CASE RECORDS AND COMMENTARIES IN OBSTETRICS AND GYNAECOLOGY

SUBMITTED BY

DR. KIGEN BARMASAI BARTILOL

FOR THE EXAMINATION OF MASTER OF MEDICINE IN THE

DEPARTMENT OF

OBSTETRICS AND GYNAECOLOGY OF THE

UNIVERSITY OF NAIROBI

NOVEMBER 2002

TABLE OF CONTENTS

PAGE

TITLE .....

DEDICATION iii

ACKNOWLEDGEMENT iv

DECLARATION v

CERTIFICATION OF SUPERVISION

CERTIFICATION vi

CERTIFICATION vii

CERTIFICATION viii

CERTIFICATION ix

INTRODUCTION 1

OBSTETRICS SHORT CASES

Obstetric case 1: Obstructed labour – Emergency caeserian section 20

Obstetric case 2: Preterm – premature rupture of membranes Emergency caeserians section 27

Obstetric case 3: Placenta previa type III posterior I Emergency caeserian section 35

Obstetric case 4: Fulminant genital warts in pregnancy Elective caeserian section-life baby 42

Obstetric case 5: Cardiac disease in pregnancy 49

Obstetric case 6: Rhesus negative – live birth 59

Obstetric case 7: Urinary tract infection in pregnancy Successful delivery 67

Obstetric case 8: Cervical incompetence – McDonald stitch Insertion 75

Obstetric case 9: Elective caeserian section due to previous caeserian section scar–inadequate pelvic 82

Obstetric case 10: Pre-eclampsia – emergency caeserian section life birth 89

Obstetric case 11: Post term successful induction of labour 96

Obstetric case 12: Deep venous thrombosis in pregnancy Live birth 102

Obstetric case 13: Twin gestation-favourable outcome after missed diagnosis and caeserian section 110

Obstetric case 14: HIV in pregnancy 117

Obstetric case 15: Malaria in pregnancy 124

OBSTETRICS LONG COMMENTARY:

Title: Knowledge attitude and practice of safe mother-Hood among women attending antenatal clinic In a rural district Narok-Kenya 131

APPENDIX:

Appendix I: Questionnaire: 170

Appendix II: Approval letter 177

GYNAECOLOGY SHORT CASES **PAGE**

Gynaecology case 1: Imperforate hymen: haematocolpos haematometrol cruciate incision 178
Gynaecology case 2: Symptomatic uterine fibroids – total abdominal hysterectomy 184
Gynaecology case 3: Endometrial hyperplasia – total abdominal hysterectomy bilateral salpingo-oophorectomy 193
Gynaecology case 4: Bartholins cyst – marsupilization 200
Gynaecology case 5: Vesico-vaginal fistula – successful repair 206
Gynaecology case 6: Incomplete abortion – manual vacuum aspiration 214
Gynaecology case 7: Burst abdomen after caeserian section – secondary repair 221
Gynaecology case 8: Long term contraception – norplant insertion 227
Gynaecology case 9: Polycystic ovarian syndrome – ovarian drilling 233
Gynaecology case 10: Cancer of the cervix – carcinoma in site 239
Gynaecology case 11: Ovarian tumour – mucinous cystadenoma 247
Gynaecology case 12: Ruptured ectopic pregnancy – right partial Salpingectomy 255
Gynaecology case 13: Chonocarcinoma – chemotherapy 263
Gynaecology case 14: Sexual assault 272
Gynaecology case 15: Pelvic abscess 278

GYNAECOLOGY LONG COMMENTARY:

Title: Abnormal cervical cytology among women in a rural Kenyan population - Narok District 284

APPENDIX:

Appendix I: Questionnaire: 323
Appendix II: Cytology Report Form: 329
Appendix I: Consent Form: 333
Appendix II: Approval letter 334

DEDICATION

To my parents, Philip and Rose Bartilol for their love, and inspiration to take medicine as a career. To my wife Rose and Ashely for being there always.My brother Chirchir and sister Sofie for always being by my side.

ACKNOWLEDGEMENT

I wish to express my most sincere gratitude to the Ministry of Health for sponsoring me through this program. I am grateful to Prof. Julius Meme, the Permanent Secretary-M.O.H, and Dr Richard Muga, the Director of Medical Services-M.O.H. for granting me study leave to undertake this training.

I would like to thank all the consultants, lecturers, senior Registrars and my fellow Registrars in the Department of obstetrics and Gynecology for their dedication and commitment to see that I achieved the necessary knowledge and skills during my training at the University of Nairobi.

I would also like to express my sincere gratitude to my supervisors, Dr Omondi-Ogutu and Dr M'Imunya Machoki for their efforts to see that my proposals and long commentaries were properly written by offering expert advice and guidance and also making very meaningful critique of the long commentaries and the short cases. .

I express my gratitude to those many friends who believed in me and encouraged me to keep going. .

Special thanks to Dr Oyieke, Dr Wanjala SHM, Dr N Gichuhi, Dr Gichangi, Dr Cheserem E, and Dr Kudoyi W for their wise counsel during days of my training.

I thank Dr Kizito Lubano and Dr Patrick O Amoth for their social and intellectual support. I benefited immensely from your suggestions and learned a lot from our casual conversations. I wish to thank Mr Erastus Muniu for statistical analysis of my data and Janet for typing most of this work. I thank the MOH -Narok, the residents of Narok and the health workers who enabled me to carry out the obstetric and gynaecologic studies.

DECLARATION

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

DR. KIGEN B BARTILOL

MBChB

UNIVERSITY OF NAIROBI

SIGNATURE _____

DATE 11/11/03

CERTIFICATE OF SUPERVISION

This is to certify that the long commentaries in this book by Dr. KIGEN B BARTILOL were researched upon under my guidance and supervision and that this book is submitted with my approval.

1. **DR. OMONDI-OGUTU M.MED (OBS/GYN)**

   CHIEF MEDICAL SPECIALIST (OBS/GYN)

   KENYATTA NATIONAL HOSPITAL,

   HONORARY LECTURER

   DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

   UNIVERSITY OF NAIROBI

   SIGNATURE _____

   DATE 4th February 2003

2. **DR. M'IMUNYA MACHOKI MMED (OBS/GYN)**

   LECTURER

   DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

   UNIVERSITY OF NAIROBI

   SIGNATURE _____

   DATE 31/1/2003

CERTIFICATION

This is to certify that Dr. Kigen managed Obstetric cases Nos. 2,10,12,14 and 15 and, Gynaecology case 15 under my supervision at Kenyatta National Hospital.

SIGNATURE_____

DATE 3.2.2003

PROF. S.B.O. OJWANG, MD, M.MED (OBS/GYN), DIP. ONCOLOGY

PROFESSOR

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

UNIVERSITY OF NAIROBI

CERTIFICATION

This is to certify that obstetric cases Nos. 3,4,7,9, and 13, and Gynaecology cases Nos. 2,5,6, and 8 were managed by Dr. Kigen under my supervision at Kenyatta National Hospital.

SIGNATURE 

DATE 14.02.2003

DR. J.B.O. OYIEKE MBChB, M. MED

SENIOR LECTURER AND CHAIRMAN

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

UNIVERSITY OF NAIROBI

CERTIFICATION

This is to certify that Obstetric cases 1,3,5,6,8, and 11 and, Gynaecology cases Nos 1,3,4,7,10,11,12,13 and 14 were managed by Dr. Kigen under my guidance and supervision at Kenyatta National Hospital.

SIGNATURE _____

DATE 2/7/03

DR. S. M. H. WANJALA, MBChB, M. MED

SENIOR LECTURER

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

UNIVERSITY OF NAIROBI

INTRODUCTION

The obstetric and gynaecology short cases presented were managed at Kenyatta National Hospital. The hospital is situated about 3 Kms from the Nairobi City Centre along Ngong Road. In addition to being a National referral hospital, it 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 GYNAECOLOGY SERVICES

The obstetric and gynaecology outpatient services are provided at the antenatal and gynaecology clinics, casualty department and Family Welfare Clinic (No. 66). The in-patient services are provided in labour ward, acute gynaecology ward and the lying-in wards (Antenatal and Postnatal wards) and the elective gynaecology ward.

In addition to the hospital laboratory services, the Department of Obstetrics and Gynaecology offers the following laboratory services for the hospital; semen analysis, hormonal radio-immunoassay, cytology, chromosome analysis, spectrophotometry, surfactant test and glucose tolerance test. Ultrasound fetal 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.The obstetric casuelty is located in labour ward and gynaecologic casualty is in ward 1D. 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 here for 24 hours a day.

ANTENATAL CARE

This is mainly a high-risk antenatal clinic although hospital staff members are also booked in this clinic. The booking is done every Monday morning. The criteria for

booking include – primigravida, grandmultipara, previous operative deliveries, medical conditions complicating pregnancy, bad obstetric history, those who have had delicate or difficult gynaecological operations like urinary fistula or myomectomy among other high risk factors.

For those that are booked, a detailed history of the patient's past obstetrical and gynaecological, medical, social history is taken. The patients are then sent to the laboratory for antenatal profiles, which include; blood group (Rhesus factor), serologic test for syphilis (VDRL), full haemogram (especially haemoglobin level), Urinalysis (protein/sugar) and Voluntary counselling ant 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. Proteinuria, glycosuria, blood pressure and weight gain are checked on every clinic visit.

The teenage mothers have their own antenatal clinic on Monday afternoons. This was started when adolescent pregnancies were found to be a major public health problem in the country. The important considerations are teenage pregnancy is associated with more complications such as hypertensive disease of pregnancy, psychological problems, low birth weight, and sometimes anaemia, and sexually transmitted infections.

During the antenatal period, any antenatal morbidity is managed accordingly either as outpatient or inpatient. At 36 weeks, clinical pelvic assessment is done on all 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 medical cadres also provide health education on pregnancy and its related problems in all clinical sessions. Emphasis is laid on better nutrition, regular clinic attendance, preparation for labour and delivery, post-partum care, breastfeeding 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 carefull to avoid infection. Those who are positive are told about the various available methods of preventing mother-to-child transmission of HIV. Mostly they are offered treatment with Niverapine 200mg at the onset of labour and their infants are given Niverapine syrup 2mg/kg Bwt within 72 hours after delivery. They are also encouraged not to breastfeed. There are other regimens of antiretroviral therapy to ptrevent Mother-To-Child HIV transmission such as the use of Zidovudine long course or short course, however, due to the constraints of cost, patient compliance and gestation at diagnosis, use of Nevirapine is found to be more feasible.

HOSPITAL ADMISSIONS

These 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. after hours or weekends. Unbooked patients are seen first in casualty before being sent to labour ward admission area. A house officer in conjunction with a senior house officer (registrar) sees the patients.

Other members of staff may be called if the need arises. Those in labour are admitted to the labour ward while those not in labour are admitted to the lying-in 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 6cm dilation, early use of oxytocin, and continous professional support. These measures are known to reduce the rates of caesarean sections and opearative vaginal deliveries as well as prolonged labour

and its attendant complications. Early amniotomy is now not routinely practiced since it has been shown not to have much effect on the duration of labour, and also in our set up with very high HIV infection (upto 30%), routine amniotomy is discouraged 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 and if they are in early labour with intact membranes a 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 examinations the cervical dilation in centimetres is noted and recorded. In addition, descent of the presenting part, presence and degree of moulding and the colour of the draining liquor is also noted and recorded. Artificial rupture of membranes is performed for all patients in active phase of labour. Urine analysis by dipstick is performed each time the patient passes urine to asses for proteinuria and gycosuria 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 hemorrhage or premature rupture of membranes. The patient is placed in lithotomy position, the vulva is cleaned with chlorhexidine 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. High-risk cases like multiple pregnancy, premature deliveries, all operative vaginal deliveries and breech presentations are delivered by the registrar in attendance. Clean delivery area and strict aseptic technique is adhered to during each delivery. The person conducting the delivery is always gowned and masked. The perineum is cleaned with chlorhexidine solution and sterile towels applied. She is encouraged to bear down with each contraction and to take deep breaths between contractions. Fetal heart is monitored every five minutes.

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

When the fetal 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 fetal 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. When found and is loose it is slipped over the head. If it is tight it is 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 grandmultiparity 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 (4th stage) and then transferred to the lying in ward for subsequent observations. "Rooming in" is encouraged and early initiation of breastfeeding within 30 mins is advocated as long as there is no contraindication. They 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 adviced on neonatal and infant care and breastfeeding as well as the symptoms for infection 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 from here 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 no 2/0 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 is contraindicated as in cardiac and hypertensive diseases or where expedite delivery is desired as in fetal 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 cephalo-pelvic disproportion and malpresentation. The fetal 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 fetal 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 fetal head the pressure is released. The mouth and nares 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 cases 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 crossmatching and two units of blood are reserved. Informed consent for general anaesthesia and operation is taken. The abdominal wall, vulva 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 vulval 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 is induced with intravenous thiopental sodium at a dosage, which is effective in sedating the patient, but it varies between 250-500 mg. Succinyl choline 50-80mg is also given intravenously for temporary muscle relaxation to enable endotracheal intubation. 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. 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 in two long artery forceps and opened. The incision is extended up and down to the incision limits taking care not to injure the bladder.

Wet abdominal packs are placed on either side of the uterus to prevent blood and liquor from running into the general peritoneal cavity. A Doyen's retractor is applied to reflect the bladder away as well as expose the 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 Doyen's retractor is shifted to include the lower part of the peritoneal fold in retraction of the bladder away from the lower uterine segment.

A small incision of about 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 semilunar 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 fetal 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 fetal 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 pressure. The trunk delivery follows readily.

Intravenous ergometrine 0.5 mg is given as 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 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 No. 1 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 1.2 mg 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 fully awake, then four hourly. Intramuscular pethidine 50-100 mg 6 hourly is given for 48 hours to relieve pain. Intravenous 5% dextrose and normal saline are given alternately as 500 mls four hourly until 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 and also urine culture is done. The stitches are removed after seven days of operation, after which the patient is discharged home with a case summary and having been explained about the nature and findings of operation. The mother is seen in the post-natal clinic after two weeks and the baby is also seen in the child welfare clinic in two weeks.

POSTNATAL 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 problems managed. Family planning advice is given and the patient referred to the family planning clinic for the various methods available.

CARE OF THE NEWBORN

All the newborn babies who are normal join their mothers after delivery unless the mother is moribund. The babies with problems or where complications are anticipated together with babies delivered by operative vaginal delivery or by caesarean section are all reviewed by a paediatric registrar. Those having problems or who may develop some problems are transferred to nursery in a warm incubator. The premature babies are managed in nursery until their weight is about 2000 grams 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 unit is run by the three firms in the department.

THE GYNAECOLOGY OUT PATIENT 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 an oncology clinic, which is on Friday mornings for follow-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 outptient in the clinic to eventually reduce the hospital stay. These include hemogram, urea and electrolytes semen analysis, pap smear, pregnancy test among others.

FAMILY PLANNING CLINIC

It is situated at the Family Welfare Centre (clinic 66). All methods of FP are offered. Also situated in this clinic is a theatre for diagnostic laparoscopy and voluntary surgical contraception procedures. Patients requiring interval sterilisation are counselled and referred to this clinic for the procedure by mini-laparotomy or laparoscopy.

GYNAECOLOGY IN-PATIENT SERVICES

Elective gynaecology admissions – ward 1B

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 1D

This is the emergency gynaecology ward having 32 beds but at the time of writing this introduction it averages 60 patients. An average of about 15 patients are admitted daily,

and more than two thirds of these are cases having abortion related complications. They are admitted mainly through the casualty department, which is located in ward 1D.

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 1D, using Karman's Cannula and syringe. They are discharged home immediately. Patients who have undergone emergency laparotomies 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 1D, where they receive emergency care i.e. blood transfusion, antibiotic 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 1B or radiotherapy unit for definitive management. The patients also receive continous care from the patient support centree 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 dilatation 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 I and III on Thursdays. The operations are done from 8.00 a.m to 5.00 p.m. The operations are performed under general anaesthesia as outlined below:

Intravenous sodium thiopentone and succinylcochline 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 Vesico-vaginal fistulae repairs are carried out under spinal anaethesia.

PRE-OPERATIVE PREPARATION

Patients for emergency laparatomies are prepared for theatre straight away in ward 1D. The abdomen is cleaned and shaved, stomach contents are aspirated if the patient has fed just before admission. Pre-medication is provided by atropine 0.6 mg intramuscularly half an hour before theatre. Blood is urgently cross-matched and an intravenous drip started.

For elective operations, basic and special investigations are done and the date of surgery fixed. The nature and purpose of the operation is explained to the patient after which she gives an informed consent. Blood is reqested and reserved for the day of the operation. The patient starves from midnight to the morning of the day of operation. The skin over the area of operation is cleaned and shaved. Pre-medication is provided by atropine at a dosage of 0.6mg and pethidine at 50 – 100 mg both intra-muscularly half an hour before wheeling the patient to theatre.

POST OPERATIVE MANAGEMENT

After the operation general anaesthesia is reversed and the patient wheeled to the recovery room where quarter-houly observation of blood pressure, pulse rate, respiratory rate and temperature are taken half hourly until she is fully awake. She is then transferred to the ward where observations are done four hourly.

Most laparotomy patients are kept in the ward for seven 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. Prophylactic antibiotics are given routinely. A check hemoglobin level is determined on the third postoperative day.

Before discharge the patient is informed about the findings at operations and a discharge summary is issued. Patients are reviewed in the gynaecology clinic after six weeks or earlier when there is an indication. Total abdominal hysterectomy 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 in situ 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 chlorhexidine and painted with iodine and then draped with sterile towels. As described above 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 infundibulopelvic 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 ligaments together with the ovarian vessels are divided between the clamps and ligated using chromic catgut no.1. The same is done for the opposite side.

The reflection of the bladder peritoneum onto the uterus is then freed by extending the incision in the anterior leaf of the broad ligament towards the midline. The bladder is thus separated from the lower uterine segment, the cervix and the vagina by careful sharp and blunt dissection of the fascial fibres beneath the bladder wall. Usually the bladder can be displaced into the lower pelvis quite easily, but if it is adherent, it is surgically released.

