CASE RECORDS AND COMMENTARIES

SUBMITTED BY

DR. WAMWANA EDMOND BARASA

FOR THE

DEGREE OF MASTER OF MEDICINE

IN OBSTETRICS AND GYANAECOLOGY

OF THE

UNIVERSITY OF NAIROBI (KENYA)

MAY 2004

CONTENTS

GYNAECOLOGY LONG COMMENTARY

DEDICATION

This work is dedicated to my late father Didymus L. Wamwana and to my mother Rosalia Wamwana who lovingly groomed me into who I am. My Wife Margaret Wanjiku and daughters Ann Nasumba and son Teddy Wamwana shall always remain my greatest inspiration.

ACKNOWLEDGEMENTS

The journey was long and distorted. The successful conclusion of this book is undoubtably attributed to the deligent guidance by my supervisors- Dr. Muia Ndavi and Dr. P. Gichangi.

The staff members at the Provincial General Hospital Kakamega were most helpful, in particular the department of Obs/Gyn and the records department for their selfless contribution in data collection. We bonded like a family and it was with a heavy heart when we finally had to part at the end of my rotation.

The Ministry Health, Population council facilitated my great exposure at the PGH Kakamega. Special thanks go to Dr. Muia Ndavi, Dr.J.G. Karanja and Dr. Jeldesa.

My brother John Wamwana and sister Benedine Makokha, thanks for being there when most needed.

Lastly I would like to thank my children for enduring turbulent times. I promise to make it up to you.

DECLARATION

This is to certify that the cases recorded and commentaries presented in this book are my Original work and have not been presented for a degree course in any other University.

I further certify that all the cases presented here were treated and operated by me under the supervision of the senior members of the Department of Obstetrics and Gynaecology at Kenyatta National Hospital, Nairobi.

SIGNED:.....

DATE:23/6/2004

DR. WAMWANA EDMOND BARASA, MD 1989

CERTIFICATE OF SUPERVISION

This is to certify that the long commentaries presented in this book were researched upon by Dr. Wamwana Edmond Barasa under our guidance and supervision and that this book is submitted with our approval.

DR. MUIA NDAVI MBCHB, M.MED, MSC EPID FHBR, DLSHTM
Senior lecturer,
Department of Obstetrics/Gynaecology,
College of Health Sciences,
University of Nairobi.

SIGNED:.....

DATE:23/6/04

DR. P. GICHANGI MBCHB, B.Sc. ANATOMY, M.MED, Ph.D.
Senior lecturer,
Department of Human Anatomy/Obstetrics and Gynaecology
College Of Health Sciences,
University of Nairobi.

SIGNED:.....

DATE:09/7/04

CERTIFICATION

This is to certify that Obstetric cases 1,2,3,4 and 5 and Gynaecology cases 6,7,8,9 and 10 were managed by Dr Wamwana Edmond Barasa under my guidance and supervision at Kenyatta National Hospital.

SIGNED:.....

DATE:.....

DR. OYIEKE J.B.O., MB.ChB., M.Med (obs/Gynae)

Senior lecturer,
Department of Obstetrics and Gynaecology,
University of Nairobi.

CERTIFICATION

This is to certify that Obstetric cases 6,7,8,9 and 10 and Gynaecology cases 11,12,13.14 and 15 were managed by Dr Wamwana Edmond Barasa under my guidance and supervision at Kenyatta National Hospital.

SIGNED:.....Wanjala.....

DATE:.....22/09/04.....

DR. WANJALA S.H.M., MB.ChB. MMed. (Obs/Gynae.)

Senior Lecturer and former Chairman
Department of Obstetrics and Gynaecology,
University of Nairobi.

CERTIFICATION

This is to certify that Obstetric cases 11, 12, 13,14and 15 and Gynaecology cases 1,2,3,4 and 5 were managed by Dr Wamwana Edmond Barasa under my guidance and supervision at Kenyatta National Hospital.

SIGNED:.....

DATE:.....

