being in customary marriages and 49 (25%) in statutory marriages. Those in statutory marriages reported the least violence, while those cohabiting reported the highest rate of violence. This difference was significant. (P<0.0001)

Chart 4: percentage of violence in the different marital status



The majority of those experiencing violence started experiencing violence after being married for 1-5 years. Twenty three per cent of those experiencing physical violence had some violence within the month preceding the interview and 5.5% within the preceding week.

Only 11 women in the study population reported drinking alcohol occasionally. Of these 6 (55%) reported violence. Of the 187 who did not drink alcohol 48(26%) had experienced violence by the partner. Alcohol consumption was associated with violence. (P=0.037). No woman reported usage of drugs of addiction

Majority of women (65%) experiencing violence were unemployed or poorly paid compared to 35% with better income. However, this difference was not statistically significant (P=0.24).

Chart 6: Percentage of violence in different income levels

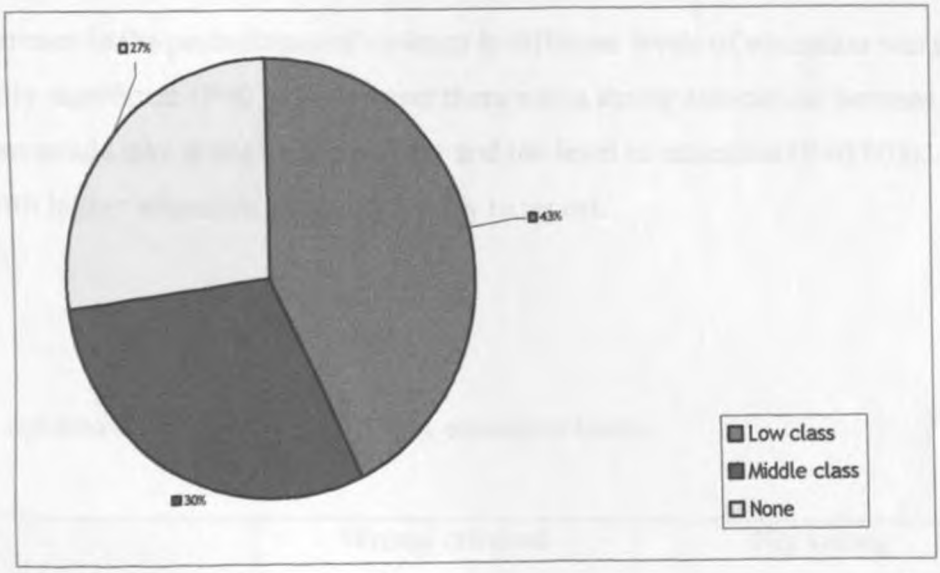


Table 6: Percentage of women who would report violence in the different education levels.

Level of education	Women experiencing violence		Women that would report	
	n	%	n	%
None	6	25	2	30
Primary	27	30	7	27
Secondary	12	22	9	75
College	9	35	5	63
Total	54	27	23	42

The difference in the percentages of violence in different levels of education was not statistically significant (P=0.59). However there was a strong association between the action one would take if she had forced sex and the level of education (P=0.003). Those with higher education were more likely to report.

Table 7: opinion on forced sex in different education levels

Level of education	Wrong/ criminal		Not wrong	
	n	%	n	
None	5	25%	15	75%
Primary	54	59%	38	41%
Secondary	40	68%	20	32%
College	23	92%	2	8%

There was also a relationship/association between the opinion one had concerning forced sex and the level of education. With higher level of education the women felt forced sex was wrong or criminal. (P=. 011)

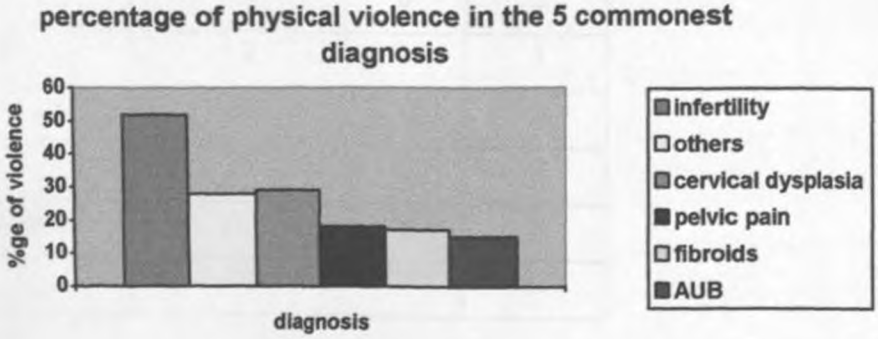
Medical Condition For Gynaecological Care

The most common diagnosis among those interviewed was uterine fibroids (35) followed by infertility (23). Physical violence was most prevalent in the infertility group. Fifty five percent of the women did not know their HIV status. Of those who knew 2 of them reported they were positive while 87 (44%) reported their status as negative.