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 from the uterus by extending the incision around the tip of the distal clamp. This enables adequate ligation. Care should be taken to avoid freeing the tissue beyond the tip of the clamp, as this could permit bleeding from the collateral vessels that are not included in the clamp. Before clamping and cutting the uterine vessels it is always advisable to palpate the 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 though 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 suture. Here, particular care is exercised to avoid the pelvic portion of the ureter as it courses along the base of the broad ligament. Next the cardinal ligaments on either side of the uterus are clamped cut and ligated.

More commonly the uterus is removed by the open technique, in which the anterior vaginal fornix is opened initially with the scalpel and the vagina is circumcised by a sharp knife or scissors. 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 or eight futures. Particular care is taken when tying the lateral angles to ensure that the descending vaginal branches of the uterine vessels are securely ligated.

Suspension of the vaginal vault is done by tying the peritonealisation suture to the lateral and mid sutures of the vault. Peritonealisation is accomplished by means of a continuous No. 1 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 the uterosacral ligament, then through the infundibulopelvic 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, ischaemia and cystic changes of the ovary. After this, abdominal viscera are well inspected. If hemostasis has been achieved and instruments and swabs counts are

correctl, the abdomen is closed in anatomical layers. The post-operative management is the same as described earlier.

COUNSELLING CLINICS

There are three 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 patients who are poor and neglected by relatives. They counsel, treat and even assist patients find their way home.

THE HIGH RISK CLINIC (HRC)

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

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

THE NAIROBI HOSPICE

Workers here also offer counselling care in addition to management of terminal disease. They also offer narcortic 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 patients who are in need. It is situated on level 2 of the tower block.

THE MOTHER'S HOSTEL

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

OBSTETRIC SHORT CASES

CASE 1

OBSTRUCTED LABOUR – EMERGENCY CAESERIAN SECTION

Name: P.W.	L.M.P.:	29/9/02
Age: 21 years	E.D.D.:	6/7/02
IP. No.0823712	Parity:	0 + 0
D.O.A:14/7/02	D.O.D.:	25/7/02

PRESENTING COMPLAINT

She was a referral from Provide International with poor progress of labour

HISTORY OF PRESENTING COMPLAINT

She started having labour pains 36 hours ago when she went to Provide International and subsequently rupture membranes 24 hours ago and was put on IV fluids. She did not progress well and was referred to KNH for further management.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She is a primigravida, her last menstrual period was on 29/9/01, and her gestation by dates was 40+ weeks. She attended her antenatal clinic in Kariobangi Health Center. Her haemoglobin was 11.5g/dl, VDRL was negative and blood group was "O" positive. The antenatal period was uneventful.

Menarche was at 16 years and her menses were regular lasting 3 days and coming after 28 days. She has not used any form of contraception.

PAST MEDICAL HISTORY

She was hospitalized in 1996 with malaria and was discharged home well.

FAMILY AND SOCIAL HISTORY

She is a housewife, the husband is a carpenter. She does not smoke or drink. There is no family history of chronic illness.

EXAMINATION

She was sick looking, moderately dehydrated, afebrile, no pallor, blood pressure was 130/60mmHg, pulse rate was 105/minute, and temperature was 37.1'C.

ABDOMINAL EXAMINATION

Fundal height was 36 weeks with a single fetus in cephalic position. Fetal heart was 115/minute and irregular. The bladder was full, there was no Bandl's ring.

PELVIC EXAMINATION

There was normal external genitalia, the cervix was fully dilated and there was 3rd degree moulding and caput 2+. Vaginal wall was dry. Catheterization was difficult and blood stained urine 50mls was drained.

DIAGNOSIS

A diagnosis of fetal distress with obstructed labour was made.

MANAGEMENT

She was for an emergency caeserian section.

She was informed of the diagnosis and mode of management and informed written consent obtained for emergency caeserian section. The pubic hair was shaved and she was premedicated with atropine 0.6mg intramuscularly. She was wheeled to theatre, placed in semi lithotomy position, vulvovaginal toilet done, vaginal examination confimed earlier findings, catheter was in situ. She was then put in supine position, cleaned and draped IV augmentin was given and general anaesthesia induced.

The abdomen was opened in 3 layers via a Pfannestein incision. A lower uterine segment caeserian section was done. A life male infant birth weight 3950g who scored poorly 2/1,

4/5, 5/10 was extracted and taken to New born unit after resuscitation. Meconium stained liquor grade III was found. The placenta was manually removed and uterus was cleaned and closed in 3 layers. The bladder was found to be oedematous. The abdomen was closed in 3 layers and a self-retaining catheter put. General anaesthesia was reversed uneventfully.

POST OPERATIVELY

She did well post operatively and was put on IV augmentin and flagyl for one week. The catheter was retained for ten days. She was started on oral sips on the 1st post operative day. The baby succumbed on the 3rd day due to respiratory distress and the mother was started on bromocriptine 2.5mg tds. The catheter was removed on the 10th day and she was observed for one day where no leakage of urine was seen.

She was discharged home on the 11th postoperative day to be seen in the postnatal clinic in 5 weeks.

DISCUSSION

P.W. presented with obstructed labour for which an emergency caeserian section was done with poor outcome. She did well post operatively.

Obstructed labour is when despite good uterine contractions, the fetus can descend no further down the birth canal. It constitutes a major obstetric problem in developing countries (1,2). It is rare in developed countries because of good social and medical services (3). On a global basis, it is said to cause 8.1% of all maternal deaths (4).

The commonest cause is cephalopelvic disproportion (3). This could arise from reduced pelvic dimension due to childhood malnutrition, infection, poliomyelitis deformity, sickle cell disease or in teenagers,or an increase in the diameter of presenting part as in malpresentation and malposition like occipito-posterior position and brow presentation.

Others causes include, fibroids, cervical stenosis, rigid perineum, fetal abnormalities such as hydrocephalous and locked twins. Rare causes included vaginal stenosis, vaginal septum and malformed uterus.

Majority of patients have not usually booked into a health facility. Typical presentation is that of a prolonged difficult labour or that of an abnormal lie.

These women are usually restless, tired, pyrexic, with tachycardia arising from dehydration, infection, hypoglycaemia and acidosis.

They may have ruptured uterus but typically, there is a high bladder. Occasionally, the distended lower uterine segment may be separated from the upper firm segment by a Bandl's ring. These signs plus the suprapubic swelling are described as the three-tumour abdomen. The patient presented had a suprapubic swelling but no Bandl's ring (3). There may be an abnormal lie. There may also be features of fetal distress like tachycarida, bradycardia or irregular fetal heart. The patient presented had an irregular fetal heart.

If the uterus has ruptured there will be features of shock namely, tachycardia, hypotension and cold clammy extremeties. Fetal parts may be easily palpated.

Vaginal examination may reveal foul smelling vaginal discharge. In prolonged obstruction, the vulva and cervix will be oedematous there will be severe caput and moulding like in the case presented.

Urethral catheterization may be difficult with low urine out put and is often blood stained due to bladder trauma.

Management of obstructed labour involves correcting the fluid and electrolyte imbalance, control of infection, resting of the bladder and immediate relief of obstruction.

Blood should be taken for haemoglobin levels, urea and electrolytes, blood grouping and cross matching, high vaginal swab taken for culture and sensitivity. Intravenous (IV) dextrose and normal saline infusion should be set up and IV broad-spectrum antibiotics given. The bladder should be catheterized. Relieving the obstruction is determined by the cause of obstruction and if the baby is alive or dead. Caeserian section is the commonest preferred mode to relieve obstruction with a live fetus (1,3,5). Other methods are symphysiotomy, obstetric forceps or vacuum in a malrotated fetus (6). When the fetus us dead then destructive operation is one option. Craniotomy is the commonest destructive procedure (7). Decapitation and cleidectomy are performed for transverse lie and shoulder dystocia respectively (7).

Complications of obstructed labour include ruptured uterus, osteitis pubis, obstetric neuropraxia and fistula formation with the associated psychosocial consequences. There are also fetal complications, which include fetal distress, intracranial injury and fetal demise.

Prevention is aimed at the predisposing factors, which include poverty, ignorance and illiteracy. Socio-economic development is required to avoid childhood malnutrition, girl education in reproductive health and teenage pregnancy (2).

Provision of effective antenatal care, hospitals and infrastructure. Adequate referral system is mandatory. Active management of labour using partogram should be instituted at all levels.

P.W. was given Bromocriptine for phamacological suppresion of lactation.
Bromocriptine has been associated with strokes, myocardial infactions, seizures and physiaciatric disorders in the puerperal woman. This lead to the manufacturer removing lactation suppresion in 1994 as an indication for bromocriptine (8)
The woman requires to be reassured about stopage of lactation and use a well fitting brazziere. Ice packs and analgesics may be used for 12-24 hours to relieve the discomfort.

REFERENCES

1. Lawson J.B. Obstructed labour in: Obstetrics and gynaecology in the tropics and developing countries 1st ed. Edward Arnold 1967:172-207.
2. Wright E.A.: Obstructed labour Nigerian Medical practitioner 1988:18, 397-400.
3. Philpoh R.H. Obstructed labour: Clinic in Obst. Abd Gynaecol 1982: 9:625-40.
4. WHO marternal mortality, in: World Health Organization: safe motherhood WHO 1998:1-3.
5. John C.T. Difficult labour: Tropical journal of obstetrics and gynaecology. 1995 12 (suppl) 26-9.
6. O'Grady J.O., Gimovsky: Instrumental delivery: A lost art?. In Sludd J (ed). Progress in obstetrics and gynaecology vol. 10 Churchill Livingstone 1992:183-217.
7. Giwa-Osogie O.F., Assann B.B.: Destructive operations. In Studd J. (ed). Progress in obstetrics and gynaecology, vol. 6 Churchill Livingstone 1987:210-20.
8. Cunnigham F.G,Gant N.F,Leveno K.J,Gilstrap L.C,Hauth J.C,Wenstrom K.D Pueperium In Williams Obstetrics 21st edn Appleton and Lange.Conneticut 2001 pg411-412

CASE 2.

PRETERM PREMATURE RUPTURE OF MEMBRANES – EMERGENCY CAESARIAN SECTION

Name:	A.K.	L.M.P.:	27.2.00
Age:	24 years	E.D.D.:	5.12.00
IP No.:	0673142	Parity:	1+0
D.O.A.:	10.11.00	D.O.D.:	17.11.00

PRESENTING COMPLAIN

She came with complains of draining of liquor vaginally for one day.

HISTORY OF PRESENTING COMPLAIN

She was well prior to the onset of drainage of liquor, which was a lot in quantity, it was draining to the floor. There was no vaginal bleeding or associated abdominal pain. There was no history of trauma.

OPERATIVE AND GYNAECOLOGICAL HISTORY

She was para 1+0; her first delivery was in 1998 by spontaneous vertex delivery to a life male infant who weighed 3.5kg. Her last menstrual period was on 27.2.00. She was attending antenatal clinic at Kariobangi Health Center since 5 months. Antenatal profile was not done.

Her blood pressure and urinalysis were normal during the antenatal period.

Her menarche was at 14 years. Her menses are regular coming every twenty-one days and last 3 days. She has not used any contraceptive.

PAST MEDICAL HISTORY

This was not significant

FAMILY AND SOCIAL HISTORY

She is a married housewife the husband is a tailor. She does not smoke or drink. There is no family history of chronic illness.

EXAMINATION

She was in good general condition. She was not pale or jaundiced. There was no edema. Blood pressure was 130/80mmHg, pulse rate was 74/minute, and temperature was 36.8°C.

ABDOMINAL EXAMINATION

Abdomen was distended with a fundal height of 32 weeks. There was a single fetus in cephalic presentation, longitudinal lie. Fetal heart was heard and regular at 132 per minute. There was no abdominal tenderness.

SPECULUM

There was normal external genitalia. The cervix was posterior 1cm long and os closed. There was pooling of liquor in the posterior fornix and liquor was seen coming from the cervix on valsalva manoever.

Investigations

Full haemogram

White cell count	-	8.5 x 105/l
Neurotrophil	-	65%
Lymphocytes	-	35%
Haemoglobin	-	11.2g/dl

Ultrasound – single intrauterine fetus al 32 weeks gestation. Liquor was adequate and biophysical profile of 8/8.

DIAGNOSIS

A diagnosis of preterm premature rupture of membranes was made

MANAGEMENT

She was put on conservative management. She was for 4 hourly vital signs. (Temperature, pulse rate, blood pressure) twice weekly blood counts, fetal heart monitoring and observation for draining. She was started on augmentin and dexamethasone 12mg twice daily. Her antenatal profile was as follows;

Blood group - B +ve
VDRL - negative
Haemoglobin - 11.5g/dl

She did well and there were no signs of infection but she still continued draining. The blood counts remained normal.

Repeat scan after 10 days showed that there was marked reduction in the liquor and she was still draining.

A decision to deliver her by emergency caeserian section was made by virtue of the oligohydramnious and poor Bishops score. She was informed of the management and written consent obtained. She was premedicated with atropine and wheeled to theatre. Emergency caesarean section was done and a life male infant, birth weight 2100g was extracted who scored 8/1, 9/1 who was taken to New Born Unit.

She did well post operatively and was started on oral sips on 1st postoperative day and oral medication on day 2. She was discharged on the 7th postoperative day to the mother's hostel. The baby also did well and was discharged on the 13th day post operative day

DISCUSSION

A.K. came with preterm premature rupture of membranes. Emergency caeserean section was done due to oligohydramnios. Spontaneous rupture of membranes occurs as a normal component of labour. It is considered to be premature if it occurs before labour and preterm if it occurs before 37 completed weeks (1).

It is said to be prolonged rupture if labour has not set in within 24 hours (2). The incidence of premature rupture of membranes (PROM) at Kenyatta National Hospital was found to be 8.2 %(3). It is found to be in 10.71% of all pregnancies (2). In 94.1% of the cases, the fetus is mature, and in 6.1 the cases are premature or immature (2).

The exact etiology of premature rupture of membranes is unknown but several factors are associated with increased risk. These factors can be divided into life style factors, which includes smoking, poor nutrition, poor weight gain, drug abuse, young maternal age and physiological stress (1).

Others include genetic factors, pregnancy related factors like polyhydromnios, twin gestation, uterine fibroid, cervical incompetence and placental pathology. Others are iatrogenic like amniocentesis external cephalic version, Macdonald stitch insertion, contraction stress test and also prior history of PROM or preterm delivery (1).

Infection has also been implicated in many cases, but its mechanism of action is complex. Infections associated with PROM include bacterial vaginosis, trichomonas vaginalis, candida vaginitis and chlamydia trachomatis infections (1).

Indirect evidence is the presence of subclinical infections in patients with PROM. Bacterial endotoxins and white cell cytokines have been implicated. Other organisms involved in PROM include *Bacteroides spp,* and *Group B Streptococcus* that produce proteases, which reduce the strength of the chorioamniotic membrane. *Baceteroides*

spp, *anaerobic Streptococci* and *Gardenalle vaginalis* produce phosphalipase A_2 in high concentrations. Phospholipase A_2 cleaves the esterified form of arachidonic acid from fetal membranes releasing free arachidonic acid, which is a precursor of prostaglandins that may help a part in initiation of uterine activity (4).

Bacteria in ascending infection will stimulate the production of phospholipase A_2, endotoxins proteinases and exotoxins which will in turn stimulate macrophages, leucocytes and monocytes which then produce cytokines (4). Bacterial endotoxins will stimulate the production of TNF (tumour necrosis factor), interleukin I and prostaglandin E_2 (4)

Multiple processes predispose to PROM, increased intra- uterine pressure and stresses on fetal membranes may increase the risk of PROM if the membranes have been damaged by infection (5).

Thus there is an interaction between host and microbial factors in the pathogenesis of PROM. Diagnosis of PROM is based on history, physical examination and laboratory test. In the history, the time of leakage, amount of leakage and colour of fluid should be elicited.

The patient may complain of a gush of fluid, small amount of leaking or perineal moistness (4). This however, may be confused with vaginal discharge or urinary incontinence. The most important examination is a sterile speculum examination. This should confirm fluid leaking from the cervical os or pooling in the post vaginal fornix. If not clear, valsava manoever may show leaking of fluid (2). Confirmations may be done by testing the pH of the fluid with nitrazene paper which changes to blue from yellow if the pH is alkaline of amniotic fluid as compared to acidic vaginal secretion. Microscopic examination may demonstrate ferning or arborization of amniotic fluid (6). Cytology can also be used to identify lunago hair and fat particles, after staining with Sudan III or fetal cells after staining with Nile blue (6).

In case all the above may not confirm PROM intra amniotic injection of Evans blue or Indigo carmine then placing a sterile pad then assessing for staining may be done. Complications of intra amniotic injections include premature labour, fetal distress and allergic reactions (6). At the same time, amniotic fluid may be taken for surfactant testing and microscopy, culture and sensitivity (2). In the case presented the draining of liquor was obvious on sterile speculum examination.

The management of PROM depends on a number of factors of which gestation is most important. PROM between 23 and 34 weeks is best managed conservatively, unless there is chorioamnionitis or severe oligohydromnios where delivery is expedited. PROM above 34 weeks is managed by delivery with 24-48 hours, as fetal survival is high: In 75-85% spontaneous labour will follow PROM within 24-48 hours (1). If conservative management is decided the aim is to enable the fetus to attain maturity.

Obstetric scan is done to asses the amount of liquor and repeated weekly to monitor the development of oligohydramnios (6). Monitoring for chorioamnionitis involves maternal pulse and temperature observations, signs of uterine tenderness and/or colour and the smell of amniotic fluid.

Twice weekly, white cell counts more important is rising counts as count normally increase in pregnancy but counts above 16,000/µl should be alarming (2). Fetal tachycardia of > 160/minute is also alarming.

C-reactive protein may be more sensitive in detecting early infection (2). Other factors that may cause leukocytosis include corticosteroids (increase of 20-25%) and labour. Frequent abdominal examination may cause uterine tenderness (2).

Delivery in conservative management is expedicted if there is chorioamnionitis or oligohydromnios. Use of prophylactic antibiotics is controversial. The question is how long will the antibiotics be used if conservative management is chosen (1). In

our unit, once delivery is decided, intravenous antibiotics are given. There is shown to reduce fetal infection and puerperal infections (2).