PROF.S.B.O. OJWANG, M. D. M. Med (Obs/Gynae), Dipl. Gyn.Oncology

Associate Professor and former Chairman,
Department of Obstetrics and Gynaecology,
University of Nairobi

INTRODUCTION

Kenyatta National Hospital (KNH) is the largest hospital in Kenya. It is situated about 5km from the Nairobi city center. KNH has a modern ten storey block that provides inpatient care for the various medical disciplines.

KNH serves as the National referral hospital and also caters for referrals from East and Central African countries for specialized treatment. It also serves as a teaching hospital for the College of Health Sciences, University of Nairobi, providing training facilities for undergraduate and postgraduate students in Medicine, Pharmacy and Dentistry. Nurses and all categories of paramedical personnel also train here.

KNH has been running as a parastatal since April, 1987 and has its own management board. The cases presented in this book were managed in the Obstetric and Gynaecology Unit of KNH between 2001 and 2003.

THE OBSTETRIC AND GYNAECOLOGY UNIT

This is one of the several divisions of the clinical services provided by the hospital. The unit consists of casualty, outpatient obstetric and gynaecology clinics and obstetric and gyanecology lying in wards.

i) CASUALTY DEPARTMENTS

After filtering all patients that present at the Kenyatta National Hospital, those with gynaecological or obstetric problems are seen by a medical officer attached to the department.

This doctor is under the supervision of the Senior House Officer on duty. All patients are reviewed, those with emergency problems are admitted, others are given consultations to be followed up in the specialized clinics while others are treated and discharged.

ii) OBSTETRIC UNIT

This department consists of an outpatient antenatal and postnatal clinic, three lying in wards, labour ward and two labour ward theatres. The services are run by doctors in different grades, from the intern, the registrar, the senior registrar and the consultants.

These are grouped into three "Firms" for the smooth running of the unit. Each of the firms runs one lying in ward and covers labour ward on weekly rotations. They also run a high risk antenatal clinic once a week. The houseman and the registrar are on duty 24 hours a day in the wards. The senior registrars conduct daily morning rounds while the consultants conduct a weekly major ward round and also attends the weekly high risk clinic. In addition they are consulted any time and respond when need arises.

a) Antenatal clinics

Selection for followup of high risk antenatal patients is done once every Monday morning by each of the firms alternately. The patients usually have consultations from either casualty, antenatal wards or city clinics. The one in charge of a booking clinic is a senior registrar. About 40 patients are booked every week. The antenatal clinic is run every Tuesday, Wednesday and Thursdays. In addition the adolescents are seen on every Monday afternoon.

A high risk approach to booking is followed. The criteria include:

- Bad obstetric history: Previous stillbirths, neonatal deaths, recurrent abortions among others.
- Patients with medical conditions complicating pregnancy
- Anaemia, diabetes mellitus, hypertension, cardiac disease, thyroid disease, deep venous thrombosis, renal disease, sickle cell anaemia, rhesus negative mothers among others.
- Primigravida, Short, teenage, elderly, paraplegic.
- Grandmultiparity:- para 5 and above
- Previous operative deliveries: Caesarean section, vacuum extraction, forceps
- Patients with previous obstetric complications, for example post partum haemorrhage, uterine rupture, vesico-recto-vaginal fistulae among others.
- Others – including multiple gestation, breech presentation and those with prolonged infertility.

After booking, a detailed history of the past obstetric performance, medical, social and family history is recorded. The patient's height, weight and blood pressure are recorded. Urine examination for protein, sugar and ketones is done.

Blood for haemoglobin level, blood group and rhesus factor, serology for syphilis and HIV antibodies is taken. Blood for special investigations like brucella antibodies, toxoplasmosis, T_3, T_4 levels, indirect Coomb's Test, oral glucose tolerance test (OGTT) and urea and electrolytes may be taken when indicated.

A complete physical examination is done on the day of the booking. Those who are not booked are advised to attend the various peripheral hospitals for their antenatal care.

b) **Antenatal Follow-up**

The gravidas are usually seen monthly upto 28 weeks gestation, 2 weekly upto 36 weeks then weekly till delivery. However, more frequent or less frequent reviews may be decided depending on the patients' condition and convenience.