Table 8: The 5 most common diagnoses and the percentage of violence in each group

Diagnosis	No of women	Physical Violence		% Of Physical Violence	
		Yes	No	Yes	No
Fibroids	35	6	29	17	83
Infertility	23	12	11	52	48
Pelvic pain	17	3	14	18	82
Cervical dysplasia	14	4	10	29	71
A U B	13	2	11	15	85
Others*	96	27	69	28	78
Total	198	54		55	45

* The other less common diagnosis and the associated level of violence are shown in one of the tables below.



The level of violence in the infertility group was highest compared to the level of violence in the AUB, cervical dysplasia and fibroids. However even though the level of violence in the infertility group was higher the difference was not statistically significant. (P=0.109)

The distribution of physical violence in the other less common Diagnoses

Diagnosis	No of women	Physical Violence	
		Yes	No
Ectopic pregnancy	11	2	9
Ovarian mass	9	3	6
Choriocarcinoma	8	2	6
Recurrent miscarriage	8	2	4
Ovarian cyst	7	2	5
Ovarian cancer	7	3	4
Endometrial hyperplasia	6	2	4
P I D	6	3	3
Pelvic abscess	5	2	3
Primary infertility	5	1	4
P O O C	4	2	2
V V F	4	1	3
Menorrhagia	3	1	2
Molar pregnancy	3	0	3
Warts	2	0	2
Uterine prolapse	2	1	1
Endometriosis	2	0	2
Lost IUCD	2	0	2
Hyperprolactinaemia	2	0	2
Total	96	28	68

Discussion

The prevalence of domestic violence (physical, psychological, verbal, economic and sexual) was 41.4%. This is collaborates the Kenya Demographic Health Survey (KDHS) findings that 44% of women aged 19-49 years had experienced physical or sexual abuse by their partners.[38] The physical violence prevalence was 27%. This is close to the prevalence reported both in Kenya and else where. A prevalence of 7-23% was reported among women attending antenatal clinic or obstetric and gynaecology clinics in America.[10,11,12] Thenya found a prevalence of 24% using client samples from four hospitals in Nairobi while Mwaliko found a prevalence of 25% among women attending family planning clinic in Kenyatta National Hospital. [16,17] However it is higher than the 9% reported among women attending ante-natal clinic in Kenyatta National Hospital.[39] It may point to the fact that though women still suffer violence in pregnancy some men may avoid physically assaulting their pregnant wives for fear of interfering with the pregnancy.

The most prevalent form of violence in this study was physical violence accounting for 65% of all violence. These findings are similar to those by COVAW in which physical violence was 68% (54 out of 82). This is possibly because it was the easiest to define and admit.[18] Of the 54 women who had experienced physical violence, 24 (44%) of them had reported at least once. Of those who reported 20 (83.4%) of them reported to family members, 2 (8.3%) to the chiefs and 2(8.3%) to human rights organizations. No woman had ever reported to the police or to medical personnel.

It appears that most cases of domestic violence are reported to family members. Many family members however feel their greatest responsibility is to save the marriage at whatever cost rather than addressing the violence.[24] Other studies have shown that when cases of domestic violence have been reported to the police, women have not received much help. Many cases remain pending in court for along time, while others are not followed up as police do not make the necessary arrests.[18] This altitude might discourage women from reporting and it is therefore not surprising that among these women none had reported to the police.

More than half (56%) of the women experiencing violence did not report the violence to anyone. This co relates closely to other studies that point to the fact that many women are reluctant to report domestic violence. In the Kajiado/Nairobi study, only 23% of domestic violence victims had reported looking for assistance.[40] Different reasons were cited for not reporting including fear of the outcome, some felt the

beating was minor while others were threatened by their husbands and hence feared retribution. Women who do nothing because the violence is minor should be encouraged to note that battering escalates. It often begins with behaviours like threats and name-calling. It may escalate to restraining, pushing, slapping, and pinching. It may then include punching, kicking, biting, sexual assault, and throwing. Finally, it may become life threatening with serious behaviours such as choking, breaking bones, or the use of weapons. It is therefore better for the women to take action while the violence is still minor to avoid a more severe result in case it escalates [5].