Use of corticosteroids to enhance lung maturity is beneficial before 34 weeks. Some authors say it may not be beneficial as PROM by itself stimulates lung maturity. In our unit, corticosteroids are given for 24 hours and delivery delayed for 24-48 hours. Our patients was given dexamethasone Complications of PROM include preterm delivery with its associated complication of respiratory distress, intraventricular haemorrhage pulmonary hypoplasia, hypoglycaemia, necrotizing enterocolitis and cerebral palsy. Acute complications include cord prolapse, abruptio placenta and chorioamnionitis. Prolonged PROM may lead to severe oligohydromnios with its associated pulmonary hypoplasia, potter like syndrome of wrinkled skin and extraordinary flexion). Our patient was delivered due to severe oligohydromnios.

New treatment techniques of preterm PROM include use of fibrin glue to try and reseal the leak, use of a cervical cap and use of IgM to the mother to try and reduce neonatal infections (7).

REFERENCES

1. Cunningham F.G., Grant N.F., Levene K.J., Gilstrap L.C., Hauth J.C., Wenstram K.D. Preterm birth. Williams obstetrics 21st ed. Appleton and Lange, Connecticut 1997:661-687
2. Pernoll M.L. Late pregnancy complications In: Current Obstetrics and Gynaecology. Diagnosis and treatment 8th edn: Appleton and Lange Connecticut 1994: 331-336.
3. Wanjala S.H.M. Premature rupture of membranes and fetal/maternal morbidity and mortality. M.med thesis, University of Nairobi 1980.
4. Walkinshaw S.A.: Preterm labour and delivery of the perterm infant In: Chamberlain Greob Turnbulls obstetrics 2nd edn. Churchill Livingstone 1995 p609.
5. McGregor J.N., French J.J., Seok.: Premature rupture of membranes and bacterial vaginosis. Am J. Obstet Gyn 169, 463, 1993.
6. Davidson K.M.: Detectors of PROM. Clin Obst Gyne 29 (4) 1986.
7. Romero R., Athayed N., Mayman E. et al.: Premature rupture of membranes In: Reece E.A., Hobbin J.C. (eds). Medicine of the fetus and mother 2nd edn. Lippincott – Raven Publishers, Philadelphia p 1581: 1999.

CASE 3:

PLACENTA PREVIA TYPE III – EMERGENCY CAESERIAN SECTION

Name:	S.O.	Age:	21 years
IP No.:	0814613	D.O.A.:	8.6.02
D.O.D.:	12.6.02	Parity:	3+0 gravida 4
L.M.P:	3.10.01	E.D.D.	10.7.02
GBD:	30 weeks		

PRESENTING COMPLAIN

Patient came with complains of PV bleeding – 4 hours.

HISTORY OF PRESENTING COMPLAIN

She was well until 4 hours age when she started having pain less PV bleeding. It was profuse and reached the floor. There was no associated abdominal pain or history of trauma. She was a para 3+0 last delivery was in 1999. She attended antenatal clinic at Nairobi City Council Baba Dogo Clinic since 5 months.

Antenatal profile

Haemoblobin- 12.1g/dl

Blood group - B +ve

VDRL -negative

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a Para 3 + 0 gravida 4

Menarche was at 16 years, menses last 3-4 days regular coming every 28 days. She has not used any method of contraception.

Para 3 + 0

1st delivery 1995 SVD a live male

2nd delivery 1997 SVD a live male

3rd delivery 1999 SVD a live female

FAMILY AND SOCIAL HISTORY

She was married and a teacher by profession. Husband was also a teacher. She did not smoke or take alcohol and there was no history of chronic illness.

PAST MEDICAL HISTORY

This was not significant

EXAMINATION

She was sick looking, moderate pallor not jaundiced and clinically afebrile

With a weak pulse rate of 120/min, her blood pressure was 90/50 mmHg

ABDOMINAL EXAMINATION

The fundal height was 30 weeks with a single fetus in cephalic presentation. The uterus was relaxed and the fetal heart was irregular at 134beats per minute

Vaginal examination was not done due to active bleeding per vagina.

MANAGEMENT

IV line was secured and IV fluids (N/S alternating with Hartmans) given. Blood was taken for urgent grouping and cross matching. She was for E.U.A./emergency caeserian section in a double set up. She was then informed of the management and a written consent form duly signed. Pubic hair was shaved and premedicated with atropine 0.6mg and wheeled to theatre.

E.U.A./CAESERIAN SECTION

She was taken to theatre and put in supine position on the operating table and general anaesthesia was induced. She was put in lithotomy position and speculum done

Findings

Obvious bleeding at introitus

Blood clots in vagina

Cervical os 2cm dilated with active bleeding from the os

Cervix bluish colouration

She was then put in supine position cleaned and draped for emergency caeserian section. The abdomen was opened in 3 layers via Pfannestein incision. Lower uterine segment caeserian section done. The placenta was found to be low lying and a male fresh still birth in breech presentation extracted who weighed 2600g. Placenta previa (type III) extracted whole: uterus cleaned and closed in 3 layers. Abdomen cleaned and closed in 3 layers. After swab and instruments found okay. GA reversed uneventfully. Estimated blood loss approximately 300mls. Intra-operatively she received 2 units of whole blood and 2 units of fresh frozen plasma.

POST OPERATIVELY

She was put on nill by mouth, IV fluids (normal saline alternating with 5% dextrose) 1 litre 8 hourly and IV antibiotics (gentamycin/xpen) and pethidine.

On the 1st post operative day she was started on oral sips and ambulation encouraged. 2nd postoperative day she started light diet, oral medication and started on bromocriptine to stop lactation. 3rd post-operative she was done a check Hb – 9.6 g/dl and started on haematinics. 4th postoperative day, the wound was exposed and found clean and was discharged home for review in the gynaecology out patient clinic in 6 weeks.

DISCUSSION

S.O. presented with antepartum haemorrhage secondary to placenta previa type II. Emergency caeserian section was done with the delivery of a fresh still birth. Ante partum haemorrhage is described as third trimester bleeding. This is common and requires medical evaluation in 5-10% of the cases.

Bleeding may occur before the third trimester hence use of the term third trimester bleeding may be imprecise (1). Causes of ante-partum haemorrhage may be obstetric or non obstetric. Obstetric causes include bloody 'show', abruptio placenta, circumvallate placenta, placenta previa, vasa previa or blood dyscracias. Non obstetric causes include local lesions like cervicitis, cervical cancer, polyps or vaginal lacerations, varices or neoplasms (2). The patient presented had placenta previa. In placenta previa, the planceta is located near or over the internal os of the cervix.

This is further classified into 4 types depending on severity.

Type I: Low-lying placenta: the placenta is implanted in the lower uterine segment but does not reach the cervical os;

Type II: Marginal placenta previa: the edge of the placenta is on the margin of the internal os;

Type III: Partial placenta previa: the placenta partially covers the internal os;

Type IV: Total placenta previa: the placenta completely covers the internal os.

Incidence of placenta previa is 0.5% in the west (1). At KNH, Ojwang found the incidence to be 0.25% (3). In 1991 the incidence was found to be 0.9% (4).

The aetiology of placenta previa is unknown but risk factors include advancing maternal age, multiparity, previous caeserian section, cigarette smoking and increased placental area due to multiple pregnancy, anaemia, erythroblastosis. Thus, other factors include scarred or poorly vascularized endometrium or abnormal placentas (2).

The bleeding in placenta previa may result from mechanical separation of the placenta, placentitis or rupture of poorly supported venous lakes in the deadua basalis (2). Placenta previa is normally associated with various forms of adherent placenta. This could be accreta, increta or percreta (1). The characteristic finding in placenta previa is painless vaginal bleeding that does not appear until near the end of 2nd trimester or third trimester. This may vary from mild spotting to profuse bleeding (1).

The patient had painless vaginal bleeding.

The uterus is normally soft, relaxed and non-tender. The presenting part may be high and in 16% there may be an oblique or transverse lie.

Confirmation of placenta previa can seldom be confirmed unless adequate vaginal examination is performed – this however is not allowed unless a woman is in the operating theatre and all preparations for emergency caeserian section in place (1). Speculum examination however, can be done to rule out other non-obstetric causes of bleeding (5). Diagnosis by sonography is precise and safe and upto 96% accuracy. This has improved the diagnosis and management of placenta previa: Other methods that can be used include soft tissue placentography, amniography arteriography and displacement placentography (10).

Management of placenta previa depends on gestational age and amount of haemorrhage. If the pregnancy has not reached term 937 weeks), the aim is to manage conservatively upto to term. This involves monitoring for bleeding and hospitalization in a well equipped and staffed maternity unit. Once lung maturity is achieved then the examination under/without anaesthesia in an operating theatre is done: If the placenta is type I or type II anterior vaginal delivery is allowed. If type II posterior, III or IV caeserian section is done.

Conservative management is abandoned if there is onset of uncontrolled bleeding, premature rupture of membranes, intro uterine fetal death or onset of labour (7).

Complications of placenta previa are abnormal lie, cord prolapse in 1.7%, premature rupture of membranes in 11%, maternal shock and haemorrhage and increased perinatal mortality. Others include placenta previa, which can lead to severe post partum haemorrhage (1).

The patient presented came in shock and had emergency caeserian section with a subsequent perinatal mortality.

REFERENCES

1. Cunninghan F.G., Gant N.F., Leveno K.J. et al: Obstetrical haemorrhage. In Williams obstetrics 21st (edn). Appleton and Lange 1997, p619
2. Pernoll M.L.: Third trimester haemorrhage. In Current Obstetrics and Gynaecologic Rx and diagnosis 18th edn: Appleton and Lange 1994, p 398.
3. Ojwang S.B.O.: Placenta previa in KNH. M.Med thesis, University of Nairobi, 1974.
4. Kirima J.: Characteristics of patients with ante partum haemorrhage M.med thesis, University of Nairobi, 1981.
5. Hibbard L.T.: Placenta previa. In Sciano JJ (ed) Gynaecology and Obstetrics. Harper and Row , Philadelphia 1986
6. Laing F.C.: Ultrasound evaluation of obstetric problems relating to the lower uterine segment and cervix. In: Fleischer A.C. Manning F.A. et al (ed). Sonography in obstetrics and gynaecology. Principles and practice 5th edn. Appleton and Lange: Stamford CT. 1996, p 720.
7. Donald I.: Placenta preavia. In Practical obstetric problem 5th edn. Hyod-Luke Ltd. Asian economy edition, Singapore 1979, p 423.

CASE 4.

FULMINANT GENITAL WARTS IN PREGNANCY – ELECTIVE CAESERIAN SECTION – LIFE BABY

Name:	J.O	LMP:	19.6.01
Age:	22 years	EDD.:	26.3.02
D.O.A.:	14.3.02	D.O.D.:	26.3.02
IP No.:	0784706	GBD:	38 weeks

PRESENTING COMPLAINS

J.O. as referred from Prime Care Nursing Home with genital warts in pregnancy.

HISTORY OF PRESENTING ILLNESS

She was well until 2 months ago when she started having vulval itching which was progressive and associated with growth on the genitalia. There was bleeding after scratching and had associated vaginal discharge for which she was treated at Prime Care Hospital.

OBSTETRIC AND GYNECOLOGICAL HISTORY

She was a para 0+0, her last normal menstrual period was on 19/6/01 her expected day of delivery is 26.3.02 her gestation was 38 weeks 2 days.

She attended her antenatal clinic from six months gestation where her blood pressure and urinalysis were normal.

Antenatal profile:

VDRL	-	negative
Blood group	-	O rhesus positive

Menarche was at 13 years, menses are regular coming every 30 days and lasting 5 days. She has not been on any contraception.

PAST MEDICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY

She is a married housewife who reached standard 8. She does not smoke or drink alcohol. Her husband is a machine operator in a can factory.

EXAMINATION

She was a young lady in fair general condition. She was not pale, had no jaundice, oedema or lymphadenopathy. Blood pressure was 120.70 mmHg, pulse rate 76/mm, temperature 37.1°c.

ABDOMINAL EXAMINATION

Abdomen was distended moving with respiration. Fundal height was term with a single fetus in cephalic presentation, longitudinal lie with a regular fetal heart at 142 beats/minute.

VAGINAL EXAMINATION

There were extensive cauliflower like growths on both labia majora covering the introitus.

DIAGNOSIS

A diagnosis of fulminant genital warts in pregnancy was made.

MANAGEMENT

She was for elective caeserian section 2° due to fulminant genital warts. She was informed of the management and written consent obtained. She was pre-test counseled for HIV test.

Laboratory test results

Haemoglobin	-	11.6g/dl
Urea and electrolytes	-	Na^+ -145 mmol/l
	-	K^+ `-4.8 mmol/l
	-	BUN- 3.2mmol/l
ELIZA for HIV	-	Negative

She had posttest counseling for HIV and prepared for elective caeserian section. She was starved from midnight, atropine 0.6mg ½ hour before theatre and pethidine 50mg.

In theatre, she was put in semilithotomy position and vulvovaginal toilet done of which 100mls clear urine was catheterized. She was repositioned in supine position and general anaesthesia induced. She was cleaned and draped. The abdomen was opened in three layers via Pfannestein incision. LUSCS was done and a life female infant 3.5 kg in cephalic presentation extracted. Placenta was delivered complete. Uterus was cleaned and closed in 3 layers and abdomen cleaned and closed in e layers. General anaesthesia was reversed uneventfully.

POST OPERATIVELY

She was put on IV fluids, IV antibiotics (gentamycin and crystalline penicillin). On the second post operative day, she was started on oral sips and third post operative day she was put on oral medication. Wound healed well and she was discharged on the fourth postoperative day on amoxil and brufen for review on the 6^{th} post natal week.

FOLLOW UP

She was seen at the clinic on the sixth post natal week. The warts were still extensive but had reduced in size. She was scheduled for electrocautery excision of the warts as a dya case.

Investigations- haemoglobin 11.2g/dl

- urea and electrolytes Na^+ - 14.6mmol/l

K^+ - 4.6 mmol/l

BUN - 2.7mmol/l

In theatre, she was put in lithotomy position. After general anaesthesia was induced, electrocautery excision of the warts was done and heamostasis achieved. Blood loss approximately 400mls. She was discharged home on sitz baths and antibiotics to be seen after 2 weeks. Follow up after two weeks showed that the warts were excised and the wound well healed.

DISCUSSION

The patient presented was J.O. a 22 year old para 0+0 who presented with fulminant genital warts at 38 weeks gestation. She underwent an elective caeserian section with a favourable outcome to a life female infant who weighed 3500g.

Her postoperative period was unremarkable and was discharged on the fourth postoperative day.

Review at the postnatal clinic where she had electrocautery for the genital warts.

Condylomata acuminata or genital warts are caused by virus of the human papilloma virus group (HPV).

There are more than 100 HPV types of which about 30 types are transmitted through sexual contact. Genital warts are mainly caused by type 6 and 11 but may be caused by type 16,18 and groups, 40's and 60's (1). It is transmitted sexually with about 2/3 of people who have sexual contact with a partner with genital warts developing warts usually within 3 months. In the female they occur on the vulva, vagina or the cervix or anus. Rarely, they may be transmitted to infants during child birth and cause laryngeal papillomatosis which is potentially life threatening to the infant requiring frequent lazer surgery (2). The warts tend to increase in size during pregnancy and immunosuppression. This may be due to increased vascularity, increased vaginal secretions or altered immune systems (3).

Typically, the warts are initially reddish brown but with time, they become gray or white and cauliflower like. Treatment in pregnancy is aimed at reducing toxicity to mother and fetus and also realizing that they normally reduce in size after the pregnancy. In pregnancy, trichloroacelic acid 50% or 80% in 70% alcohol applied topically 3 times or once weekly is the least expensive (1). Cryotheropy and lazer ablation are preferred modes in pregnancy (4). The other modes of treatment using podopyillin and 5-flourourocil should not be used in pregnancy 95).

Occasionally, surgery may be used or the use of alpha interferon locally injected into the warts.

Occasionally the warts may grow to big sizes during pregnancy where they may bleed or obstruct the introitus necessitating the delivery by caeserian section as the case presented. Genital warts may be found in association with H.I.V. infection and caeserian section may be done to prevent mother to child transmittion(6)

The use of carbon-dioxide lazer therapy in pregnacy is getting more enthusiasm (6).

REFERENCES

1. Ferenczy A.: Epidemiology and clinical pathophysiology of condylomata accuminata. Am. J. Obst. Gynaecol. 172:1331, 1995.
2. Tseng C.J., Lin C.Y., Wang R.L., Cheng L.J.: Possible transplacental transmission of HPV. Am. J. Obst. Gynecol166;35:1995
3. Rando R.F., Lindheims Hosty L. et al.: Increased frequency of detection of HPV DNA in exfoliated cells during pregnancy. Am. J. Obstet. Gynecol 161:50 1989
4. Bergman A., Bhalia N.N., Broem E.M.: Cryotheroms for treatment of genital condyloma during pregnancy. J. Reprod. Med. 29 432 1984
5. American College of Obstetricians and Gynaecologists: Genital human papilloma virus infection. Technical Bulletin no. 193 June 1994.
6. Ferency A.: Treating genital condylomata during pregnancy with carbon-dioxide 1030 Am. J. Obstet Gynecol 1984 148:9

CASE 5

CARDIAC DISESE IN PREGNANCY

Name: C.W.		L.M.P.:	1.10.01
Age:	35 years	E.D.D:	8.7.02
IP No.:	0811178	Para:	3+0
D.O.A.:	27.5.02	D.O.D.:	24.7.02

Known patient with rheumatic heart disease since the age of 10 years who came with complains of difficulty in breathing orthopnea, dyspnea at rest for 6 weeks.

HISTORY OF PRESENTING ILLNESS

She was well until April when she developed the above symptoms. She had dyspnea at rest and orthopnea, which was relieved by being propped up. She also had paroxysmal nocturnal dyspnea. She also had palpitation and cough. The cough was not productive. She subsequently developed leg swelling which was worse after walking. She was admitted at Kerugoya District Hospital for one week before being transferred to KNH for further management.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

Para 3+0 1st delivery 1998 SVD to live male infant weighing 3.9kg

2nd delivery 1993 SVD to live male infant 2.7 kg

3rd delivery 1997 SVD to live male infant 3.2 kg

She had no complications in previous pregnancies. Menarche was at 14 years, menses were regular lasting 3 days and coming after 24 days. Contraceptive use, she used oral contraceptive in 1987,and copper T between 1993and1997.