During each visit, the patient is weighed to assess the rate of weight gain; particular attention is paid to the Blood Pressure and any proteinuria in urine and also edema. The patient is questioned regarding any symptoms. The abdomen is then prepared to assess fetal growth, presentation, any polyhydramnios; auscultation for fetal heart sounds is done.

The fetal heart can be heard with a fetoscope after 24 weeks, and the position and presentation can be determined with reasonable accuracy in normal women after 30 weeks.

All primigravidas, patients with previous caesarean section due to non-recurrent cause and those with breech presentation in whom vaginal delivery is contemplated must have a pelvic assessment at 36 weeks. An Erect Lateral Pelvimetry (ELP) is done when desired.

Other patients admitted to the antenatal ward from the clinic are those with medical complications such as severe anaemia, severe toxaemias, higher grades of cardiac disease who will require investigations and management.

Health education, emphasizing on personal hygiene, diet, breast care, labour, puerperal and baby care, are routinely given during the antenatal visits and in the lying wards by both nurses and the medical officers. Family planning and the various methods offered are also discussed.

i) LABORATORY AND RADIOLOGICAL BACKUP

The department has specialized laboratory services which supplement those offered by the hospital facilities. Tests which are carried out include the surfactant bubble test, pregnancy tests, Kleihauer-Betke test, HIV screening among others in obstetrics and gynaecological investigations such as semenalysis, radioimmunoassay for hormones, antisperm antibody tests.

The hospital's department of radiology offers ultrasonography examinations for pelvic masses, scans for pregnancy and estimation of fetal gestational age and number of fetuses and aids during amniocentesis. Erect lateral pelvimetry, intravenous urograms and hysterosalpingograms are also in the department.

ii) **LABOUR WARD**

This is managed as the acute obstetric unit. Patients admitted to labour ward are those in labour, those who are very ill during pregnancy or in the immediate postpartum period.

It is run by the "Firm" on call each week. The team is composed of nurses, midwives, intern, senior house officers, senior registrars and consultants.

The ward consists of 10 cubicles for patients in the first stage of labour and 4 delivery suites one of which is used as an intensive care room for the high risk patients such as those with severe toxaemia in pregnancy and eclampsia, cardiac disease in pregnancy, in heart failure or in labour, diabetes mellitus with complications like ketoacidosis, sickle cell anemia in crisis. This "acute room" handles both antenatal and those immediately postnatal who have complications.

a) **The Labour ward Theatres**

These are two in number. The main theatre is bigger and handles the routine emergency as well as elective surgery while the other is usually reserved for postpartum tubal sterilizations and emergency surgery when the need arises.The second one is currently under renovation and is due to be opened soon.

Most of the surgery carried out is caesarean sections, examination under anaesthesia and repair of cervical and vaginal tears and exploration of uterus. Elective insertion of Macdonalds' stitch for cervical incompetence is also done. Occasionally, caesarean subtotal and total hysterectomy are done for ruptured uterus.

iii) **LYING IN WARDS**

There are 3 lying in wards each with a capacity of 32 beds. Each of those wards has one acute room, 2 antenatal rooms, 2 postnatal rooms and 2 isolation rooms.

Obstetric procedures

The procedures described here are mentioned in various cases presented in this book. They are described here to avoid repetitions.

i) **Amniocentesis**

This procedure is used to obtain amniotic fluid mainly for surfactant test to assess fetal lung maturity, bilirubin spectrophotometry in rhesus isoimmunization, and alfa-feto protein for suspected neural tube defects. To assess fetal lung maturity this procedure is done usually after 37 completed weeks.

The patient is explained the purpose and nature of the procedure. She empties her urinary bladder and lies in supine position on a couch. The abdomen is palpated for fetal lie, presentation and engagement. The fetal heart is noted. The anterior abdominal wall is cleaned with antiseptic solution usually methylated spirit and draped with sterile linen. The surgeon, after scrubbing and putting gloves, displaces the presenting part upwards with the left hand. With a sterile large-bore needle attached to 10cc syringe the right hand is used to obtain 5-10mls of amniotic fluid suprapubically.