It is worth noting that none of the patients had reported the violence to the health workers. Many of these clients were regular clinic attendees who were in contact with the health workers yet they did not state this problem that could impact their reproductive health negatively. Healthcare providers need to have a very high index of suspicion for domestic violence. Lack of training to sensitise the medical staff is a common barrier in the screening for partner violence. The situation is compounded by lack of time, feeling of personal inadequacy and a tendency to deny the existence of domestic violence [41,42] According to COVAW many private health care facilities declined to treat women who had suffered assault due to the criminal nature of the cases. They avoided the possibilities of getting summons to the court to give evidence. They felt the cases took too long and took a lot of their time. This means the survivors of violence do not get enough support from the health care providers. [18]
Disclosure of domestic violence is facilitated by an understanding and supportive altitude. Women need to feel respected and not condemned or looked down upon; the health care providers and other authorities need to cultivate a relationship of trust with their client, which is enabling the women to open up. Inappropriate responses such as shock, horror or disbelief should be avoided. It may be necessary to involve professional counsellors who will provide the necessary information and support, to facilitate the women to make a balanced informed choice. Direct advice such as to leave or go to court may result to the woman feeling misunderstood in her situation, may feel worthless or even drive her away from seeking help. [35,43]

Many women (28.6%) felt that it is normal for a husband to beat their wife. As Gichangi observed some women consider wife beating as part of marriage.[14] Women

need to be educated on their rights. The United Nations commission on human rights resolution 2001/49 affirms that any act that results in, or is likely to result in, physical, sexual or psychological harm or suffering to women, including threats of such acts, coercion or arbitrary deprivation of liberty, whether occurring in public or in private life is violence. As such women should not tolerate violence of any nature as part of their responsibility in a marriage.[44]

Sexual violence was the least reported with only 5% of the women reporting it. This is most likely under-reporting since many women feel the husband has the right to have sex with them even if they are not willing. Thirty three per cent (33%) of the women interviewed considered it right for a husband to force his spouse to have sex. Only 28% of the women felt that being forced to have sex by husband is criminal. Of all the women interviewed only 26.8% would consider reporting if their spouses sexually assaulted them. Forty six percent of the women who considered forced sex by husband wrong would not report if this happened to them due to embarrassment. It is important to encourage the society to discuss issues of sex more freely. This will reduce the embarrassment experienced and encourage more women to seek help when necessary. It is notable from this study that many women would not know where to report. This is mainly because the women are not confident they will get the necessary help even if they reported.

Domestic violence continues to be a problem in this country. Twenty three percent of those experiencing physical violence had some violence within the month preceding the interview and 5.5% within the preceding week. In the KDHS 29% of the women in abusive relationships had experienced the violence in the year preceding the survey.[38]

Most of the women experiencing violence in this study were in the age group 50-54 years. This co relates with the Kenya Demographic Health Survey 2003 report that older women were more likely to report having ever been abused than the younger women. This may be due to be a factor of longevity of the relation. The longer the relationship the longer one has to show abusive tendencies. Another factor might be that the older women find it easier to disclose the violence.

This study shows that the literacy level of Kenyan women is still low. 20 (10%) of the women did not have any form of education while 92(46%) had only a primary school

education. Many parents favour the education of male children as compared to the female.[45]

The level of education was not associated with domestic violence in this survey unlike in the Kenya Demographic Health Survey 2003 where women with secondary education were less likely to be victims of violence than the less educated women. Up to 34.6% of women with college education were experiencing violence compared to 29.3% of those with only primary education and 25% with no education. The difference in the percentages of violence across the different levels of education was not statistically significant (P= 0.59). A similar situation is seen in Odula's study in which majority (55%) of those abused had more than a secondary education.[39] However there was a strong association (P=0.003) between the action one would take if she had forced sex and the level of education. Also there was an association (P=0.011) between the opinion one had concerning forced sex and the level of education. The more educated women felt forced sex by the spouse was wrong and they were more wiling to report.