PAST MEDICAL HISTORY

Had been getting monthly penicillin injection in the 1970's and 1980's but stopped. Was admitted in 1970 because of the cardiac disease. No history of blood transfusion or surgery.

FAMILY AND SOCIAL HISTORY

She has been married since 1986. She is a small-scale farmer and the husband a tailor. No family history of diabetes, hypertension or cardiac disease. They both don't drink and smoke.

EXAMINATION

Sick looking in respiratory distress, no pallor, jaundice or lymphadenopathy. She had bilateral pedal oedema. Blood pressure was 80/60mmHg, temperature 36.7°C.

CARDIOVASCULAR SYSTEM:

Pulse rate 100/min, which was irregularly irregular

Praecardium was heaving

Palpable thrills in the left parasternal border

Apex beat was at the 6th intercostal space, anterior axillary line

On ausculation, there was a gallop rhythm with pansystolic murmur and diastolic murmur.

RESPIRATORY SYSTEM

No abnormality detected

ABDOMINAL EXAMINATION

Fundal height was 34, fetus in cephalic presentation, longitudinal lie with regular fetal heart rate of 132/minute. Other systems were essentially normal.

Investigations

Haemoglobin	Haemoglobin – 11.4g/dl
	PCV - 33.7%
Urea and electrolytes	Na+ -133mmol/l
	K+ -4mmol/l
	BUN -6.2mmol/l
	Creatinine -74 μmol/l

Echocardiography showed; moderate mitral stenosis, mitral regurgitation, moderate pulmonary hypertension and mild aortic regurgitation consistent with rheumatic heart disease.

Obstetric scan showed a single fetus in cephalic presentation, placenta- fundal anterior, gestation 34 weeks.

She was graded as cardiac disease grade IV at 34 weeks gestation. She was to be propped up in bed and was put on the following treatment;
Digoxin 0.125mg P.O. OD
Ranferon T BD
Amoxil 50g PD MD
Heparin 5000iu SC BD

She was then followed up with fetal monitoring, 4 hourly blood pressure, pulse rate, respiratory rate and temperature. She was for delivery vaginally since there was no obstetric indication for operative delivery. She went into spontaneous labour on 10.6.02, at 36 weeks gestation. She was wheeled to the labour ward.

MANAGEMENT OF LABOUR

She was put in left lateral position and propped up position. She was found to be 4cm dilated and the head was 2/5 down with regular fetal heart and good contractions. An IV line was put started and oxygen by mask was given and prophylactic antibiotics of augmentin and morphine started stat.

She progressed well and had had spontaneous vertex delivery to a live male infant 2300grams who scored 8/1, 10/5 and was taken to New Born Unit due to low birth weight. The placenta was delivered by controlled cord traction. An IV drip with 20iu sytocinon was run after delivery and IV frusemide 80mg given.

POST DELIVERY CARE

She was transferred to the acute room and kept in propped up position. Vital signs were observed ½ hourly and she continued on IV antibiotics digoxin, heparin and haematinics. She was reviewed by a cardiologist and found to be stable. She was transferred to the postnatal ward after 48 hours. She developed ascities and oedema in the ward and lasix was increased to 80mg TDS. She did well and was discharged on the sixth week with the baby.

She was advised on using barrier method (condom) as she discussed with the husband on possible interval surgical sterilization.

DISCUSSION

C.W. was admitted with cardiac disease grade IV. She subsequently delivered by SVD to a live male infant. They both did well postnataly.

Cardiac disease in pregnancy is associated with significant maternal morbidity and mortality (10). Incidence of cardiac disease is at about 1.3% (1). At Kenyatta National Hospital, the incidence is 0.6 (2). In developing countries, rheumatic heart disease is still the commonest cause of heart disease (3). In the developing world, rheumatic heart disease is now less common and congenital heart disease are seen more commonly (3).

In his study, Ngotho found rheumatic heart disease responsible for 86.4% of cardiac disease in pregnancy. Other causes of heart disease in pregnancy include hypertension, thyroid, coronary, syphyilitic, cardiomyopathy, pericarditis and other congenital heart diseases (1,3). Majority of the patients with cardiac heart disease in pregnancy were found to be young. Spencer and Makene in 1977 found majority of patients to be in the age group of 20-24 years (4). C.W. had rheumatic heart disease and was 35 years old.

The predominant lesion in rheumatic heart disease was found to be mitral stenosis (5). C.W. had mitral stenosis, mitral regurgitation and pulmonary hypertension since childhood.

Cardiovascular changes in normal pregnancy tend to worsen or unmask cardiac disease. They also tend to make it difficult to diagnose heart disease (1).
During pregnancy, total blood volume increase by 50% above non-pregnant levels by 32 weeks.

Cardiac out put increases early and by 12 weeks it is 36% above non-pregnant levels. During labour, cardiac out put increase by 34% in 1st stage with further increase in 2nd stage due to increase in stroke volume and heart rate (6). There is also steady increased in blood pressure. Signs and symptoms associated with heart disease are often present in normal pregnancy. These include fatigue, dyspnea, orthopnea, oedema, and palpitations.

In pregnancy, dyspnoea at rest, orthopnoea, angina, haemophtysis and palpitations with arrythmias and syncope signify heart disease. Others include cyanosis, finger clubbing, raised jugular venous pressure, cardiomegally and parasternal heave (5). A systolic murmur is a normal finding but diastolic murmur, pansystolic murmur, late systolic and ejection systolic murmur may signify cardiac disease (5).

Cardiac disease can be graded according to function disability according to the New York Heart Association classification. This is based on past and present disability and is not influenced by physical signs.

Grade I: Uncompromized patients have signs of cardiac disease but no symptoms limiting ordinary life.

Grade II: Slightly compromised patient with cardiac disease and slight limitation to physical activity. They have dyspnoea on ordinary physical activity.

Grade III: Markedly compromised patient with cardiac disease and marked limitations of physical activity. They have dyspnoea on mild physical activity

Grade IV: Severely compromised. They have cardiac disease and inability to perform any activity without discomfort. They have orthopnoe or dyspnoe at rest.

Patients with pure mitral stenosis, previous congestive cardiac failure of cardiac surgery falls into this class (5).

They can also be classified according to the risk of mortality associated with pregnancy in 3 classes (7).

i. Low risk – mortality < 1%:

This includes atrial septal defect, ventricular septal defect, patent ductal arteriosus, corrected tetratology of fallot, procaine valve, mild mitral stenosis and pulmonary/tricuspid disease.

ii. Medium risk – mortality 5-15%:

Congenital heart disease without pulmonary, hypertension, hypertrophic obstructive cardiomyopathy, symptomatic mitral stenosis, Ebstein's anomaly,

aortic stenosis, coarctation of aorta, uncorrected teratology of fallot, artificial valve and previous myocardial infaction.

iii. High risk – mortality 25-50

Severe aortic stenosis, pulmonary hypertension with reversed central shunt and marfan syndrome with aortic involvement.

The patient presented had cardiac disease grade IV. Successful management of cardiac disease in pregnancy requires a close cooperation between the cardiologist and abstinence. A combined clinic is preferable with pre-conceptional visit preferable. Accurate assessment of the disease and counseling of risks and outcome. Patient with high risk should be advised to terminate the pregnancy in 1st trimester if possible but not infrequently high desire for children may lead to dismissal of the advice. Surgical correction can be done then. During antenatal period investigation should be done this include electrocardiograph (ECG), echocardiography and obstetric ultrasound.

Careful monitoring to avoid heart failure should be done with special emphasis on risk factors, which include infections (especially urinary tract), hypertension, anaemia and multiple pregnancies.

Patients with grade I and II disease are seen weekly until term then admitted to await labour. Patients with grade III and IV are admitted through the duration of pregnancy. C.W. was admitted on the 1st contact at 34 weeks. In management of labour, spontaneous labour and vaginal delivery is preferred. Most patients have rapid uncomplicated labour especially of taking digoxin (5). Caeserian delivery is limited to obstetric indication. The patient is propped up and vital signs monitored ½ hourly.

An analgesic is important as it reduces cardiac out put and anxiety. Epidural analgesia acts as agood analgesic and also helps to reduce cardiac output by reducing pre-load and causing pheripheral vaso-dilation. Narcortic analgesics (Morphine, Pethidine) are also used. Oxygen is also given to ensure optimal saturation of the blood and also to prevent decompensation(5)

IV fluids should be carefully monitored to avoid fluid overload and pulmonary oedema associated with injudicious fluid loading.

Second stage of labour should be shortened by elective assisted delivery to minimize dramatic increase in blood pressure. In our set up, elective vacuum extraction is carried out (5). C.W. had a premature labour and hence elective vacuum was not done.

Close monitoring of third stage will prevent haemodynamic changes associated with post partum haemorrhage. Oxytocin is preferable to ergometrine as ergometrine will come cause hypertension and peripheral vasospam associated sudden intravascular overload (1,5). Use of prophylactic antibiotics to prevent infective endocarditis is necessary as complications often occur without warning. Bactereamia following normal delivery is rare but many obstetricians prefer to give antibiotics (8).

Our patient received prophylactic antibiotics. In our unit, cardiac diseases is observed for at least 24 hours in labour ward before transfer. Our patient was observed for 72 hours.

Post partum period is also critical and monitor for infective endocarditis, congestive heart failure and thromboembolic disease is a must. Our patient was put on prophylatic heparin antibiotics and early mobilization was emphasized.

Of cardiac disease in pregnancy, grade II and IV account for 85% of the 0.5% mortality rate; Complications of cardiac disease in pregnancy include premature labour and delivery, low birth weight and higher incidence of congenital heart disease (1,5). Our patient had a premature delivery.

Contraception post partum is important and surgical sterilization is the preferred method (1,8). Other methods that can be used are oral contraceptives and condoms. Use of oral combined pils is avoided in those with mitral valve disease and those with mechanical

valves where risk of thrombosis and embolism is high.Most of these patients require anti-coagulation with warfarin (8)

Intrauterine devices are not frequently used due to the high frequency of infection (8).

REFERENCES

1. McPaul P.B., Dorman J.C., Bayle D.: Pregnancy complicated by maternal heart disease. A review of 519 women. Br. J. Obstet Gynaecol. 1988; 95:861-7
2. Ngotho D.K.: Cardiac disease in pregnancy at KNH: M.Med thesis, University of Nairobi, 1982.
3. Szekely P., Turner R., Snaith L.: Pregnancy and the changing patterns of rheumatic heart disease: Br. Heart J. 1973:35:1293-303.
4. Spencer D.D., Makene J.W.: Rheumatic heart disease in Tanzania . E.Afri. Med. J. 49:900 1972.
5. Cunningham F.G., McDonald P.C., Levene K.J., Gant N.F., Gilstrap L.G.: Cardiovascular diseases: Williams obstetrics 21st edn. Appleton and Lange, Connecticut p1086 1997
6. Robson S.C., Dunlop W., Boys R.Y., Hunter S.: Cardiac output during labour. BMJ 1987 296:1169-72.
7. Forfar J.C.: Heart disease in pregnancy. In: Weatherall D.J., Ledingham J.G., Warrell on ed. Oxford textbook of medicine, Oxford University Press 1996 134
8. De Sweil M.: Cardiovascular problems in pregnancy. In: Chamberlains G. ed Turnbull. Obstetrics London Churchill Livingstone 199 369-381.

CASE 6:

RHESUS NEGATIVE-LIVE BIRTH

Name:	J.N.	L.M.P.:	25.9.01
Age:	26 years	E.D.D.:	2.7.02
IP No.:	0822122	Parity:	1+0
D.O.A.:	5.7.02	D.O.D.:	8.7.02

PRESENTING COMPLAIN

She came with complain of lower abdominal pains for 6 hours. She had associated draining of liquor for 1 hour.

HISTORY OF PRESENTING COMPLAINS

She was okay until 6 hours ago when she developed lower abdominal pains, which were increasing in frequency and duration. She also developed draining of liquor. She had no vaginal bleeding.

OBSTETRIC AND GYNAECOLOGY HISTORY

She is a para 1+0 last, last delivery in 1995 at Nyeri Provincial Hospital to a live male infant who weighed 3.2 kilogram.

She attended antenatal clinic at Kenyatta National Hospital since 6 months where her urinalysis and blood pressure were normal. Her haemoglobin was 12.1g/dl, HIV was negative and VDRL negative. Blood group was B negative. She had received anti-D after her last delivery. Indirect coombs test was negative.

Menarche was at 16 years and menses are regular and coming after every 30 days and lasting 2 to 3 days. She used an intra-uterine contraceptive device since 1996 to 2000.

PAST MEDICAL HISTORY

This was not significant and there is no history of blood transfusion

FAMILY AND SOCIAL HISTORY

She is a married accounts clerk. Her husband is a businessman. She does not smoke or drink alcohol. There is no family history of chronic illness. Her husband's blood group is 'O' positive.

EXAMINATION

She was in good general condition, not pale nor jaundiced. There was no oedema. Her blood pressure was 120/70mmHg, pulse rate was 88/min.

ABDOMINAL EXAMINATION

The fundal height was 36 weeks, with a single fetus in cephalic presentation. Fetal heart tones were heard and regular at 144/minute. There were good contractions.

VAGINAL EXAMINATION

There was normal external genitalia with drainage of clear liquor. Cervix was central, soft, fully effaced and 7 centimeters dilated. Presenting part was well applied and 3/5 down.

MANAGEMENT

She was put on a partogram, 1M tramol and for review after 4 hours.

She delivered by spontaneous vertex delivery to a live male infant who scored 8/1, 10/5 and weighed 3300 grams. The placenta was delivered by controlled cord traction complete.

The cord blood was taken for Hb level, blood group and direct comb levels.
Serum billirubin levels and reticulocyte counts were not done.

The Hb was 16g/dl, blood group was B positive and direct comb test was negative

The mother was given anti-D globulin 300mg on the second post natal day and discharged home for post natal review after six weeks.

DISCUSSION

The patient presented is an unsensitized rhesus D negative mother who had a spontaneous vertex delivery. The baby's blood group was B positive and she was given anti-D.

In the red blood cells there are 250 recognised antigenic factors of which ABO and rhesus are most common. The rhesus factor was discovered by Landsteiner and Weiner in 1940s (1).

Inheritance of the rhesus antigen is half from mother and half from the father, therefore the fetus may have a different blood group from the mother (2). Rhesus antigens are inherited independently of the other blood groups. The rhesus (Rh) blood group is the most complex human blood group. Rhesus antigens are grouped into 3 pairs Cc Dd and Ee. The major group is Rh D or rhesus factor which is responsible for severe haemolytic disease of the new born.

There is considerable variation of rhesus negatively the highest being the Basque populations with 30-35% rhesus negatively, Caucasians 15-16%, African Americans 7-8% and nil for the Mongoloid races (2). The incidence in Nairobi is reported at 5% of all antenatal clinic attending mothers (3).

At Kenyatta National Hospital the incidence is said to be 4.1% (4). Rhesus isoimmunization occurs either following a rhesus negative mothers carrying a rhesus positive fetus or following blood transfusion.

With no apparent predisposing factor, fetal red cells have been found in maternal circulations in 6.7% of women in 1st trimester, 15.9% in 2nd trimester, 28.9% in 3rd trimester (2). Predisposing factor to fetematernal haemorrhage include abortions, amniocentesis, placenta previa, abtuptio placenta, abdominal trauma, fetal death, manual removal of placenta or caeserian section. 0.1ml of fetal blood is enough to cause sensitization but 0.25mls is the critical volume (5). 30% of mothers do not become sensitized (non responders) and ABO incompatibility offers protection (2,5).

Initial response of a negative individual to rhesus positive also is formation of IgM antibodies later IgG antibodies are formed. This is normally in 6 weeks to 6 months. IgM do not cross the placenta but IgG cross the placenta and cause haemolysis of fetal cell (2,3). The initial isoimmunization reaction is minimal but becomes more severe in subsequent pregnancies (5). The patient presented was 'O' rhesus negative. Following sensitization haemolytic disease of the new born occurs.

The IgG antibodies cross the placenta and destroy the fetal red cells. This leads to haemolysis and anaemia stimulating extromedulary erythropoesis of nucleated immature red cells. If the haemolysis is severe and exceeds production erythroblastosis fetalis occurs due to severe anaemia (3,5). This characterized by anaemia, extramedulary erythroporesis, ascites, heart failure, oedema and pericaridal effusion.

Haemolysis leads to heame production and hence billirubin production both of which are neurotoxins but are effectively cleared by the placenta. Following delivery, severe anaemia and billirubin accumulation leads to more red cell destruction and kernicterus following billirubin deposition in the Basal ganglia (5).

Following introduction of anti-D gamma globulin has reduced the frequency of rhesus isoimmunization (6). The administration of anti-D prevents rhesus isoimmunization but competitive inhibition and by also preventing antigen processing (6).

Routine administration of anti-D prevents up to 95% of rhesus sensitization (7). Anti D should be given during the last trimester and post natally within 72 hours. There is a 1-6% failure rate following post partum administration of anti D as compared to 0.1% of antenatal administration (7).

The standard recommendation is to give anti D within 72 hours. If the woman has not been given anti D, she should still be treated. Some other recommend treatment upto 72 hours (2).

Anti D should also be given following abortion, amniocentesis and antepartum haemorrhage. If the pregnancy goes for more than 12 weeks following the injection of anti D a repeat dose should follow. Failure of prevention of rhesus isoimmunization may occur if there is allergic reaction to anti D, failure to give treatment after abortion, amniocentesis or delivery, occurrence of sensitization is infancy or before prophylaxis is given or failure of anti D to give protection due to a massive haemorrhage (8).

Once isoimmunization has occurred as confirmed by indirect Coomb's test, regular monitoring the fetus should be done (2). A litre higher than 1:16 suggests severe haemolytic diseases. Abnormal heart rate patterns are seen in severe rhesus isoimmunization (6). Sinuosidal and deceleration patterns are seen with low haemoglobin and high perinatal levels (6).