If the presenting part cannot be displaced from the pelvis, the second choice of site is infront of the fetus among the limbs, usually at the umbilicus of the mother. This site may be hazardous since the fetus is often facing the placenta. As a last resort, liquor may be obtained from behind the neck. In both these situations the depth of the middle of the pool of liquor from the abdominal skin surface is measured from the point of spinal needle and with a finger on the needle to act as a guard it is

plunged with one movement into the uterus. There are occasions where there is apparently no available liquor or where the only liquor is under the placenta. In these circumstances the risks of attempting and failing to obtain liquor must be weighed against the benefits if liquor is obtained.

After the fluid is aspirated and the needle withdrawn, the patient is observed in left lateral position for 1-2 hours, during which time the fetal heart beat is auscultated quarter hourly.

ii) **Surfactant Test**

The bubble test is used in this situation. 1ml of amniotic fluid and an equivalent amount of 95% ethylalcohol are added to test tube A. 0.5mls of normal saline and 0.5mls of amniotic fluid together with 1ml of 95% ethanol are added to test tube B. Both tubes are vigorously shaken for 15 seconds and left to stand in the test tube rack in good light for 15 minutes. The persistence of an intact ring of bubbles at the air-liquid interface is a positive test.

iii) **Pelvic Digital Examination**

The patient is explained the purpose of the examination. She is then asked to lie comfortably on the examination couch with legs flexed and abducted. The obstetrician scrubs and puts on sterile gloves. The vulva is cleaned with an antiseptic solution such as hibitane or savlon. The vulva is usually scrabbed from above downwards and away from the introitus. The vulval folds are carefully cleaned.

An inspection of the labia, vestibule and introitus is made. The examiner's gloved thumb and forefinger of the left hand separate the labia to expose the introitus. This is to prevent the examining fingers from coming into contact with the labia. The right index and middle fingers are then introduced into the vagina (second one first).

The following points are noted:

i) Cervix - Consistency – whether soft or firm, Position, effacement (length of cervix) and dilatation.

ii) Membranes - Whether intact or ruptured and the colour of the liquor.

iii) Cord - Presentation or prolapse

iv) Presenting part- Nature, position, station, presence and degree of caput and/or moulding.

v) Clinical pelvimetry- Adequacy of the pelvis is assessed from the diagonal conjugate, sacral promontory, sacral curve, prominence of Ischael spines, pelvic sidewalls, subpubic angle and the intertuberous diameter. The pubic angle is checked whether it is smaller or greater than 90%, the intertuberous distance is assessed whether it can accommodate 4 knuckles of a closed fist.

vi) The vagina and perineum - distensibility and moistness, presence of any discharge. For obstetric patients, if the cervix is dilated 3-4 cm and the membranes are bulging, amniotomy is done with Kocher's forceps after membranes have been swept free from the lower uterine segment. The presence of meconium is noted.

iv) **Speculum Examination**

Preparation of the vulva is as described above. Both the labia majora are separated by the thumb and index finger of the gloved left hand. Cuscos speculum is then introduced into the vagina under good light with the blades horizontal and the valves opened to visualize the cervix. The latter is inspected for dilatation, bleeding, drainage of liquor, any local lesions or presence of any discharge. The vaginal walls are also inspected as the speculum is withdrawn gently in the same manner it was introduced.

MANAGEMENT OF LABOUR

Patients in the lying in wards coming to labour ward for elective induction of labour are given a warm bath and a soap enema on the morning before induction. Other patients are managed as they come. Each patient is reviewed by the senior house officer on duty.

i) **First Stage of Labour**

If the patient is in active labour, a partogram is started immediately. This is a useful chart for monitoring the progress of labour. The following are recorded on the partogram.