Most women (64.8%) experiencing violence were unemployed or poorly paid compared to (35%) who with better income. However, this difference in the percentages of violence between those who do not earn any income, the low-income earners and the middle class income earners was not statistically significant. (P=0.24) The typical battered woman is thought to be a woman of low social economic status with low or no education level and a non-professional. This may not always be so, women of higher social economic class may opt to act all is well to avoid the embarrassment associated with domestic violence and maintain their respect. Violence against women is not just a disease of the poor.[46] However it is important to economically empower women so that in case of abusive relationships, financial dependence does not cause them to cling on to their partners.

Ninety percent of the women were married or living in an informal union with a man. Those in statutory marriages experienced the least violence while those cohabiting had the highest rate of violence with 43.5% of them having experienced violence compared to only 6% of those in statutory marriages This may suggest that those couples that have formally committed themselves to a marriage have better relationships than those who live together informally. Among the12% that had

divorced, 74% of these sited violence as the reason for the divorce. This again indicates that domestic violence is a big problem in marriages. More effort should be put in identifying possible ways of stopping the vice without necessarily ending the marriage. However the woman's life should not be endangered in an effort to preserve the marriage.

Only 11(6%) of the women reported drinking alcohol occasionally. Of these 55% reported violence compared to 26% who experienced violence among those who do not drink alcohol. Thus there was more violence among those who drunk alcohol compared to those who did not. (P=0.037). It has been shown that battered women tend to have a higher consumption of alcohol and cigarettes as compared to the non-abused.[35]

The most common diagnosis among those interviewed was uterine fibroids followed by secondary infertility. Physical violence was most prevalent in the secondary infertility group. In most African countries infertility is a stressful situation associated with social stigma and profound psychological sequel. [36,37] The female partner bears most of the blame though not necessarily responsible. With so much blame on the woman and the stress associated with infertility, one may suspect that the woman may be at risk for battering. However even though the level of violence in the infertility group was highest compared to the level of violence in the AUB, cervical dysplasia and fibroids. the difference was not statistically significant. (P=0.109).

Conclusions

1. The prevalence of domestic violence is still high and many victims are reluctant to disclose it; physical violence is more likely to be disclosed while sexual violence is the least. Older women were more likely to disclose abuse.
2. Domestic violence victims were more likely to report the violence to family members, none reported to the police or to the health workers.
3. As the level of education rises recognition of the fact that sexual violence by the partner is wrong increases among the women and their reluctance to report decreases.

Recommendations

1. Women and girls need to be educated more so that they are more knowledgeable about with domestic violence and how to deal with it.
2. Health workers need to be more intentional in looking out for domestic violence victims and offer the necessary support.
3. More support is needed so that the women can overcome the barriers that keep them from seeking help.

REFERENCES

1) Hazel O Ayanga: Violence against women in African oral literature as portrayed in proverbs. In Violence Against Women by Grace Wamue and Mary Getui Acton Publishers Nairobi pg 13, 1996

2) David Daniels, MD Marshal G. Lula MD Frank Ochberg MD
Violence and the struggle for existence. Work of committee on violence in the Department of psychiatry, Stanford University School of medicine. Published by Little Brown and company Pg 1-80 1990

3) The Problem; Why Do Men batter women? The American National Coalition Against Domestic violence. Oct 1999 www.ncadv.org

4) SB Gutto.The status of women in Kenya A study of paterlism, inequality and under- Privilege IDS Discussion paper, Univ of Nairobi, 1975

5) Cited in What is Abuse? By: Dr Sam Vaknin July 1, 2003
Suite101.com/article.cfm/18046/101757

6) Walker, L.The Battered Woman. New York: New York: Harper and Row, 1979.

7) Musimbi Kanyoro Feminist: Theology and African culture; Violence Against Women, Grace Wamue and Mary Getui, Acton Publishers Nairobi pg 4

8) J klaus PA, Rand MR
Family Violence, Bureau of justice statistics Special Report, Washington DC, 1984

9) Rousanville B. Weissman M.
Battered women: A medical problem requiring detection. Int J Psychiatry med 1977;8:191-210

10) MC Farlane J Battering during pregnancy: Tip of an iceberg revealed, women and health. 1989:15:69-84

11) Helton AS, Snodgrass SC
Battering during pregnancy and substance abuse. Intervention strategies Birth 1987:14:142-147

12) Amaro H, Fried L E Cabral H et al: Violence during pregnancy and drug abuse Am J public health 1990: 80: 575-579

13) Glander SS, Moore ML, Michielutte R et al. The prevalence women seeking abortion. *Ostet gyncol 1988;91: 1002-1006*

14) Gichangi P B Domestic Violence: Ruptured spleen, splenectomy, Mmed Thesis Abortion short case record. Mmed Thesis univ of Nairobi 1999

15) Stewart D E, Cecutti A: A Physical abuse in pregnancy Can. Med assoc J 1993; 149:1257-1263

16) Thenya SM, Gichangi PB, Kamau J: Domestic violence. A baseline study of Kenyan situation unpublished Report 2000

17) Mwaliko: Prevalence of domestic violence among attending family planning services at Kenyatta National Hospital and its association with contraceptive choice. Mmed Thesis obs gyn Feb 2002med.