Risks of rhesus sensitization is 16% in ABO compatible infant and 1.5-2% in ABO incompatible infant (2). Ultrasound examination should be done at 14-16 weeks to confirm gestation and look for features of ascites or oedema. At 18-22 weeks amniocentesis should be done and spectrometry of the amniotic fluid done. This is then plotted on the Lileys charts against gestation. The charts put the fetus into three zones.

The unaffected or mildly affected will fall in zone I. This will require repeat amniocentesis every 2 to 3 weeks and delivery should be a term. Zone II will be at moderate risk and amniocentesis is repeated every 1-2 weeks. Delivery is normally before term or as soon as lung maturity is achieved.

Zone III is the severely affected and intervention is usually required. Exchange transfusion or immediate delivery may be required. In our unit, exchange transfusion is not usually done (2). In our unit, perinatal mortality is 600 per 1000 of the rhesus isoimmunised patients (9).

Other methods like plasmaphoresis, immunosuppression with high dose steroids have been tried unsuccessfully.

Rhesus negative donor sperm can be used for those with repeated pregnancy losses (7). Out patient remained unsensitized. Prevention of sensitization is still the best option.

REFERENCES

1. Norman BB. John S.W.: Rhesus isoimmunization in pregnancy: A review. Obstet Gynecology survey 1993 46(12) 80.
2. Pernoll M.I.: Late pregnancy complications In: Current obstetrics and gynaecology. Diagnosis and treatment 8th edition. Appleton and Lange 1994:339-343.
3. Mati J.K.G., Aggarwal V.P., Sanghvi H.G. et al. Nairobi birth survey II A: Antenatal care in Nairobi J. Obstet Gynecol. East and Central Africa. 1983 2(1):1
4. Mulandi T.M.: A 2 year prospective study to show effectiveness of anti D gamma globulins in preventing rhesus isoimmunization in rhesus negative pregnant (Africa) women in KNH. M.Med thesis U.O.N. 1985.
5. Whitfield C.R.: Blood disorders in pregnancy In: Dewhurts Text book for obstetrics and gynaecology for post graduates 5th edition edited by Charles R. Whitfield Blackwell science 1995 chapter 16 page 228-250.
6. Magnus S.: Rhesus isoimmunization. New perspective in maternal fetal medicine. Obstet Gynaecol Surv. 1991, 46 v(4):71
7. Thornton J.: Efficacy and long term effects of antenatal prophylaxis will anti-D immunoglobulins. Obst Gynecol Surg. 1990 45:117.
8. Creasy R.K., Resnick R.: Maternal – fetal medicine principles and practice Saunders 1984
9. Kaggia J.W.: Review of management of rhesus negative mothers at KNH. 1975 1986-M.med thesis UON 1986.

CASE 7:

URINARY TRACT INFECTION IN PREGNACY – SUCCESSFUL DELIVERY

Name:	S.W.	L.M.P.:	19.8.01
Age:	32 years	E.D.D.:	26.5.02
IP no:	0682499	Parity:	2+0
D.O.A.:	13.5.02	D.O.D.:	15.5.02

PRESENTING COMPLAINS

S.N. came with complains of lower abdominal pain for 2 days

HISTORY OF PRESENTING COMPLAINS

She was well prior to onset of lower abdominal pains, which were increasing. She had not vaginal bleeding or discharge. She also had dysuria and frequency.

OBSTETRICS AND GYNAECOLOGICAL HISTORY

She is a para 2+0 gravida 3. The last normal monthly period was on 19/8/02. Her expected date of delivery was 26/5/02. Her gestation was 38 weeks. She attended antenatal clinic in a Langata private clinic since 5 months gestation. Her antenatal profile was as follows:-

Haemoglobin	-	11.5g/dl
VDRL	-	negative
Blood group	-	B positive

Her urinalysis and blood pressure were normal through out the antenatal visits. She has had two previous deliveries in 1998 and 1999 which were by spontaneous vertex deliveries at Nakuru Nursing Home. The first was a male who weighed 3000grams and the second a male who weighed 3400grams. They are both alive and well. Her menses are irregular coming after 24 to 30 days and lasting 3 days. She was on microgynon from 2000 to March 2001 . Menarche was at 14 years.

PAST MEDICAL HISTORY

This was not significant

FAMILY AND SOCIAL HISTORY

She is a secretary by profession and married to an accountant. She does not smoke cigarettes or drink alcohol. There is no family history of chronic illness.

PHYSCIAL EXAMINATION

She was in fair general condition. She was not pale, no oedema and clinically febrile. Her blood pressure was 120/70mmHg, temperature 37.8°C, pulse rate was 92/minute.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended with a single fetus in cephalic presentation and longitudinal lie the fetal heart was heard and regular at 140 beats per minute. There was marked tenderness in the suprapubic area. She had tenderness in the flanks.

PELVIC EXAMINATION

There was normal external genitalia, the cervix was long, firm and posterior. There was no discharge.

DIAGNOSIS

A diagnosis of acute mylonephritis at 38 weeks gestation was made.

MANAGEMENT

Mild stream specimen of urine as taken for microscopy, culture and sensitivity. She was started on IV augmentin and paracetamol and IV fluids.

Laboratory results

Urinalysis

Ph - 6

Glucose - nil

Protein - nil

Specific gravity - 1010

Blood +

Leucocytes +++

Deposit:

Pus cells 15-20/HPF

No TV or yeast cells seen

Culture and sensitivity: E. coli seen, sensitive to augmentin, nitrafurantoin, cefuroxime, cotrimaxazole. Resistant to ampicillin, nalidixic acid.

She did well and was afebrile on the second day and was discharged home on the third day to attend antenatal clinic on augmentin and paracetamol.

FOLLOW UP

She came in labour on 28/5/02 and had a spontaneous vertex delivery to a live female infant who weighed 3350grams. Both mother and child did well and were discharged home on the 2nd post natal day to attend post natal clinic at the nearest health facility.

DISCUSSION

S.W. came with urinary tract infection in pregnancy. She was treated and discharged home.

Urinary tract infection (UTI) during pregnancy is common, asymptomatic bacteriuria has an incidence of 2-7% as in the commonest of the urinary tract infections. Others include cystitis and pyleronephritis. Incidence of acute cystitis is 1% while that of pyelonephritis is 1-2% (1). Asymptomatic bacteriuria is when there is actively multiplying bacteria within the urinary tract. Diagnosis is obtained by finding more than 100,000 organisms per ml of clean voided specimen of urine. If not treated, 25-30% will develop acute symptomatic infection but with treatment, the rate is only 10% (2).

Pregnant women are at an increased risk of UTI's starting from the sixth week and peaking at 22 to 24 weeks. 90% of pregnant women develop ureteric dilation, which remains until delivery. Pregnant women also have increased bladder volume, decreased bladder tone together with reduced urethral tone leading to urinary stasis and urethrovesico reflux (3). 70% of women also develop glycosuria in pregnancy. All these lead to increased likelihood of developing UTIs in pregnancy.

Organisms that cause UTIs are those usually found has normal perineal flora (2). Escherichia coli is responsible for 80-90%. Others are gram negative rods like proteus mirabilis and klebsiella pneumoniae. Gram positive rods like Group B Streptococci and Staphylococci are less common. Others that are less common include ureaplasma ureolyticum, gardenella vaginalis and chlamydia trachomatis (4). The three principle presentation (4) of UTI's are asymptomatic bacteriuria, acute cystitis and acute pyelonephritis.

Asymptomatic bacteriuria is usually found by screening of urine and finding growth of at least 10^5 organisms per ml of urine, however, lower colony counts may represent active infection.

Asymptomatic bacteriuria is associated with a number of adverse pregnancy outcome. These include preterm births, perinatal mortality, anaemia and low birth weight infants (5). Thus there is need for routine screening and aggressive treatment of asymptomatic bacteriuria.

The American College of Obstetric and Gynaeoclogy recommends urine culture during the first prenatal visit and repeat in the third trimester (6). In addition, dip stick urinalysis should be done at every visit (2).

Other less expensive tests such as leukocyte esterase – nitrite dip stick were found to give variable results (7) and thus were not reliable.

Treatment of asymptomatic bacteriuria involves use of several antimicrobial regimes: selection may be on the in vitro susceptibilities but most often, is empirical.

The antibiotic should be safe for both mother and fetus. Ampicillin has historically been the drug of choice but studies have shown a 20-30% resistance of *E.coli* (8). Treatment with nitrofuratoin has proved effective in most women. Other regimens include amoxyxillin, cephalosporins. Sulfanomides may be used in the 1st and 2nd trimester but should be avoided in the 3rd trimester because the risk of kernicterus to the infant. Usual treatment is for 7 to 10 days but some authorities recommend single dose antimicrobial regimens.

Recurrence rate for all three regimens is 30% and these women may benefit from suppressive treatment in the remainder of the pregnancy (2).

In acute cystitis, there is dysuria, urgency and frequency with few systemic findings. Often there is pyuria as well as bacteriuria occasionally, there may be haematuria. 40% of patients with myelonephritis have preceding symptoms of lower tract infection (9). Some patients may have a sterile culture because of urethritis caused by chlamydia trachomatis but a majority will have cervicitis. Treatment involves antimicrobials fo 7 to

10 days, but recently there is a trend to use a 3 day course of therapy that has proved effective (2).

Acute pyelonephritis is a serious medical complication of pregnancy with an incidence of approximately 2%. It is more common after mid pregnancy often unilateral and on the right side in more than half of the cases (2). Diagnosis is made on the finding of fever, chills, flank pain with fever and the presence of bacteuria. There may or may not be signs of lower tract infection. 15% of patients with pyelonephritis have bacteriamia. It may be mistaken for labour, chorioamnionitis, appendicitis, abruptio placenta or puerperal sepsis. Most of the clinical findings are as a result of endotoxeamia. Some even develop respiratory insufficiency. Usually the patients are toxic and hospitalization is required.

Hydration to ensure adequate urinary out put is essential. The choice of intravenous antimicrobial therapy is usually empirical. Urine and blood cultures plus haemogram and urea and electrolytes should be obtained (2).

Normally, withint 24-48 hours most patients are symptomatic and are discharged when afebrile for 24 hours on antimicrobial treatment for 7-10 days.

Ninety five percent of women with pyelonephritis will be afebrile within 72 hours. If symptoms don't resolve within 48 to 72 hours, then re-evaluation should be done. None response may be due to urolithiasis, congenital abnormalities or peri-nephric abscess (10).

Investigations include renal ultrasound which may show pyelocalceal dilatations, urinary calculi, intrarenal or perinephric abscess.

Sometimes the scanner is not accurate and plain abdominal x-ray is required as 90% of renal stones are radio-opaque. Possible benefits outweigh fetal risk from radiation. If this is negative, the one shot pyelogram may be used.

The patient presented had acute pyelonephritis and was put on IV fluids and antibiotics with good recovery.

REFERENCES

1. Manoj K.B., Perloff D.: Cardiac, haematologic pulmonary, renal and urinary tract disorder in pregnancy In: Current obstetrics and Gynaecologic Diagnosis and Treatment. 8th edn edited by Decherney Alt ana M Pernoll Appleton and Lange 1994 chapter 22 p 428
2. Cunninghma F.G., Gant N.F., Levens K.J. et al. Renal and urinary tract disorders In: Williams Obstetrics 21edn Appleton and Lange 1997 chapter 47 p 1251.
3. Lucas M.J., Cunningham F.G.: Urinary tract infection in pregnancy. Clin Obstet Gynecol 1993 36:855-68.
4. McDowell D.R., Buchanan J.D., Fawley K.F., Gilbert G.C.: Anaerobic and other faslidious microorganism in asymptomatic bacteruria in pregnant women J. Infect Dis 1981:144:114-22
5. Harris R.E., Thomas V.L, Shelokov A. Asymptomatic bacteriuria in pregnancy: Antibody coated bacteria, renal function and intra uterine growth retardation Am. J. Obstet Gynecol 1976 126:20-5.
6. Antimicrobial therapy for obstetric patients A.C.O.G educational bulletin no 245. Washington Dc. American College of Obstetricians and Gynaecologists March 1998 245:8-10.
7. Bachman J.W., Heisse R.H., Naessen J.M., Timmerman M.G.: A study of various tests to detect asymptomatic urinary tract infections. In: An Obstetric Population JAMA 1993:270:1971-4
8. Sander C.C., Saner W.E.Jr.: Bete Lactam resistance in gram negative bocteria: Global trends and clinical impact. Clin Infect Dis 1992:15 824-39
9. Gilstrap L.C. III, Cunningham F.G., Whaley P.O.: Acute myelonephritis in pregnancy. A retrospective study. Obstet Gynecol 1981 57:409
10. Laughin K.R.: Management of urological problems during pregnancy: Urology 1994:44 159-69

CASE 8:

CERVICAL INCOMPETENCE - MACDONALD STITCH INSERTION

Name:	P.N.	L.M.P.:	4.3.00
Age:	30 years	E.D.D.:	11.12.00
IP. No.:	0816136	Para:	0+3
D.O.A.:	14.6.00	Gestation:	14 weeks
D.O.D.:	20.6.00		

PRESENTING COMPLAINT

P.N. was admitted from antenatal clinic with a diagnosis of cervical incompetence for insertion of MaDonald stitch.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She is a para 0+3 who had spontaneous abortions. First one was in 1999 at two and a half months gestation, she had two, one in the year 2000 and another in the year 2001 both and at six months gestation. The first abortion started with vaginal bleeding which was in clots this was associated with lower abdominal pains.

The other two abortions started with drainage of liquor followed by contractions and expulsion of the fetus. She was done evacuation of all the three abortions at a private hospital in Dagoretti.

Her last monthly period was on 4th of March and her gestation is 14 weeks. She attended antenatal clinic since 6 weeks gestation.

Menarche was at 14 years and her menses are regular, coming every twenty eight days and lasting 3 days. She has not used any form of contraception.

PAST MEDICAL HISTORY

She has only been to hospital because of the pregnancy losses.

FAMILY AND SOCIAL HISTORY

She is a housewife, the husband is a car salesman. They both do not smoke cigarettes and drink alcohol. There is no family history of diabetes, hypertension or chronic illness.

EXAMINATION

She was in good general condition, not pale, jaundiced or cyanosed. She had no oedema. Her blood pressure was 110/70, pulse rate 76/minute, respiratory rate 18/minute and temperature 36.2°C.

ABDOMINAL EXAMINATION

Abdomen soft with no organomegally. The uterus was 14 weeks.

VAGINAL EXAMINATION

She had normal external genitalia. The cervix was ½cm long, posterior with no defect. The uterus was 14 weeks and the adnexiae was normal.

DIAGNOSIS

An impression of cervical incompetence was made and she was for MaDonald stitch insertion.

Investigations

ELISA for HIV	-	negative
Haemoglobin	-	13g/dl
VDRL	-	negative
Blood group	-	B positive
Urea and electrolytes	-	Na^+ 136mmol/l
		K+ 3.84 mmol/l
		Creatinine 85mmol/l

DISCUSSION

The patient presented was a para 0+3 who had one 1st trimester abortion and two 2nd trimester abortions. Clinically, the cervix was short ½ cm long and was inserted the McDonald stitch at 14 weeks and delivered a life male infant at term after the stitch was removed.

P.N. presented with habitual abortion,which is defined as 3 or more consecutive pregnancy losses before 20 weeks gestation.There are several cuases of habitual abortions and are classified into;

1.Genetic-in 50% there is no knwn aetiological factor,in the remaining case the commonest cause istrisomy followed by monosomy.This normaly cause first trimester abortions.

2.Anatomical-this accounts for 33% of second trimester abortions,and they include congenital uterine anomalies,cervical incompetencs,sub-mucous fibroids and ashermans syndrome.

3.Hormonal-these account for 25% of habitual abortions and include thyroid dissorders, progesterone inssuficiency and diabetes.

4.Infections-these include toxoplasmosis, rubella, cytomegalovirus, syphylis,herpes,listeria,brucella and malaria.

5.Immunological

6.Systemic infections-this includes collagen diseases and diabetes.

Cervical incompetence was first described by Lash in 1950 and is defined as premature ripening of the cervix (1).

The incidence of cervical incompetence is only 1-2% of all pregnancies and the cause of 20-25% of 2nd trimester abortions (2).

Diagnosis of cervical incompetence is usually made on the woman's past medical history (3). Classically, the patient has one or more 2nd trimester or early third trimester pregnancy losses. Usually, they start with painless leaking of liquor or dilatation of the cervix which is later followed by contractions. This is because the cervix begins to dilate and efface before the pregnancy has reached term (3). Not all patients present with this classical history. P.N. presented with the classical history.

Diagnosis may also be found incidentally during perinatal ultrasound. Diagnosis of cervical incompetence before conception, though promising is not yet a good predictor. This involves testing the resistance of the cervix with a dilator or specialized instruments (3). Transvaginal ultrasound has shown promises in diagnosis: usually cervical length is 4cm. Length of less than 2.5cm have 50% risk of preterm labour. Other studies which apply trans-fundal pressure (cervical stress tests) at 15-24 weeks may show funneling (4).

Etiology of cervical incompetence include congenital or acquired. Congenital are due to DES exposure in utero or congenital mullenan ducts disorders (3). Acquired include Ebler Danles syndrome and surgical trauma which includes loss of connective tissue or damage to the structured integrity. Majority of the cases, the cause is idiopathic.

Treatment of cervical incompetence is surgical consisting of reinforcing the weak cervix (5). This is contraindicated in bleeding, ruptured membranes or if contractions are present (5). The stitch can either be placed vaginally or abdominally; the latter is used when the vaginal stitches fail and requires elective caeserian section. Vaginal stitch can either be Shirodkar or MacDonald (5).

Insertion of the stitch should be after 14 weeks to ensure abortion due to other factors have been competed (5).

The stitch is removed at 37 completed weeks. Success rates of preventing premature labour with MacDonald or modified Shirodka stitch are 85-90% (6). After they require bed rest and treatment for preterm labour.

Complications of ceclage include rupture of membranes, preterm labour, bleeding and infection (7).