1) Patient's particulars and visit number. Time of each observation and procedure and any medications given.
2) The fetal heart rate and rhythm
3) The frequency and strength of uterine contractions
4) Station and position of presenting part
5) Cervical dilatation and degree of effacement
6) State of fetal membranes and time of rupture
7) Colour of liquor
8) Degree of caput formation and moulding
9) Urinalysis at each voiding for glucosuria, proteinuria and ketonuria.

The Partogram is charted every 30 minutes. A pelvic examination is performed every 4 hours or more often as the circumstances. Alert and action lines are drawn to a gradient 1cm/hour cervical dilatation at the time of first recording and then 4 hours apart after cervical dilatation of 3cm and 4cm for primigravidae and multigravida patients respectively.

Active management of labour is practiced at this institution. Using the partogram, any abnormalities of labour such as maternal or fetal distress, poor progress due to incoordinate uterineaction, cephalopelvic

disproportion, or malpresentation and malposition can be detected early and intervention made.

A cervical dilatation of at least one centimeter per hour is expected for labour to be progressive. Delivery is aimed at 12 hours or earlier after the onset of labour. Amniotomy is done early in labour at 3-4cm dilatation, however ,amniotomy is delayed until 4 hours to delivery to minimize vertical transmission of the virus

Analgesia is given in the early stages of labour. Pethidine and pethilophan are used here but morphine is preferred for cardiac patients and sicklers and also those with antepartum haemorrhage.

Augmentation of labour is done with oxytocin as an infusion in 5% dextrose or Ringer's lactate. The patient is encouraged to lie in the left lateral position.

Those for induction, amniotomy and oxytocin drip are instituted in the morning. Prostagladin vaginal pessaries are used for cervical ripening. Extra-amniotic prostaglandin induction of labour is preferred in cases with intrauterine fetal distress.

ii) **Second Stage of Labour**

This starts from the time of full cervical dilatation to the complete delivery of the baby. Normal uncomplicated vaginal deliveries are usually conducted by student midwives under supervision or by qualified midwives. The high risk deliveries, such as breech deliveries, twin deliveries, premature deliveries or vacuum extractions are carried out by the obstetric senior house officer.

The one conducting the delivery is usually scrubbed, gowned and gloved. Vulval and perineal toilet using antiseptic solution is done. The patient is then draped with sterile linen. The delivery tray is kept handy and it contains among other things cord clamps, scissors, syringe and needle, cord ligatures and towels for wrapping the baby in. Digital examination is done to confirm that the patient is in second stage. The patient is encouraged to bear down with each contraction. If the perineum is tight it is infiltrated with 10-20mls of 1% lignocaine hydrochloride or 2% procaine hydrochloride solution. Then episiotomy is given (usually a mediolateral one). An episiotomy is also indicated as when the baby is preterm during crowning of the head.

The episiotomy is made using a blunt tipped Mayo's scissors. The index and middle fingers are used to stretch the perineum and to guard the baby's head, while the right hand is used to make the incision. The incision involves the vaginal mucosa, the skin over the ischiorectal fossa, the bulbospongiosus and the superficial perineal muscles.

With further uterine contractions and maternal efforts to bear down, the presenting part of the fetus distends the perineum, which should be supported using a sanitary pad. Once the head is delivered the nostrils and mouth are wiped with gauze to remove mucus and blood. One finger is passed around the neck, and if the cord is found that is tight, it is divided between clamps otherwise it is simply slipped over the head.

The anterior shoulder is then deliverd while at the same time ergometrine injection is given unless it is not indicated as in cardiac disease, hypertension, in multiple gestation, sickle cell disease or severe anaemia.

The posterior shoulder, trunk and legs are delivered. If the cord has not been cut, it is now clamped and cut. The baby is shown to the mother briefly before being taken away by an assistant for Apgar Scoring and

resuscitation if necessary. The baby is examined for any birth defects. Warmth is provided, and an identification band is fastened to one of the baby's limbs. Finally the baby is weighed.

iii) **Third Stage of Labour**

A kidney dish is placed against the patient's perineum to receive blood and placenta. Signs of placental separation are awaited. Once they appear the placenta is delivered by controlled cord traction as described by Brandt-Andrews. The signs of placental separation are:

1) Hardening of the fundus as it becomes more globular
2) The uterus rises in the abdomen as the separated placenta passes down to the lower segment and vagina, where its bulk pushes the uterus upwards
3) There is often a sudden gush of blood
4) The umbilical cord protrudes further out of the vagina indicating that the placenta has descended.