18) In pursuit of justice A research report on service providers "Response to cases of violence Against Women in Nairobi Province; coalition on violence against women – Kenya October 2002.

19) Constance R.A Shisanya A theology reflection on economic violence against women In Violence Against Women by Grace Wamue and Mary Getui Acton Publishers pg 60-66 1996

20) Musimbi Kanyoro; Feminist Theology and African culture, In Violence Against Women by Grace Wamue and Mary Getui Acton Publishers Nairobi, pg 4 1996

21) Mc Cauley J, Kern D E, Kolodner J et al : The battering syndrome prevalence and clinical characteristics of domestic violence in primary care internal medicine practices *Ann Inter med. 1995;123;737-746*

22) Demetrios and colleagues: Risk factors for injury from Domestic Violence, *New Eng J Med 1999; 341; 1892-8*

23) Gielen A C, O Campo P J, Fadn RR et al: Interpersonal conflict and physical violence during child bearing year soc sci med 1994;133-135

24) The Problem; Barriers to leaving A Violent Relationship, The American National Coalition Against Domestic violence. www.ncadv.org

25) The Problem; Why do women stay? The American National Coalition Against Domestic violence. Oct 1999 www.ncadv.org

26) Dr Sam Vaknin, The Mind of the Abuser, The Psychology of Torture 1, HMTL Document 2003

27) Alpert E: Violence in intimate relationship and practicing internist; New Disease or New agenda *Ann intern med. 1995;123:774-781*

28) Mc Cauley J, Kern D E, Kolodner J et al: The battering syndrome prevalence and clinical characteristics of domestic violence in primary care internal medicine practices *Ann Inter med. 1995;123;737-746*

29) Sutherland C,Baybee D. Sullivan C: The long-term effects of battering on women s health, 1998:4:41-70

30) Walker, L., The Battered Woman Syndrome 1984 New York: Guillian Press, p. 95-97.

31 Lozano Ascencio R, The health impact of domestic violence: Mexico City. Too close to home: domestic violence in the Americas, edited by Andrew R. Morrison and Maria Loreto Biehl. Washington, D.C., Inter-American Development Bank, 1999. :81-101.

32) Watts C; Ndlovu M; Njovana E; Keogh E Women, violence and HIV / AIDS in Zimbabwe. Women's Edition. 1999 Apr;:28-9. : SAFAIDS NEWS. 1997 Jun;5(2):2-6.

33) Rapkin, A.J., Kames, L.D., Darke, L.L., Stampler, F.M., & Naliboff, B.D. (1990).History of physical and sexual abuse in women with chronic pelvic pain *American Journal of Obstetrics and Gynecology, 76,92-96*

34) Mark L. Elliott, PhD Chronic Pelvic Pain: What Are The Psychological Considerations? January/February 1996, APS Bulletin volume 6, number 1

35) Alpert E J Violence in intimate relationship and practicing internist; New Disease or New agenda *Ann intern med. 1995;123:774-781*

36) S.K. A SINEI: Infertility in developing countries – a health paradox. *Journal of Obst. Gyne East, Central Africa* 5, 1986 pg 3)

37) Ladipo OA (1978): Pseudocyesis in Infertile Study. Infertility 2 (1): 63

38) Kenya Demographic Health Survey 2003, Preliminary Report; CBS, MOH Kenya, KEMRI, CDC.