Indications for removal of stitch includes labour, rupture of membranes and vaginal bleedings.

P.N. had successful insertion of MacDonald stitch with favourable outcome.

REFERENCES

1. Lash A.F., Lash S.R.: Habitual abortions: The incompetent internal os of cervix. Am J.Obste Gynecol 1950:59:86
2. Callen ultrasound in obstetrics and gynaecology Saunders 4th edition 576-599
3. Editorials NEJM 1996: Cervical incompetence and preterm delivery N. Engl. J. Med 334: 9 Feb 1996
4. Parulekar S.G., Kuni R.: Dynamic incompetent cervix uteric. Sonographic observations of ultrasound Med. 1988 pg 481
5. Cunningham F.G., Gant N.J., Leveno K.Y. et al. Abortion: In William Obstetrics 21st edition Appleton and Lange 1997: p 863
6. Caspi E., Schneider D.F., Mar Z. et al.: Cervica; internal os cerclage. Description of a new technique and comparison with Shirodkar operations. Am. J. Perinatol 1990:7:347
7. Rush R.W., Isaacs S., McPherson K., Jones L., Chalmers I. and Grant A.A.: A randomized control trial of cervical cordage in women at high risk of spontaneous preterm labour. Br J. Obstet Gynecol 1984:91:924-30

CASE 9.

ELECTIVE CAESERIAN SECTION DUE TO PREVIOUS CAESERIAN SECTION SCAR-INADEQUATE PELVIS

Name:	L.M	D.O.A: 9.4.02
Age:	35yrs	D.O.D:13.4.02
IP. No:	0801400	Para 1+0

PRESENTING COMPLAIN

Para 1+0 gravida 2 admitted through the antenatal clinic at 38 weeks gestation for elective caeserian section due to previous caeserian section scar with inadequate pelvis

HISTORY OF PRESENTING COMPLAIN

She had caeserian section in 1999 due to cephalo-pelvic disproportion. She had prolonged labour at KNH and an emergency caeserian was done. A life male infant weight 3.6kg was extracted who is alive and well

OBSTETRIC AND GYNAECOLOGICAL HISTORY

A Para 1+0 gravida 2. Her last monthly period was on 5.7.01 and her expected date of delivery was 23.4.01. She started attending antenatal clinic at 14 weeks gestation

-Antenatal profile was normal

HB-11.8g/dl

Blood group O-positive

H.I.V-negative

VDRL-negative

-An ultrasound examination done at 16weeks gestation confirmed fetal well being and Dates.

-Erect lateral pelvimetry done at 36 weeks gestation showed a true conjugate of 9.3cm.

PAST MEDICAL HISTORY

There was no significant history

FAMILY AND SOCIAL HISTORY

She is married since 2000 and works as a nurse. Her husband is a teacher by profession. Nor she or her husband smokes cigarettes or drinks alcohol. There is no family history of congenital disease or chronic illness

PHYSICAL EXAMINATION

She was in good general condition, not pale or jaundiced. She was afebrile with no oedema

Her blood pressure was130/70 mmHg and pulse rate 70/min

ABDOMINAL EXAMINATION

The fundal height was term with a single fetus in cephalic presentation and longitudinal lie. Fetal heart tones were heard and regular at 136/minute

Laboratory results

Haemoglobin-13.4g/dl

Urea and electrolytes

Na+-142mmol/l

K+-4.5mmol/l

She was counseled about the operation and consent form signed. Blod was grouped and x-matched. Pubic hair was shaved the day before the operation. Atropine 0.6mg IM was given 1/2hr before theatre

OPERATION

She was wheeled to theatre and put in semi lithotomy position .Vulvo vaginal toilet done and catheterization of 100mls of clear urine done. She was put in supine position cleaned and draped. General anaesthesia was induced. The abdomen was opened in 3layers after excision of the old scar. Lower uterine caeserian section was done and life female infant in occipital posterior position weighing 3800grams delivered who scored 7/1 and 9/5.The placenta was delivered by controlled cord traction. Uterus was cleaned and closed in 3 layers. The right fallopian tube was found adherent to the posterior uterine wall. abdomen was cleaned and closed in 3 layers after swab and instrument count found

correct. Haemostasis was achieved and VVT done. General anaesthesia was reversed uneventfully

POST OPERATIVELY

Vital signs were monitored 1/2hourly until she was fully awake then 4hourly. She was put on intra venous x-pen/gentamycin.She did well post operatively and on 2nd day was started on oral sips.Wound was exposed on third day and she was discharged home with the baby on the same day.

DISCUSSION

L.M. underwent elective caeserian section due to cephalo pelvic disproportion with previous scare to a life female infant weighing 3800 grams.

Caeserian section rates have continued to rise in the world. Incidence of caeserian section rate at Kenyatta National Hospital was 17.8% o which 59.8% were repeat sections (1) In 1989, the rate was found to be 21.1% (2). Prior caeserain section is the commonest indication for elective sections accounting for 53.3% of all sections (3). The caeserian section rates increased dramatically in the 1970s and 1980s; but reduced in the 1990s due to increase in the vaginal birth after caeserain section (4).

The closure of the uterine is conventionaly closed in 3 layers, current authors recommend closure in 1 or 2 layers. They base their argument on the basis that the peritonium reverts to its original position within 48hours and closure of the myometrium and endometrium in 2 layers causes strangulation of the blood supply hence impairing the healing process. There are some authorities who recommend closure of the peritonial layer to prevent adhehesion formation(2)

Successful trail of previous scar varies from place to place and rates vary from 60-80% (5). At Kenyatta National Hospital, the success rate was 73.9 (1).

Recommendations of the American College of Obstetricians and Gynaecologists (1999) for vaginal delivery after caeserian section include one or two prior low transverse incision, clinically adequate pelvis, no other uterine scars or previous rupture, physician available throughout labour and capable to perform an emergency caeserian, availability of anaesthesia and personal for emergency caeserian (6).

At Kenyatta National Hospital, trial of scar is not recommended for 2 previous caeserian section scars (3).

Women with transverse incision have lower risk of rupture but patient with classical or T incision were considered contraindications (4). Indications of prior caeserian section determine the success rate of trail of scar. If the first caeserian section was due to non-recurring complications like breech or fetal distress, then the success rate were high. If it was due to cephalo pelvic proportion or dystocia, then success is least likely (4). If a woman has had a prior vaginal delivery before or after the caeserian section then success rates are high (4).

The patient presented had a caeserian section due to cephalo-pelvic disproportion and has had no prior vaginal delivery.

In our centre, trial of scare is allowed if the following criteria is met:

- One previous lower uterine section incision
- Clinical adequate pelvis
- True conjugate of 10.5cm or more by pelvimetry
- No other obstetric complications
- Estimated fetal weight of 2500-3500grams

The patient presented did not meet all the conditions. Clinical pelvimetry is considered more superior to radiological pelvimetry as radiological pelvimetry is static and maternal focused. It does not assess changes that occur during labour. The best method to successful trial of labour is intrapartum monitoring in a well-equipped centre (7).

Use of oxytocin is not contraindicated but requires caution and close patient monitoring using intro uterine pressure monitoring (4). This is not practical at our centre.

Examination of the scar after delivery is currently not practised unless there is significant bleeding or pain (4).

When elective caeserian section is indicated, is imperative that lung maturity is confirmed prior to the caeserian section. In our centre, surfactant test is done. Other

methods include ultrasound examination or use of clinical follow up, if it is well documented (4).

REFERENCES

1. Karanja J.K.: A review of caeserian delivery at Kenyatta National Hospital in 1980. M.Med Thesis, University of Nairobi 1982.
2. Bodaya C., Bartha J.L., Rodrique I. et al: Trial of scar labour after caeserian section in patients with or without prior vaginal deliveries. Int. J. Obstet Gynecol 1992: 38:285-289
3. Walton S.M.: The antenatal and intrapartum management of patients with previous caeserian section scars. Easr Afri. Med. J. 1978:33-47
4. Cunningham F.G., Gant N.F., Leveno K.J. et al. Caeserian delivery and post partum haemorrhage In: Williams Obstetrics 21st edn Appleton and Lange 2001 ch 23 pg 538-553
5. Ralph W.H.: Operative delivery In: Current Obstetric and Gynaecologic Diagnosis and Treatment. Decherney A.H., Pernoll M.L. ed. Appleton and Lange 1994:570-71
6. American College of Obstetricians and Gynaecologists. Vaginal birth after previous caeserian delivery practice bulletin No. 5 1999
7. Ogutu O.W.: A prospective study of x-ray pelvimetry and trial of scar and mode of delivery at Kenyatta National Hospital. M.Med Thesis, University of Nairobi 1985

CASE 10:

PRE-ECLAMPSIA – EMERGENCY CAESERIAN SECTION – LIVE BIRTH

Name:	H.W.	Age:	22 years
Parity:	0+0	L.M.P.:	30.11.01
D.O.A.:	3.9.02	D.O.D.:	7.9.02
E.D.D.:	23.8.02	GBD:	37 weeks

PRESENTING COMPLAIN

H.W. came from home with complains of reduced fetal movements.

HISTORY OF PRESENTING ILLNESS

She was well until today when she noticed the baby was not playing well. She had been attending antenatal clinic at Kenyatta National Hospital since 4 months. At 8 months, she started having swelling of the feet and blood pressure was found to be 150/100.

She was started on aldomet 500g TID, junior aspirin once a day and phenobarbitone 30mg twice a day. Her blood pressure remained at 140/80 – 140/90 during the other visit, with proteinuria +.

ANTENATAL PROFILE

Blood pressure – O+ve

HIV – negative

Hb – 12.4g/dl

VDRL – negative

Scan done at 32 weeks showed normal fetus at 23 weeks gestation with no abnormalities seen.

GYNAECOLOGICAL HISTORY

Menarche was at 14 years. Menses were regular coming every 30 days and lasting 4 days. There was no history of contraceptive use.

PAST MEDICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY

She was married and a secretary by profession. She does not smoke or drink alcohol. Her husband is a businessman. There is no family history of chronic illness.

EXAMINATION

She was in food general condition, not pale, not jaundiced. She had bilateral pedal oedema and facial oedema. Blood pressure was 140/90mmHg, pulse rate 82/minute.

ABDOMINAL EXAMINATION

The abdomen was distended, fundal height was term, cephalic presentation, fetal heart was heard and was irregular at 148 per minute. There was no organomegaly.

VAGINAL EXAMINATION

She had normal external genitalia, the cervix was 2cm long, posterior, firm and cervical os was closed.

Urinalysis-protein +++

DIAGNOSIS

A decision to deliver her by emergency caeserian section was made. Informed written consent was obtained. She was shaved pubic hair and IV line secured and wheeled to the operating theatre.

In theatre, the abdomen was cleaned and draped. General anaesthesia was induced and abdomen opened via Pfannestein incision. A life female infant weighing 2550grams and scored 6/1, 9/6 was extracted and was taken o the New Born Unit. The placenta and cord were found grossly normal.

The uterus was cleaned and closed in 3 layers. The abdomen was cleaned and closed in 3 layers. General anaesthesia was reversed uneventfully.

POST OPERATIVELY

She did well, her urine put was good and by the second day the blood pressure was 130/80 mmHg. The baby was taken to the mother on the 3rd day. Both mother and baby were discharged on the 4th day after the mother's wound was exposed and found clean and dry.

She was put on antibiotics for review in the post natal clinic after 6 weeks.

FOLLOW UP

She was seen after six weeks and the wound had healed. Blood pressure was 120/80mmHg. She was referred to family welfare clinic for family planning.

DISCUSSION

H.W. presented above came with severe pre-eclampsia and emergency caeserian section was done with favourable outcome.

Pre-eclampsia is one of the hypertensive diseases of pregnancy, others are gestational hypertension, eclampsia, pre-eclampsia, super imposed on chronic hypertension and hypertension.

Hypertensive disease, haemorrhage and infection form the deadly triad that results in most of the maternal mortality and morbidity related to pregnancy.

Incidence of HDP (hypertensive disease of pregnancy) is 5-8% in the United States (1). In Kenya, the incidence varies from 1.5-9% (2) and that of pre-eclampsia to be 3.7% (3) and 5.4% (4).

H.D.P. is the third after haemorrhage and sepsis as a cause of maternal morbidity and mortality (5).

Gestational hypertension is diagnosed as hypertension during pregnancy without proteinuria that goes back to normal within 12 week post partum (1). Hypertension is diagnosed as blood pressure as 140/90 mmHg or greater using krotakoff phase V to define diastolic pressure.

Chronic hypertension is hypertension diagnosed before pregnancy or before 20 weeks or persistent after 12 weeks post partum.

Pre-eclampsia is blood pressure >140/90 mmHg plus proteinuria of >30 mg/liter and eclampsia that can not be attributed to any other causes (1).

Pre-eclampsia is further divided into severe and mild although mild disease may rapidly progress to severe. Severe pre-eclampsia is characterized by blood pressure of >

160/110mmHg, proteinuria of 2g, serum creatinine of > 12mg/dl, thrombocytopeania (platelets < 100,000/mm^3) haemolysis, fetal growth restriction, elevated liver enzyme, convulsions, headaches, upper abdominal pains and visual disturbances (1).

Etiology of pregnancy induced hypertension remains unknown. Predisposing factors include nulliparity, family history, diabetes, multiple gestation, extremities of age, preexisting hypertension and molar pregnancies (6).

The patient presented was nulliparous with no other predisposing factors.

Several theories have been proposed to explain the pathophysiologic basis. This includes immunologic therapy which is supported by the fact that HDP is common in nulliparous women, nulliparous with new spouse, immuno-compromised women (7).

Other possibilities include genetic predisposition, increased pressor response, endothelins, nitric oxide and endothelial cell activation.

Abnormalities of plancetation lead to failure of the convertion of the musculo-elastic layer of the spiral vessels to a fibrous dilated vessels (7). Pathological effects of H.D.P. is presumably as a result of vasospasm and ischaemia. This can be maternal or fetal. Maternal effects include:-

- Brain – cerebral oedema, infacts with development of convulsions
- Cardiovascular system - increased peripheral resistance with decreased cardiac output and extravasation of fluid into the extrocellular compartment.
- Renal – reduced renal perfusion and glomerular filtration and renal failure may develop
- Liver – peri-portal haemorrhagic necrosis and even hepatic rupture
- Blood – thrombocytopaenia
- Eye – retinal artery vasospasm or even retinal detachment (1).

Fetal effects – this is due to reduced placenta perfusion due to vasospasm which may lead to growth restriction and even fetal demise. Occasionally, abruptio placenta may develop (1).

H.W. developed reduced fetal movement probably secondary to vasospasm and fetal distress. Immediate delivery was indicated.

Management of hypertensive disease is pregnancy is aimed at termination of pregnancy with least possible trauma to mother and fetus, birth of an infant who thrives and complete restoration of the health of the mother (8).

Delivery is accomplished if the pre-eclampsia is worsening or the fetus's health is in jeopardy.

Systemic investigation are taken and blood pressure recordings done regularly. Management is bed rest, blood pressure control and sedation (9).

Several methods have been used to prevent HDP which include calcium supplementation, low dose aspirin and antioxidants(vitamin E).

REFERENCES

1. Cunningham F.G., Gant N.F., Leveno K.J. et al. Hypertensive disorders in pregnancy In Williams Obstetrics 2nd edn Appleton and Lange 2001 p 568.
2. Mati J.K.G.: Study of pregnancy hypertension M.Med thesis University of Nairobi 1975.
3. Bansal V.P.: Pre-eclampsia and eclampsia: A profile of Pumwani Maternity Hospital East Afr. Med. J. 62 (10)76:1985
4. Kibani J.G.: Outcome of pregnancy in patients with hypertensive disease. M.Med thesis 1992.
5. Kirumbi L.W., Makhokha A.E., Mukolwe B.A., Kigondu S.C., Nicholas D.D. Causes and prevention of maternal mortality in three districts in Kenya, Kilifi, Kirinyanga, Kakamega. J. of Obstet Gynecol East and Cent. Africa 1998 vol. 14 No. 2:72-77.
6. Chesley L.C.: Epidemiology of pre-eclampsia – eclampsia In: Hypertension disorders in pregnancy, New York Appleton – Century Croffts 1978 p 17.
7. Walter J.J., Dekker G.A. The aetiology and pathophysiology of hypertension in pregnancy. In Walter J.J., Gant N.J. (ed) Hypertension in pregnancy London Chapman and Hall Medical 1977 p 39.
8. American College of Obstetricians and Gynaecologists. Hypertension in pregnancy Technical Bulletin no. 219 January 1996.
9. Sibai B.M.: Treatment of hypertension in pregnant women. N. Eng. J. Med. 335:257 1996.

CASE 11

POST TERM – SUCCESSFUL INDUCTION OF LABOUR

Name:	A.W.	L.M.P.:	15.8.02
Age:	26 years	E.D.D.:	12.5.02
Parity:	1+0	D.O.A.:	10.6.02
IP. No.:	0741135	D.O.D.:	12.6.02

PRESENTING ILLNESS

A.W. was admitted from the antenatal clinic with post datism for induction of labour.

HISTORY OF PRESENTING ILLNESS

She was a para 1+0 who has been attending antenatal clinic since 5 months gestation, which was uneventful. Her blood group was O positive. Her HIV status was negative, haemoglobin was 12.6g/dl and urinalysis was normal through out. Her blood pressure was also normal. She confirmed her pregnancy at her first antenatal visit. Quickening was at 4 months. Her dates corresponded to her fundal height during the antenatal period.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She is a para 1+0. Her last delivery was in 1998 by spontaneous vertex delivery at 40 weeks to a life female infant who weighed 3.6kg.

She used copper 'T' since 1999 to 2000. Her menses were regular coming every 30 days and lasting 4 days. Menarche was at 15 years.

PAST MEDICAL HISTORY

This was not significant

FAMILY AND SOCIAL HISTORY

She is a teacher by profession, married to a nurse. She does not smoke or drink alcohol. There is no family history of chronic illness like diabetes or hypertension.

EXAMINATION

A young lady in fair general condition. She was not pale nor jaundiced and there was no oedema. Blood pressure was 120/70mmHg, pulse rate was 76/minute and temperature was 36.7°C.