Delivery of the placenta is accomplished by applying upward pressure to the body of the uterus with one hand that is placed on the abdomen, while with the other hand gentle downward traction is applied on the umbilical cord. Once the placenta reaches the introitus, pressure on the uterus is stopped. The placenta is gently lifted away from the introitus taking care not to tear off the fetal membranes. If the membranes start to tear, they are grasped with a clamp and removed by gentle traction.

After delivery, the placenta and membranes are examined for completeness, infarcts, retroplacental clots and any abnormalities. The placenta is then weighed and discarded.

The third stage of labour normally lasts within 20 minutes. After that time, a diagnosis of retained placenta is made, and this will require manual removal under general anaesthesia. The perineum, vagina and cervix are examined for any tears. Minor perineal and vaginal tears are repaired

under local anaesthesia, while those more extensive including cervical tears are repaired under general anaesthesia.

iv) **REPAIR OF EPISIOTOMY**

This is usually done by whoever conducted the delivery. The patient is placed in lithotomy position. The vulva and perineum are cleaned with antiseptic solution such as hibitane. A sterile gauze is packed high up in the vagina and the posterior vaginal wall is retracted with the index and middle fingers to expose the apex of the episiotomy. The vaginal wall is repaired from the apex outwards using continuous 2/0 chromic catgut suture on a round bodied needle. The perineal muscles are approximated using interrupted number 0 chromic cutgat. The perineal skin is finally repaired using interrupted 2/0 chromic catgut with the knots buried under the skin. The vaginal pack is removed and a digital rectal examination is performed to make sure the rectal mucosa has not been involved in the repair. A sterile sanitary pad is applied. Post delivery observations are made and delivery notes completed. The birth is notified in the Register.

"Fourth Stage" of Labour

The hour immediately following delivery of the placenta is a critical period and has been designated by some obstetricians as the "Fourth Stage of Labour". Post-partum haemorrhage as a result of uterine relaxation is most likely to occur at this time.

During this stage the uterus is frequently evaluated and massaged whenever signs of relaxation are seen. The perineal region is also inspected to promptly indentify any excessive bleeding.

CARE AFTER VAGINAL DELIVERY

The patient is kept in labour ward for about two hours, and then transferred to the lying in wards when her general condition and vital signs are stable. Most patients are discharged home withing 24 hours of delivery. The patient is advised on perineal hygiene and use of

sit baths. Post-natal exercises are encouraged and the patients are reviewed in the postnatal clinic after 6 weeks.

OBSTETRIC OPERATIONS

These include procedures such as amniotomy, episiotomy, manual removal of placenta, repair of cervical tears, vacuum extractions, insertion of cervical stitches, postpartum tubal sterilizations, caesarean sections and occasionally subtotal hysterectomies. The vacuum extraction and caesarean section operations will be discussed while the rest will be mentioned where appropriate in the case histories.

i) Vacuum Extraction

This is an assisted vaginal delivery when the vertex is presenting. Indications for its use include poor maternal effort, foetal distress, cord prolapse at or near full cervical dilatation and to shorten the second stage of labour in cardiac disease, hypertension, respiratory disease, abruptio placentae or severe anaemia. It is usually an elective or an emergency procedure.

The patient is placed in lithotomy position and preparations are as of any vaginal delivery as described above. The bladder is catheterized. A medio-lateral episiotomy is usually made under local anaesthesia. The largest vacuum cup that is able to slip inside the vagina and through the cervix is then applied onto the fetal scalp at the vertex and a finger passed round its perimeter to confirm exclusion of maternal tissues from its hold of the cup. The vacuum suction pressure is then built gradually at a rate of $0.1kg/cm^2$ to $0.5kg/cm^2$ and should not exceed $0.8kg/cm^2$ to achieve a well-formed chignon.