39) Odula C A: Prevalence of domestic violence among clients attending Antenatal clinic in Kenyatta National Hospital. M med Thesis U O N 2002

40) Dr Wangui Njau; Dr Enos Njeru: Women and violence in Nairobi and Kajiado Districts in Kenya Final Report Feb 1997

41 Chambliss L R, Bay R C Jones R F: Domestic violence an educational imperative? *Am J obst gynae* 1995*; 172:1035-1038*

42) Parsons L H Zaccaro D, Wells B et al: Methods and altitudes toward screening obstetrics and gynaecology patients for Domestic Violence Am *J obstet Gynae 1995; 173: 381-387*

43) Mezel G C, Bewlery S: Domestic violence and pregnancy. *Br J 0bstet gynae 1997;1*

44) World Health Organisation, Putting Women First; Ethical and safety, Recommendations for Research on Violence Against women, WHO/FCH/GWH/ 0.1

45) G S Eshiwani women access to higher education in Kenya. A study of Opportunities and attainment in science and mathematics in G.S Were, ed., Women and development in Africa -*Journal of Eastern Research and development, vol 13,* Nairobi,1985

46) Schmuel E:Schenker J G: Violence against women, the physians Role Eur. *J obst Gynae* Reproductive Biol 80(2): 239-45 1998 Oct

APPEDIX 1

CONSENT EXPLANATION FORM (DOMESTIC VIOLENCE)

Dr shiphrah Kuria

Po box 182 00202 KNH Nairobi

Tel 0722300279

Statement

I am requesting you to take part in a research study. The purpose of this form is to give you information that will help you decide on whether to take part or not. Please read it carefully. You are free to ask any questions for further clarification.

I am working in Kenyatta National Hospital pursuing a post graduate course in gynaecology in the University of Nairobi. As part of my studies, the programme requires me to do some research. I have chosen to study the prevalence of domestic violence among the patients being followed in the gynaecology clinic in KNH.

Domestic violence is believed to affect the health of the victims negatively. I hope to collect more information that will hopefully improve the level of care to those with this problem. Anyone found to have this problem will be advised accordingly and helped to get further help from the necessary quotas.

You will be asked a series of questions to which you will be expected to give answers. This will take about 30 minutes of your time. Any information you give will be treated with confidentiality.

Your involvement will be highly appreciated but is not a condition for you to receive treatment in this hospital.

You may contact my supervisors or me; Dr Bukusi or Dr Tekle (department of obstetrics and gynaecology), or the Ethics and Research Committee of Kenyatta National Hospital P.O. Box 20723, Nairobi. Tel 726300 – 9.

Consent

I,..... volunteer to participate in this study on Domestic violence It has been explained to me by---------- and I have had the opportunity to ask questions.

Patient's name..... Signature .....

Witness ..... Signature

Date:

CONSENT EXPLANATION (KISWAHILI)

Dr shiphrah Kuria

Po box 182 00202 KNH Nairobi

Tel 0722300279

Maelezo

Ninakuliza ushiriki katika utafiti . Isome kwa makini na ukiwa na swali uliize.

Mimi nini daktari Kenyatta National Hospital, na ninasomea juu ya matibabu ya magojwa ya wanawake. Katika masomo yangu ninahitajika kufanya utafiti kati ya wagonjwa tunao hudumia. Nitafanya huu uafiti chini ya madaktari Dr Bukusi na Dr Tekle wanaohudumu katika hospitali hii. Ningependa kufanya huu utafiti kati ya wagonjwa wanaokuja clinic ya gynaecology. Nimechagua kuangalia juu ya vita nyumbani, hasa mabibi wanao chapwa na mabwana wao.

Utaratibu

Utaulizwa maswali kadhaa utakayoijibu. Hii itachukua kama nusu saa hivi. Utahojiwa na mwanamke mkiwa peke yenu. Habari utakayoitoa itabaki ya siri.

Kuhusika kwako kutafurahiwa lakini si sharti ili upate matibabu. Swali lolote kuhusu utafiti unaweza kuniuliza ama kuuliza wahusika wengine: Dr Bukusi (department of obstetrics and gynaecology), Dr Tekle (department of obstetrics and gynaecology), au Kamiti inayozingatia mambo ya utafiti katika hospitali ya Kenyatta kwa S.L.P 20723, Nairobi. Simu 7236300 – 9.

Mimi---------- nimejitolea kuhusika katika utafiti huu unao angalia juu ya vita nyumbani. Nimeelezewa na---------- na nikapata nafasi kuuliza maswali.