ABDOMINAL EXAMINATION

Fundal height was term with single fetus in longitudinal lie and cephalic presentation. Head was 5/5 up. Fetal heart was heard and regular at 138/minute.

VAGINAL EXAMINATION

There was normal external. The cervix was posterior, 1cm long and firm. The cervical os was closed.

INVESTIGATIONS

Haemoglobin – 12.6g/dl

Blood group – B positive

VDRL – negative

HIV – negative

Obstetric scan on 4/6/02

Life fetus in cephalic presentation at term gestation. Liquor adequate, placenta fundal posterior,

Biophysical profile 8/8

Amniocentesis for surfacant test – positive

DIAGNOSIS

Para 1+0 with postdatism

MANAGEMENT

She was admitted for induction of labour. The cervix was unfavourable and cervical ripening was commenced by insertion of prostaglandin E_2 pessary 300mg which was inserted in the posterior fornix. A repeat pessary was inserted after 8 hours.

After 4 hours, she was noted to be contracting. Vaginal examination found the cervix to be 4cm dilated. Artificial rupture of membranes was done with drainage of clear liquor and augmentation in syntocinon in 5% dextrose commenced.

She was for partogram.

She progressed well and had a spontaneous vertex delivery to a live male infant who weighed 3600g, Apgar score 9/1, 10/5.

Placenta was delivered which weighed 600 grams. The infant showed wrinkling of skin and reduced subcutaneous fat and was taken to New Born Unit for observation.

POST NATAL CLINIC

Both mother and child did well and were discharged on the 2nd post natal day to attend post natal clinic on the 6th week at the nearest health centre.

DISCUSSION

A.W. presented above had post term pregnancy and had successful induction of labour.

Post-term pregnancy or prolonged pregnancy is a pregnancy that has completed 42 weeks or more from the first day of the last menstrual period (1).

Post mature or dysmaturity is when the fetus shows features of pathological impairment of growth which include weight loss, reduced subcutaneous tissue wrinkled, patchy peeling skin, unusually alert opened eye with typically long nails.

This can be classified into 3 stages (2)

Stage 1 - clear amniotic fluid

Stage 2 - skin stained green

Stage 3 - yellow-green skin discolouration.

Incidence of post-term pregnancy is 4 to 14% (3). Incidence of post-term pregnancy increases to 27% if there is a previous history and to 39% if there are two previous histories. There is also reports of post datism occurring across generations (3).

Fetus with anecepahly and placental sulfatose deficiency are often associated with prolonged pregnancy (4). Post term pregnancies are associated with maternal and fetal risks. Maternal risk include dysfunctional labour, cephalopelvic disproportion and shoulder dystocia (5).

Fetal risk are related to placental insufficiency oligohydramnios or extraordinary fetal size (5).

Diagnosis of post-term pregnancies should start by confirmation of gestational age using earlier records, pregnancy test, LNMP, quickening and earlier examinations including fundal measurements.

The highest risk of ?post-datism is the delivery of a premature infant.This can be prevented by amniocentesis for amniotic fluid surfactant test to confirm lung maturity(5)

Diagnosis is confirmed by ultrasound which frequently show oligohydramnios (6).

Management of post term pregnancy is aimed at reducing risk. Many authorities will not allow gestation to go beyond 41 weeks and nearly all agree not to go beyond 42½ weeks (1). If pregnancy is allowed to go on monitoring of pregnancies should be done using none stress test and maternal fetal count (6).

Delivery is indicated if there is fetal compromise e.g. oligohydramnios or fetal distress or if 42 week gestation is reached (6).

If the cervix is unfavourable, the ripening of the cervix is done followed by induction. A.W. underwent successful ripening and induction of labour.

REFERENCES

1. Pernoll M.L.: Late pregnancy complication. In: Current Obstetrics and Gynaecologic Diagnosis and Treatment eighth edn. Decherney A.H., Pernol M.L. eds. Appleton and Lange Ch. 15 p. 338-39 1994
2. Clifford S.H.: Post maturity with placental dysfunction. Clinical syndromes and pathologic findings. J. Paediatr. 44:1 1954.
3. Bakkterg L.S., Bergsjo P.: Post term pregnancy, magnitude of the problems. In: Chalmers I. enkin M., Kierse M. (eds). Effective care in pregnancy and childbirth. Oxford: Oxford University Press 199 p 765.
4. Eden R.D.: Perinatal characteristics of uncomplicated post date pregnancies. Obtel Gynecol: 1987 69:296.
5. Calkins L.A.: Post maturity. Am. J. Obstet Gynecol 1987: 56:167.
6. Cunningham F.G., Gant N.F., Leveno K.J. et al (eds) Post term pregnancy In Williams Obstetrics 2001:p 729-741.

CASE 12.

DEEP VENOUS THROMBOSIS IN PREGNANCY – LIFE BIRTH

Name:	A.A.	Age:	21 years
Sex:	Female	Parity:	0+0
D.O.A.:	6.8.02	D.O.D.:	18.10.02
WARD:	GFA		

PRESENTING COMPLAIN

She was referred form Pumwani Maternity Hospital with complains of painful swelling of the right leg for 1 week.

HISTORY OF PRESENTING COMPLAIN

She was well until one week prior to admission when she developed swelling of the left leg which began in the thigh region then extended to the calf by evening.

It was associated with pain which increased progressively and was unbearable by the third day. The pain was non-radiating and worsened by walking. She then sought help from Pumwani Maternity Hospital and was started on heparin 5000 IU 8 hourly without improvement and was referred to Kenyatta National Hospital due to lack of laboratory back up. She had no history of trauma nor chest pain.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She is a para 0+0. Her L.M.P was on 19.1.02 and her E.D.D. was 26.10.02, gestation by dates was 29+ weeks.

She attended ante-natal clinic at Umoja. There was no antenatal profile done. Her menarche was at 14 years, cycles were regular lasting 3 days and coming after 28 days. There was no history of contraception use.

PAST MEDICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY

She is a married housewife who does not smoke cigarettes or drink alcohol. Her husband drinks occasionally. There is no family history o chronic illness.

EXAMINATION

She was in fair general condition, not pale, clinically afebrile. Her blood pressure was 120/80mmHg, pulse rate 76/minute.

RESPIRATORY SYSTEM

Not in distress with normal breath sounds.

ABDOMINAL EXAMINATION

Fundal height was 30 weeks, longitudinal lie, cephalic presentation. Fetal heart was heard and regular at 148/minute.

MUSCULO SKELETAL SYSTEM

Left lower limb was swollen at the thigh and calf with increased local temperature. It was shiny and tender.

A diagnosis of deep venous thrombosis in pregnancy at 29+/40 gestation was made.

MANAGEMENT.

She was started on heparin infusion of 8000IU 8 hourly and the lower limb was elevated using pillows.

Investigation

Blood group A +ve

VDRL – negative

HIV – negative

Blood count - WBC – $4.3x10^9/l$

- Hb 9.8 g/dl
- HCT 29.5g/dl
- Platelets $325x10^9/l$

Doppler ultrasound showed no flow through the left femoral and poplitiel veins with fresh hypoechoic masses on left femoral veins.

Baseline coagulation screen was done on 8.8.02

APTT test - 37 seconds

APTT control - 33 seconds

Prothrombin time - test 15 seconds

- control 14 seconds

Prothrombin time index 93%. Subsequent follow up coagulation screens showed an increased INR to between 1.5-2.0 baseline.

FOLLOW UP.

She was put on sc heparinand after one week and then changed to wafarin . Both were given for 3 days. Measurements of both thigh and calf circumferences were done which showed a reduction in the left leg circumference.

At 36 weeks she was converted back to heparin.

She subsequently went into spontaneous labour on 12/10/02 at 38 weeks gestation. Heparin was withheld and Protamine sulfate injection kept at standby. She was grouped and cross matched. She underwent an uneventful labour and had a spontaneous vertex

delivery to a life male infant weighing 3400 grams and scored 8/1, 9/5. She did not develop post partum haemorrhage.

She continued on heparin and converted to warfarin 10mg daily for 3 months and for follow up in the medical outpatient clinic.

She was adviced not to use oral contraceptive and was put on copper "T" after 6 weeks.

DISCUSSION

A.A. presented with deep various thrombosis (DVT) of the left leg at 28 weeks gestation. She was managed on heparin and warrfarin. She went into spontaneous labour at 38 weeks. DVT and its sequelae, pulmonary embolism is a major cause of maternal morbidity and mortality (1).

Incidence of DVT in pregnancy is 1-5/1000 pregnancies, this increase in women with previous DVT to 12-35% (2).

Virchow's trial of damage to vessel wall, reduced blood flow and increase in blood coagulability is the pathophysiology of DVT. All these factors increase in pregnancy levels of procoagulant proteins such as factor VII, VIII and fibrinogen increase in pregnancy. There is also decrease in natural anticoagulants: There is also a decrease in antithrombosis III and protein S in pregnancy (3).

Other risk factors of thrombosis include age (>35 years), smoking, cancer, surgery, fractures, immobilization and oral contraceptives. Inherited thromphilias give an increased tendency to thrombosis this include defeaciencies of antithrombin, protein S., protein C. factor V leiden mutation (3).

Almost 90% of DVT in pregnancy are on the left side as compared to 55% in those not pregnant.

This may be due to compression of the left iliac vein by the ovarian arteries (4). Clinical presentation of DVT varies greatly depending on the site, intensity of thrombosis and inflammatory response.

In pregnancy, 72% are illiofemoral as compared to calf vein thrombosis in 9%. Classically, puerperal thrombophlebitis or phlegmonia alba dolens of the lower extremity is abrupt in onset, with severe pain and oedema (3).

Diagnosis of DVT include contrast venography, biochemical assays and biochemical assyas: venography is the gold standard but it is expensive, time consuming and cumbersone that it has been replaced by non-invasive methods.

Real time ultrasound along with duplex and colour Doppler ultrasound is currently the procedure of choice (5).

Other investigations that can be used include magnetic resonance imaging, computed tomographic scanning and impendence plethysmography.

Pulmonary embolism is the major most dangerous complication of DVT. DVT precedes clinical DVT in only half of the cases, the remaining half are usually asymptomatic until pulmonary pulmonary embolism (PE) has occurred.

Clinical evidence of PE include dyspnoea, chest pains, cough, haemophysis and findings include tachypnoea, dyspneoa, pleuric pain.

Investigations for PE include CXR, and ventilat6tion – perfusion scintigraphy, pulmonary angiogram is the fold standard for diagnosis (6).

Treatment of DVT is intially the line of heparin, bed rest and analgesia. Heparin was started as an intravenous bolus of 5000 unit followed by an infusion of 1400 units/hour until the patient is stable then converted to subcutaneous heparin to maintain an APTT 2-2½ times normal.

Warfrarin was associated with congenital malformation and should be avoided in the 1st 12 weeks and after 36 weeks. Low molecular weight heparin (LMWH) is now being used in pregnancy and has been found to be safe and effective especially for home therapy.

In case of LMWH, control is determined by measuring LMWH levels in blood 6 hours after injection (7).

If Warfarin is used after delivery, it should be given with heparin for 3 days and an INR of 2-3 times. Major side effects of heparin are osteoporosis, thrombocytopaenia and haemorrhage.

During labour, the heparin should be stopped and is started after delivery if there is no bleeding post partum within several hours. Protamine sulfate is the anti-dote for heparin with 1mg being given per 100units.

Veno-caval filters like the green filed and Gunther tulip filters may be placed to avoid pulmonary embolism (3).

In the pueperium, Warfarin should be used for six weeks or six months since DVT occurred.

Estrogen containing contraceptives should be avoided (8).

REFERENCES

1. Maternal and neonatal haemostasis task guidelines on the prevention, investigation and management of thrombosis associated with pregnancy. J. Clin Pathol 1993: 46:489-496.
2. Rutherford S., Montora M, McGehee W., Strong T. Thromboembolic disease associated with pregnancy: In 11 year review. Am. J. Obstet Gynecol 1991:164 Supp 286
3. Antonie L.W.A., Pradon's P., Martin H.P., Buller H.P. Deep vein thrombosis Lancet 1999:353:479-85
4. Geer I.A.: Thombosis in pregnancy: Maternal ana fetal issues Lancet 1999:353 1258-1264.
5. Polak J.F., Wilkinson D.L.: Ultrasonographic diagnosis of symptomatic deep venous thrombosis in pregnancy. Am. J. Obstet Gynecol 1991:165:625-629.
6. PIOPED investigators: Value of the ventilation/perfusion scan in acute pulmonary embolism JAMA 1990:263:2753-275.
7. Ginsberg J.S., Hirsh J.: Use of antithrombolic agents during pregnancy. Chest 1992 102 Suppl 385-390.
8. Kierkegaard A.: Incidence ano diagnosis of deep vein thrombosis associated with pregnancy Acta Obstet Gynecol Scnd 1983:62:639-43

CASE 13.

TWIN GESTATION- FAVOURABLE OUTCOME AFTER MISSED DIGNOSIS AND CASERIAN SECTION TO BREECH 1ST TWIN WITH TWIN TO TWIN TRANSFUSION

NAME:	A.N	AGE	24YEARS
IP NO	0806242	DOA	6.05.2002
DOD	13.5 .2002	WD	1A

PRESENTING COMPLAINT

Patient was referred from a clinic in Kawangware with labour pains for one day and breech presentation

HISTORY OF PRESENTING COMPLAINT

She was well prior to on set of lower abdominal pains which were progressive and increasing in intensity. She then went to the clinic in Kawangware were artificial rupture of the membrane was done of clear. Breech presentation was found and she was refereed to KNH

OBSTRETRIES AND GYNAECOLOGICAL HISTORY

LMP 6.7.01 EDD 13.4.02 She was at a gestation of 42 weeks

She had her menarche at 14 years.

Her cycles were regular lasting 4 days and coming every 30 days, she had not used any *method of contraception. She is a para 2 gravida 3 Her 1st delivery in 1993 was a S.V.D* to a male infant weighing 3.1 kg who is alive and well.

2nd delivery in 1997 was a S.V.D to a female infant weighing 2.9 kg who died of pueumonia at 1 and half years of age.

Antenatal Clinic

She had attended antenatal clinic in Kawangware since 12 week gestation.

Blood Group B+ve

HB	12.3gld
UDRC	negative

She had received two doses of tetanus toxoid. No diagnosis of twins was made. She made a total of 6 visits

FAMILY AND SOCIAL HISTORY

She is a married house wife, Husband is a teacher, neither smoke cigarettes or drink alcohol. There is no family history of twins or chronic illness.

PAST MEDICAL HISTORY

This is not significant

PHYSICAL EXAMINATION

She was a young lady in good general condition ,afebrile ,no pallar or jaudice
Pulse rate was 94/min,BP 120/80,Respiratory rate of 18 per min.

ABDOMINAL EXAMINATION

Fundal height was term. The abdomen was tense and distended. Presentation was breech ,Fetal heart was heard and regular at 145/min.

VAGINAL EXAMINATION

There was normal external genitalia. The cervix was fully dilated and effased fully Breech presentation with meconium stained liquor and the station was 2/5 down

DIAGNOSIS

Para 2 to gravida 3 with Breech presentation and big baby for delivery by caeserian section

MANAGEMENT

She was informed about the diagnosis and mode of management and informed consent obtained, blood was taken for grouping and crossmatching , the pubic hair was shaved and premedication of 1M atropine 0.6 mg ½ hour before theatre given.

At theater the abdomen was cleared and draped and opened in three layers after general anaesthesic was induced .Twin gestation was found intro operatively.1st twin was breech life male infant weighing 3600g who scored 6/1 7/5 2nd twin was also breech ,life male infant weighing 2000g who scored 6/1 7/5 and was taken to new born unit due to small for gestation age.

There was a single placenta with two amniotic sacs. Uterus was cleaned and closed in three layers after swab and instrument count was found okay. Heamostasis was achieved.

POST OPERATIVELY

The mother did well post operatively and was discharged on the 5th post operatively day to be seen in the clinic in 6 weeks .

The second twin did well and was discharged after a week.

DISCUSSION

A.M presented above had twin gestation which was not diagnosed until caeserian section was done .The out come was favourable, both twins and the mother were discharged well.

Twin gestation occur most commonly after fertilization of two ova this results in fraternal or dizyogotic twins. About one third of twins is due to embryonic splitting.
If splitting is before day three two fetus with separate placentas and amniotic cavities.
If splitting occurs between day 4 and 7 a single placenta with separate amniotic cavities and after 8 days the monochorionic – monoamniotic twining will occur. After day 12 conjoint twins will form (1).

Incidence of twin pregnancy varies geographyically. Monozygolic twining occur at the rate of 2.3-4 per 1000 pregnancis and the rate is constant not influenced by age,heredity or other factors.Dizygotic twin is influced by hereditary age of the mother,race,panty,use of fertility drug (2). The incidence of twining at Kenyatta National Hospital was found to 1:58:8 deliveris by Oyieke (3) and 1:46 deliveries by Mutungi (4). The gestation is associated with increased pregnancy related complication which include fetal abnormalities,spontaneous abortions,hyperemesis,aneamia,polyhydramnious pregnancy induced hypertension,prematurity, premature rapture of membranes, twin to twin transfusion and post partum heamorrhoge (2).

The patient presented had twin to twin transfusion.
Early disgnosis of twin pregnancy is essential to avoid complication .This is made from history of twining increased weight gain ,fundal height and multiple fetal parts.
Examinaton may be reveal a uterus larger than date multiple fetal parts and more than two fetal parts.

Cardiac asscultation may find two fetal hearts in two different areas with heart rate differing by 10 beats.

Diagnous of twin gestation before labour is mode in on 30-50 % Oyeke found that in 38 % diagnosis was made in labour or after delivery of 1st twin (3). Ultrasonography alfa fetoprotein and HPL may aid diagnosis. Once diagnosis is made routine antenatal follow up with monitoring for any complication should be done .Iron folic supplementations should be given.