The patient is then encouraged to push with each uterine contraction except in cardiac cases where pushing is discouraged. Gentle traction is applied at the height of each contraction with the ventonse along the midline plane of the pelvis.

Once the baby's head is delivered, the suction pressure is released and the cup removed. The rest of the delivery is then completed as in a normal delivery.

ii) **Caesarean Section**

The lower uterine segment caesarean section is the most frequently done. Classical sections are rarely performed.

With the exception of emergencies, the patient is starved from midnight of the day of operation. Shaving of hair on the abdomen and vulva is done. Informed consent is obtained from the patient and relevant consent forms are signed. Premedication is given about half an hour before the scheduled operation with intramuscular 0.6mg atropine sulphate – but for cardiac patients 0.4mg hyoscine is preferred. The patient is then taken to theatre.

On the operating table, the patient is placed in dorso-lithotomy position; vulvovaginal toilet is done with hibitane or savlon solution. The urinary bladder is aseptically catheterized, and the catheter left in situ for the duration of the operation. The patient is repositioned supine. An intravenous infusion of 5% dextrose or saline is meanwhile started and 100% oxygen is given as the surgeon and the assistants are scrubbing.

The surgeon, an assistant and an instrument nurse put on sterile theatre gowns and gloves after scrubbing. The abdomen is then cleaned with an antiseptic solution such as hibitane then with spirit before being draped with sterile linen. General anaesthesia is induced with intravenous thiopentone sodium 150-500mg and maintained with nitrous oxide 6 litres per minute, and oxygen 5 litres/min given through an endotracheal tube. Muscle relaxation is achieved with 50-100mg of succinylcholine intravenously after induction and thereafter tubocurarine is used in a dose of 0.5mg/kg.

The abdomen can be opened through a pfannenstiel or a lower midline scar. The former is the one usually preferred in this hospital.

An incision is made extending from about 2cm below the umbilicus to the pubic hair line. Using a fresh scalpel the incision is deepened through the layer of subcutaneous fat and superficial fascia to reach the rectus sheath. The rectus sheath is opened vertically using Mayo's curved scissors. The rectus abdominis muscles are reflected sideways. The parietal peritoneum is exposed, picked up at its upper third between two spencer-wells forceps placed 2 cm apart. The tented fold of peritoneum between the two clamps is visualized and prepared to exclude any omentum, bowel or bladder before being opened with a knife or scissors.

The bladder is retracted downwards using a Doyen's retractor. If the uterus is dextrorotated this is corrected. The gut is packed away by two moist abdominal packs on either side of the gravid uterus.

The utero-vesicle peritoneum is opened 2cm above the bladder and the incision extended laterally with dissecting scissors. The perineal flaps are reflected downwards by sponge holding forceps. The retractor is then placed between the lower flap and the lower uterine segment. A short transverse incision is then made into the lower segment and deepened carefully until the membranes are exposed.

The incision is extended laterally using a pair of curved scissors guided by two fingers to protect vessels. The membranes are punctured with a knife. The right or left hand is then placed gently underneath the baby's head, and the head is delivered gently out through the uterine incision, after the Doyen's retractor has been removed. Delivery is aided by modest transabdominal fundal pressure.

To minimize aspiration by the fetus of amniotic fluid and its contents, the exposed mouth and nose are aspirated or wiped with soft gauze before the thorax is delivered. The shoulders are then delivered using gentle traction plus fundal pressure with the rest of the body following readily.

As soon as the shoulders are delivered, intravenous 0.5mg ergometrine is administered. The umbilical cord is divided between clamps and the baby received by the midwife and handed over to the paediatrician for any necessary resuscitation. The placenta and membranes are delivered manually or by controlled cord traction.

Green Armytage uterine clamps are applied to control bleeding, especially at the lateral angles, and also to define the incision margins. Blood is evacuated from the uterine cavity. Adequate haemostasis from the placental bed is ensured before closure. The Doyen retractor is then reapplied.
The uterine wound is closed with a locking continuous chromic catgut suture from end to the other .A second layer of simple continuous suture is placed to bury the first layer and thus achieve complete haemostasis. The utero-vesicle peritoneum is then repaired with a continuous chromic catgut No. 0 suture. The perineal cavity is cleaned after the abdominal packs are removed.