Jiana---------- Sahihi----------

Shahindi ----------

Tarehe ----------

APPENDIX 2

THE QUESTIONNARE (DOMESTIC VIOLENCE)

1) Date of birth Study number
2) Diagnosis
3) Marital status. Single Married Cohabiting
   Divorced Engaged Widowed
4) Highest level of education completed?
   None Primary Secondary College
5) What is your occupation?
   Employed (specify) Housewife
   Business (specify) Unemployed
6) Do you drink alcohol? Yes No
7) If yes to above how much/often (specify)
8) Do you use drugs of addiction? Yes No
9) Do you have a regular sexual partner? Yes No
10) How long in years has the relationship lasted? 0- 1 1-5 6-10 >10
11) What kind of marriage are you in? (the married)
   Customary Statutory Others specify
12) Have you ever been married before? Yes No
13) If yes why did you leave? Violence by partner Other reasons
14) How many times have you ever been pregnant? Of these how many
   Miscarriages Normal deliveries still births
15) How many living children do you have now? None 1-3 > 3
16) What sex are they? Male female
17) How many children would you like/ liked to have?
   The no I have More Less

18) How many children would your husband like/liked you to have?

The no I have More Less

19) For how long in years have you been trying to get a baby? < 1 1-5 >5

20) Does your husband think you are the cause of the lack of children? Yes No

21) What is your opinion on a woman being forced to have sex by the partner?

Its wrong (criminal) Not wrong Wrong (but not criminal)

22) If it happened to you would you report him? Yes No

23) If yes, to who?

Police Chief Family members Others (specify)

24) If no, why would you not report?

25) Has your partner ever abused you in any of the following ways?

Abused	physically	verbally	economically	psychologically	sexually
Yes					
No					

25) If yes how long in years after you were married did he abuse you?

Abused	physically	Verbally	economically	psychologically	sexually
Before marriage					
< 1					
1-5					
> 5					

26) How long ago did he abuse you last?

Abused	physically	Verbally	economically	psychologically	sexually
1 week					
1 month					
1-5 years					
>5 years					

27) when he last abused you ,what did you do about it?

Nothing Reported Others (specify)

28) Do you think he abuses you because you do not have the number of children he would like? Yes No

29) Have you ever reported him? Yes No

30) To who?

police Chief Family members Others (specify)

31) If you did not report what prevented you?

a) He stopped me physically
b) He threatened me
c) I was embarrassed
d) It is normal for a husband to beat the wife
e) Others (specify)

32) Did your father ever beat your mother? Yes No

33) Were you ever abused as a child? Yes No

34) HIV status +ve -ve unknown

APPENDIX 3

THE QUESTIONNARE (QUALITY OF ANC)

1) Date Age Study number

2) Marital status. Single Married Cohabiting

Divorced Engaged Widowed

3) Education level, highest level completed?

None Primary Secondary College

4) Occupation? Employed Housewife Business(specify)

5) Residence? ----------

6) Religion? Christian Muslim Hindu Others(specify)

7) Ethnicity? African Asian Caucasian Others (specify)

8) Where did she attend antenatal clinic?

9) Parity. Primigravida Para 2-4 Para 5 and above

10) How long ago in years was her last delivery? < 1 1-2 ≥ 3

11) Gestation at booking 1st Trimester 2nd Trimester 3rd Trimester

12) Medical history

None significant chronic illness (specify) HIV status

Others (specify)

13) Was physical exam done? Yes No

14) Was pallor looked for? Pale Not pale

15) Of those who were found to have pallor what actions were taken?

None Haematinics given Admitted Transfused TDI

Haemoglobin level estimation done Stool exam for ova/cysts

16) Where haemoglobin level was done what was the level in g/dl?

<4 4-6.9 7-10.9 ≥11

17) What was done for those found to be anaemic?

Nothing Haematinics given Admitted Transfused TDI

18) Where stool test was done, what were the findings?

No Abnormality Detected Abnormality (specify)

19) Was any of these tests done?

Test	done	Results available	not available
Full blood count			
haematocric			
serum ferritin levels			
Peripheral film			
blood slide for malaria parasites			
sickling test			

20) How many visits did the mother make? <4 4-8 ≥9

21) Of those visits how many times was she examined for pallor?

None 1-3 4-8 >8

22) Was she on routine supplementation? Yes No

23) Was a repeat haemoglobin estimation done? Yes No

24) What was the level in g/dl?

<4 4-6.9 7-10.9 ≥11 Not Indicated (NI)

25) At what gestation was the repeat haemoglobin estimation done?

28+ - 36 36+ - 37+ 38+ NI

APPENDIX 4

CONSENT FORM

Dr shiphrah Kuria

Po box 182 00202 KNH Nairobi

Tel 0722300279

I am requesting you to take part in a research study. The purpose of this form is to give you information that will help you decide on whether to take part or not. Please read it carefully. You are free to ask any questions for further clarification.