Complication of twin pregnancy include

- Prematurity-This can be prevented by bed rest,tocolylic therapy and use of coarticosteroids for lung maturity.No significant reduction in preterm deliveries has been shown with the use of cerclage
- Malformation-There are increased congenital malformations in multiple pregnancies.These may be detected early by use of ultrasound
- Intrauterie fetal death of one or more fetus,this can be recognized early by scanning
- Conjoined tiwins,accordiac twin and twin to twin transfusion
- Maternal complications include pre-eclampsia, placenta previa polyhydramnios, aneamia and complication of delivery.
- There is need for early diagnosis of multiple pregnancy and monitoring of complication(6)

Twin to twin transfusion occurs in dizygotic monochononic twins with aterio-venous anastomosis which are not balanced. This results in the donor twin having aneamia ,intra uterine growth retardation and may develop hydrops ard heart failure.

The receipient is phlethoric, hypetensive, oedematos with hepatomegally,ascities and polyhydromnios. If the transfusion is severe there is high risk of prêterm delivery and even intrauterine death of the donor twin. Diagnous involves routine ultrasound scanning.

Treatment involves amniocentesis to draining the polyhydraminous and recently ablation of the communication vessels by neodium: YAG laser guided by fetloscopy (5)

The second twin normally has higher rate of complications from delivery and labour. Delivery is determined by presentation of first twin is cephalic then vaginal delivery is allowed. Any other presentation should be delivered by caesarian section (1)

At delivery the pediatrician together with the obstetrician should be present.The patient had postdatism which is rare in twin pregnancies as premature labour is a major complication.

REFERENCES

1. Cunnigham FG Gant N,Leveno k J et al Multiple Pregnancy In Williams Obstretric 21st ed Applet on and Lange conneticut 1997 p 765
2. Benson RC Multiple pregnancy ,In Pernoll M.L (ED) current obstretric and gyneacologic diagnosis a treatment 8th edn Appletion and Lang conneticut pg 357 1994
3. Oyieke JBO at 21/2 year review of some aspects of twin deliveries at Kenyatta National Hospital mmed thesis University od Nairobi 1978
4. Mutungi AK A prospective study of twin deliveries at Kenyatta National Hospital and Pumwani Maternity Hospital antenatal care and delivery M.Med thesis University of Nairobi 1990
5. Hecherk,Plath H et Endoscopic laser surgery versus serial amniocentesis in treatment of severe twin-twin transfusion syndrome.Am J obster gynecol 1999 180 (3pt 1) 52-6

CASE 14

HIV IN PREGNANCY

Name:	A.E.	Ward:	GFA
IP No.:	0839100	Age:	22 years
DOA:	2/10/02	DOD:	8/10/02

PRESENTING ILLNESS

She is a para 1+0 admitted through antenatal clinic for elective caeserain section.

HISTORY OF PRESENTING ILLNESS

She was well until she had routine HIV testing at the clinic and was told she is positive. She started attending antenatal clinic at 28 weeks.

VDRL was negative, blood group O +ve and haemoglobin was 10.2g/d. She was adviced on prevention of mother to child transmission (PMCT) and was started on AZT at 34 weeks gestation and adviced on elective caeserian section. No CD4 or viral loads were taken

OBSTETRIC AND GYNAECLOGIC HISTORY

She is a para 1+0 last. Her last delivery was in 1998, home delivery to a life male infant who is alive and well. Her LMP was on 8.1.02 and her EDD 15.10.02. Gestation by dates was 38 weeks.

FAMILY AND SOCIAL HISTORY

A married housewife who stays with her husband in Nairobi. She does not smoke cigarettes nor drink alcohol. Her husband is a salesman who does not smoke but takes alcohol.

PAST MEDICAL HISTORY

This was not significant.

EXAMINATION

She was in fair general condition, not pale or jaundiced. She was clinically afebrile. Her blood pressure was 120/70mmHg, pulse was 74/minute and temperature was 37°C. There was no lymphadenopathy nor oral thrush.

ABDOMINAL EXAMINATION

Fundal height was term with cephalic presentation and longitudinal lie. Fetal heart was heard and regular at 136 per minute.

LABORATORY RESULTS.

Hb - 13.3g/dl

Urea - 2.7mmol/l

K^+ - 40mmol/l

Na^+ - 139mmol/l

She was informed about the operation and written consent obtained in the ward and was grouped and cross matched. In the morning before the operation she was premedicated with atropine 0.6mg ½ hour before theatre and zidovudine 300mg given stat. She was wheeled to theatre and an elective caeserian section done. The outcome was a life female infant who weighed 2.9kgs and scored 9/1, 10/5.

POST OPERATIVELY

She did well and was started on sips on the first post-operative day. On the second post operative day she was started on light diet and oral medication.

She was discharged on the fourth post operative day on oral antibiotics and analgesics.

FOLLOW UP

She was to given formula feed and not to breast feed. The baby was to have HIV test on review at the clinic in 3 weeks and a repeat at 6 months and one year.

DISCUSSION

A.E. is a 22 year old para 1+0 with HIV in pregnancy. She was given AZT from 34 weeks gestation and underwent an elective caeserain section at 38 weeks to prevent mother to child transmission (MTCT). She also was to avoid breast feeding.

The human immunodeficiency virus (HIV), an RNA retrovirus existing in two forms. HIV 1 is the commonest of the infections while HIV 2 is confined to certain parts of West Africa.

The sero prevalence of HIV among pregnant women in Africa exceeds 20% in many areas. In Kenya, HIV prevalence in urban sentinel sites in 1998 among pregnant women was reported to range between 4-10% in low sero prevalence areas to 20-35 in high sero prevalence sites: Heterosexual contacts account for 90% of all HIV infection, the remaining resulting from inoculation with infected blood/blood products, use of contaminated needles and vertical transmission from mother to child (1).

In Kenya in 1999, 10% of all reported AIDS cases in children were under 5 years of age and 90% of infection of children is due to MTCT (1). In Kenya, about 100,000 children are infected by HIV annually due to MTCT (1). Transmission can occur during pregnancy, labour, delivery and breast feeding. Overall, transmission rate is 30-45% with 10-20% in antenatal period, 35-50% during labour and delivery and 40-50% during breast feeding.

There are factors that are known to affect MTCT of HIV. Those are that have strong evidence that they affect transmission include high viral load, viral genotype and phenotype, advanced disease, HIV infection acquired during pregnancy or breast feeding period, vaginal delivery, rupture of membranes for more than 4 hours, prematurity and breast feeding. Those that have limited evidence include viral resistance, vitamin A deficiency, anaemia, sexually transmitted diseases, chorioamnionitis, frequent unprotected sexual intercourse, multiple sexual partners, smoking, injection, drug abuse,

invasive procedures, episiotomy, antepartum haemorrhage, external cephalic versus genetic and lesions of skin or mucous membranes (2).

Factors know to reduce MTCT are elective caeserian section, non breast feeding and anti retroviral therapy (2).

The care of HIV infected women during antenatal period, labour, delivery and post-partum period includes intensive counseling and voluntary testing (both pre and post), screening/obstetric intervention, laboratory investigations, treatment and prophylaxis, laboratory tests include HIV test, CD4 lymphocyte count, viral load, haemogram, urea and electrolytes and liver function tests (3).

There are several regimens used in the prevention of MTCT as listed below.

Anti-retroviral therapy

Drug	ANC	Labour	Baby
Zidovudine	100mg p.o. 5 times Daily from 14-34 Weeks gestation	iv 2mg/kg stat then 1mg/kg/hr	2mg/kg p.o 6 hourly x 6 weeks
Zidovudine	300mg p.o. once A day from 36-40 Weeks gestation	300mg p.o. hourly	Nil
Nevirapine	No	200mg as a stat Dose at the onset Of labour	2mg/kg single dose in the first 72 hours

In the Kenyan set up, the Thai regime is most advocated and it is started at 34 weeks as many of our patient deliver before the 40 weeks gestation. This results in an 50% reduction of transmission (2). A.E. was on the Thai regimen from 34 weeks.

During antenatal care, the patient should have regular antenatal visit but invasive procedures like amniocentesis, chanonic villous sampling should be avoided. External cephalic version should also be avoided.

The mothers should have nutritional support and supplementation should be done: weight loss is a poor prognostic sign.

Behaviour change should be encouraged and this involves discouragement of smoking, alcohol and drug abuse, avoiding having unprotected sex and breast feeding. Medical treatment for common pregnancy complications and also for HIV related complication. Prophylactic treatment for HIV women include iron and follate, multivitamin supplementation, tetanus toxoid, sulfadoxine-pyrimethamine for malaria, pneumocystis pneumonia prophylaxis with cotrimoxazole for women with

$CD4^+$ counts below 200 per mm^2 and INH prophylaxis if montoux test is reactive for one year (2).

During labour and delivery practices are modified, vaginal cleansing with hibitane reduces the transmission rates(current studies have shown that this may not be beneficial and this is no longer recommended), routine episiotomy should be avoided and routine rupture of membranes should be avoided as rupture of membranes for greater than 4 hours is associated with increased risk of transmission. Where possible, elective caeserian section should be done (7).

During delivery, episiotomy should be avoided and vaginal cleansing should be done. The baby's cord should be clamped immediately and mouth and eyes wiped. Suction should be avoided. Avoidance of breast feeding should be done (4). The baby should be washed with warm 0.24% chlorhexidine solution to remove maternal secretions.

A.E. opted not to breast feed and child was put on formulae diet.

The management of HIV in pregnancy is evolving following the trends in non-pregnant HIV persons. Because of the devastating consequences of the infection on the un-treated persons a shift has occurred from elusive focus on fetal protection to a more balanced approach to treatment of mother and fetus.

A combination of nucleoside analogs – Zidovudine, didanosir, zalatabine and lamivudine – given with either a protease inhibitor non-nucleoride analogue or another nudovideine analogue is shown to be effective (5).

REFERENCES

1. Guidelines to antiretroviral drug therapy in Kenya Ministry of Health: 2001 February (1).
2. Nduati R., John G., Mbori Ngacha D. et al (March 2000) effect of breast feeding and formula feeding on transmission of HIV 1: A randomized clinical trial JAMA vol 283: No. 9 pgs 1167-1174.
3. DeCook K.M., Fowler M.G., Mercier E. et al (March 1 2000). Prevention of mother to child HIV transmission in resource – poor countries: translating research into policy and practice JAMA vol. 283 No. 9 page 1175-1182.
4. UNAIDS (May 1999) Prevention of HIV transmission from mother to child: Strategic options.
5. Kass N.E., Talor H.A., Anderson J. Treatment of human immunodeficiency virus during pregnancy. The shift from an eclusive focus on fetal protection to a more balanced approach. Am. J. Obstet Gynecol 179:856, 2000.

CASE 15

MALARIA IN PREGNANCY

Name:	S.W.	Age:	25 years
IP. No.:	0845118	LMP:	23.01.02
DOA:	1.10.02	EDD:	30.10.02
DOD:	5.10.02	Parity:	0+1

PRESENTING COMPLAIN

S.W. came with complains of vomiting, headache, joint pains, fever and chills for the lat 3 days.

HISTORY OF PRESENTING COMPLAIN

She was well until 3 days ago when she developed the above symptoms. She was vomiting three times a day which was not projectile. She had no history of traveling outside Nairobi in the last 6 months.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

Para 0+1 at gestation of 36 weeks. Last pregnancy in 2001, missed abortion at 5 months. She attended antenatal clinic since 18 weeks at AAR clinic uneventfully. Haemoglobin was 11.3g/dl, blood group A+ve, VDRL negative. Her menarche was at 14 years. She did not have a history of contraceptive use.

PAST MEDICAL HISTORY

This was not significant

FAMILY AND SOCIAL HISTORY

She is a married housewife. She does not smoke cigarettes nor drink alcohol. There is no family history of chronic illness.

EXAMINATION

She was sick looking, clinically afebrile with mild dehydration. There was no pallor or jaundice. Her blood pressure was 110/70 mmHg, temperature 38.9°C and pulse rate 98/minute.

ABDOMINAL EXAMINATION

Fundal height was 36 weeks with a single fetus in cephalic presentation, longitudinal lie. Fetal heart tones were heard and regular at 138/minute. Other systems were essentially normal.

An impression of malaria in pregnancy at 36 weeks was made.

INVESTIGATIONS

Blood slide for malaria	-	moderate malarial parasites seen
Haemogram	-	Hb 8.1g/dl
		WBC $9.3x10^5$/ml
		Platelets $265x10^5$/ml
Urinalysis	-	normal

She was started on IM artenam and IV aspergic and paracetamol. She was also started on haematinics. Repeat blood slide on 4/11/02 showed no malaria parasites.

She was discharged on 6/11/02 on haematinics and weekly metakelfin till 6the week post partum and to continue antenatal clinic.

Her remaining antenatal period was uneventful and she went into spontaneous labour on 26/10/02 when she was 38 weeks.

EXAMINATION IN LABOUR WARD

She was in good general condition, not pale, clinically afebrile. Her blood pressure was 130/70mmHg and temperature 36.8°C.

ABDOMINAL EXAMINATION

Fundal height was term with single intrauterine fetus in cephalic presentation with fetal heart rate at 144/minute, regular. Contractions were good lasting 40 seconds and coming every 3 minutes.

VAGINAL EXAMINATION

There was a normal external genitalia. The head was down 3/5 cervix 7 cm dilated and fully effased. Artificial rupture of membranes was done with clear liquor. She was put on the partogram and subsequently delivered by spontaneous vertex delivery to a life male infant who weighed 3250grams and scored well.

She was discharged home on the 2nd post natal day on anitmalarial prophylaxis (metakelfin) for 6 weeks.

DISCUSSION

S.W. came with malaria in pregnancy with moderate anaemia. She was treated with artenam with good results. She delivered a life male infant who scored well and the mother and baby were both discharged from hospital well.

Malaria is caused by the protozoa of plasmodium species parasitizing the red blood cells and liver. The plasmodium species are transmitted by the female anopheles mosquito. There are four plasmodium species that infect man, these are: P. Falciparum, P. Ovale, P. Malariae and P. vivex (1).

Ninety eight percent of malaria in most of the country and Eastern and Southern Africa are caused by P. falciparum and this causes the most severe form of malaria (1,2).

The level of malaria endemically varies from region to region with the lake and coastal regions being hyperhaloendemic and highland areas of Mount Kenya and Aberdare Ranges being malaria free (1).

Malaria in pregnancy is common with a prevalence of 21.2% in the Coastal region (3) and 42% in Western Kenya (4).

Malaria may either be uncomplicated or severe and complicated. It is uncomplicated if there is not altered consciousness or parasitaemia of less than 5%. Severe malaria is characterized by diminished level of consciousness, prostraction, convulsion, severe anaemia (Hb < 5g/dl), renal failure, respiratory distress, parasitaemia of over 5%, black water fever and hypoglycaemia (1). S.W. had uncomplicated malaria.

An individual's immune status can determine if a person will develop malaria and the severity of the infection. Immunity is mounted through cellular and humoral factors. Via intermittent parasitaemia cellular immunity is the form of phagocytosis by macrophages and humoral immunity via production of specific antibiotics (5).

Individuals who live in endemic areas are usually less susceptible to infection unless their immunity is impaired as in pregnancy multiparity tends to offer protection against malaria but susceptibility is increased in the 1t pregnancy (1).

During pregnancy, there is an increased susceptibility of severe malaria like cerebral malaria, acute renal failure, hypoglycaemia, disseminated intravascular coagulation and acute pulmonary oedema. The pregnancy outcomes are usually poorer with higher incidences of abortions, preterm labour, low birth weight infants, still births and maternal mortality (1,5). This is usually due to immune suppression due to high cortisal levels and decreased cellular immunity. There is also sequestration of the parasites in the placenta and inhibition of transformation of lymphocytes into macrophages by the glycoproteins of pregnancy (6).

During pregnancy, placental parasitization results in reduced placental blood flow hence impaired fetal growth. The fetus us protected from malaria because Hb F (fetal haemoglobin) is more resistant to malaria parasites and the circulating maternal antibodies (2). Congenital malaria may occasionally develop in non-immune women (4).

Anaemia may result from haemolysis, bone marrow suppression, hypersplenism, folic acid deficiency or hyperferritinaemia. This may lead to fetal demise, post partum haemorrhage or congestive cardiac failure (5). S.W. had moderate anaemia.

Hyperpyrexia may lead to abortion or preterm labour due to uterine activation or it may lead to fetal demise (4).

Diagnosis of malaria is usually confirmed through laboratory investigation. Other causes of pyrexia like urinary tract infection, typhoid and meningitis should be ruled out. Blood smears (thicj and thin) usually confirms the presence of the parasite. Parasite F is used to detect the antigens (5).

Treatment of malaria is aimed at eradicating the parasite and providing supportive care. Hyperpyrexia, hypoglycaemia, renal failure and anaemia are corrected. Treatment of uncomplicated malaria involves the use of pyrimethamine-sulfonamides combinations as the first line. In case of resistance or severe malaria, the drug of choice is quinine for 7 to 10 days. Other drugs are the derivatives of quinto... such as artenam. S.W. received artenam.

After treatment, malaria chemoprophylaxis is important and options include amodiagquin, proquanil and sulfanamide – pyrimethomine combinations (2). Chloroquine is not used since the development of widespread resistance.

REFERRENCES

1. National guidelines for diagnosis, treatment and prevention of malaria for health workers. Ministry of Health, Kenya, 1997.
2. Nathwani D., Currie P. Plasmodium falciparum malaria in pregnancy. A review. Brit. J. Obstet. Gynaecol 99, 118, 1992.
3. Rukana R.M. In vivo and vitro response of plasmodium falciparum to chloroquine in pregnant women in Kilifi District, Kenya. M.Med thesis, University of Nairobi, 1990.
4. Stekelee R.W., Brandling B. In vivo response of P.falciparum to chloroquine in pregnant women in Siaya District. Bull WHO 65 (6) 889:1987.
5. White N.J., Breman J.G. Malaria and other red blood cell parasites in Fauci A.S., Braundwad E., Isselbacher K.J. et al (eds). Harrison's Principles of Internal Medicine 14th edn vol. 1, McGraw Hill Companies, Inc., New York 1998 p 1180.
6. McGregor I.A., Wilson M.E. Malaria infection of the placenta in the Gambia, West Africa: Its incidence, relationship to still birth, birth weight and placental weight. Trans R. Sec. Trop Med. Hyg 77(2) 232:1983.