I am working in Kenyatta National Hospital pursuing a post graduate course in obstetrics in the University of Nairobi. As part of my studies, I am carrying out a study on the prevalence of anaemia among women delivering at the Pumwani maternity hospital and the quality of care they receive.

You will be asked to avail your ante-natal card to the reseacher who will fill in your details into the questionnaire. Your name will not appear in the questionnaire and this information will not be used for any other purpose other than the research.

Your involvement will be highly appreciated but is not a condition for you to receive treatment in this hospital.

CONSENT EXPLANATION (KISWAHILI)

Dr shiphrah Kuria

P O box 182 00202 KNH Nairobi

Tel 0722300279

Ninakuliza ushiriki katika utafiti . Hii barua ni ya kukuelezea kuhuzu utafiti huu ili uweze kuamua kama utashiriki. Isome kwa makini na ukiwa na swali uliize.

Mimi nini daktari KNH, na ninasomea juu ya matibabu ya magojwa ya wanawake.

Katika masomo yangu ninahitajika kufanya utafiti kati ya wagonjwa tunao hudumia

Nimechagua kuangalia juu ya upungufu wa ndamu wakati wa mimba.

Utaulizwa utupatie kadi ya clinic ili tupate habari inayohitajika. Habari hii itabaki ya siri.

Mimi nimejitolea kuhusika katika utafiti huu. Nimeelezewa maana yake.

Jina---------- Sahihi----------

Tarehe ----------

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.

Tel: 726300-9
Fax: 725272
Telegrams: "MEDSUP", Nairobi.
Email: KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/01/2270

Date: 2 June 2004

Dr. Shiphrah Kuria
Dept. of Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr. Kuria

RESEARCH PROPOSAL "DESCRIBING THE PREVALENCE OF DOMESTIC VIOLENCE AMONG FEMALE PATIENTS ATTENDING GYNAECOLOGY CLINIC AT KENYATTA NATIONAL HOSPITAL" (P40/4/2004)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** your above cited research proposal for the period 2 June 2004 - 1 June 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, KNH-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: Dr. E Bukusi, Dept. of Obs/Gynae, UON
Dr. Tekle, Dept. of Obs/Gynae, KNH

PUMWANI MATERNITY HOSPITAL

Tel: 02/6763291– 4
Fax: 02/6762965

P.O. Box 42849
Code: 00100- GPO
Nairobi.

PUMWANI MATERNITY HOSPITAL RESEARCH AND ETHICAL REVIEW COMMITTEE

Dr S Kuria
UNIVERSITY OF NAIROBI
DEPT. OF Obs/Gyn

24TH AUGUST 2004

RE: APPROVAL OF RESEARCH

It is our pleasure to inform you that your proposal entitled "The Quality of Antenatal Care in respect to Assessment of Anemia at Pumwani Maternity Hospital Nairobi" has been reviewed and approved by the Pumwani Maternity Hospital Research and Ethics Committee.

The proposal has been reviewed on the research merit, ethical considerations, sampling, methodology and relevance to the care at our institution. The PMH –REC requires that you be supervised by a member of our management staff in the field to be studied. Your supervisor will be Dr. Musili. Please get in touch with him before you begin for him to orientate you.

The REC also requires that you **submit a copy of your final study/ thesis** on completion of research.

All the best as you carry out your research

SIGNED:-

MEDICAL SUPERINTENDENT ---------- DEPUTY MEDICAL OFFICER OF HEALTH PUMWANI MATERNITY HOSPITAL DATE 24/8/04

CHAIRMAN PMH RERC ---------- DATE 24/08/04

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.

Tel: 726300-9
Fax: 725272
Telegrams: "MEDSUP", Nairobi.
Email: **KNHplan@Ken.Healthnet.org**

Ref: KNH-ERC/01/2385 **Date: 11 August 2004**

Dr. Shiphrah Kuria
Dept. of Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr. Kuria

RESEARCH PROPOSAL "THE QUALITY OF ANTENATAL CARE WITH RESPECT TO ASSESSMENT OF ANAEMIA IN PREGNANT WOMEN AT PUMWANI MATERNITY HOSPITAL, NAIROBI" (P41/4/2004)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** the revised version of your above cited research proposal for the period 11 August 2004 – 10 August 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, KNH-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: Dr. E A Bukusi, Dept. of Obs/Gynae, UON
Dr. G Tekle, Dept. of Obs/Gynae, KNH