The uterus, fallopian tubes and ovaries and other abdominal viscera are inspected for any abnormalities. Swabs and instruments are checked. If these are found to tally with the first count the abdomen is closed. First, the parietal peritoneum is closed with continuous chromic catgut No. 0 suture, then the rectus sheath with continuous No. 2 chromic catgut suture and finally the skin with No. 1 interrupted nylon or silk suture.

The wound is cleaned with savlon and dressed with sterile gauze. The bladder catheter is removed unless there is an indication for its retention. Urine is inspected for any blood. The uterus is massaged and clots evacuated per vaginum. The vulva and thighs are cleaned and a sanitary pad applied.

General anaesthesia is reversed with 1.2mg intravenous atropine sulphate and 2.5mg Neostigmine. Extubation is done and the larynx sucked to remove secretions. The total blood loss is estimated. The patient is then taken out of

theatre for postoperative care in the ward after the initial postoperative vital signs are stable.

Post Caesarean section Care

The pulse, temperature, respiratory rate and blood pressure are recorded half hourly till the patient is fully awake then 4 hourly. Analgesia is achieved with intramuscular injection or pethidine 100mg six hourly for 48 hours. Thereafter oral paracetamol or indomethacin is given. Prophylactic antibiotics are administered routinely to all patients in this department. In the first two days the drug is given parenterally. The patient is given nothing orally until the bowel sounds return usually within 24 hours. Meanwhile she is maintained on intravenous fluids, 5% dextrose infusion alternating with normal saline given ½ litre six hourly.

Normal diet is gradually introduced after liberal intake of fluids and light diet. Blood transfusion may be necessary if the haemoglobin level before surgery was low or if there was excessive blood loss during surgery. Initial observations are made in labour ward. If the patient's condition remains satisfactory, she is transferred to the lying in wards.

Early ambulation is the rule. Haemoglobin level and urine bacteriological exam are done on the third postoperative day. The patient is reviewed on the 4^{th} postoperative day and if she is stable with no signs of infection, the patient is discharged home with a case summary. She is advised to have non absorbable sutures removed on the 7^{th} postoperative day and to attend the child welfare clinic and the postnatal clinic in two weeks and six weeks respectively. Those with absorbable sutures do not need to have them removed. The baby is usually given BCG and oral Polio Vaccine while still in the ward.

NEWBORNS

Unless the mother is very sick, all babies delivered normally join their mothers immediately after delivery. Breast-feeding is encouraged soon after delivery.

A paediatric senior house officer is usually available to review babies born by caesarean section, vacuum extraction, or by breech extraction. He/She also reviews high risk babies such as those of mothers with diabetes, Rh negative blood group, premature babies and babies with congenital malformation or neonatal asphyxia. Admission to nursery is done whenever necessary. Preterm babies are managed in nursery until they weigh 2000grams.

The mothers with babies in nursery are accommodated in the mothers' hostel.

POSTNATAL CLINIC

This is conducted on Friday mornings. Patients are asked about their general health since delivery, whether they have vaginal discharge, pelvic pain, resumption of menstruation, any urinary symptoms and whether lactation is well established.

A physical examination is performed to check for anaemia and any breast lumps. A Bimanual pelvic examination is carried out to find out whether the uterus is well involuted and for any adnexial masses. Any medical illness discovered is treated or referred to the appropriate specialized clinic for further management. For contraception the patients are referred to the family welfare clinic.

THE GYNAECOLOGY UNIT

This is managed by three firms. Outpatient services are provided in casualty gynaecology clinics, family welfare clinic and laparascopy clinic 66. The latter is temporarily suspended.

Inpatient emergency services are offered in ward 1D. Cold cases are managed in ward 1B where the three firms share beds. Emergency and non-emergency gynaecology theatres are among the other 10 main theatres.

Gynaecology clinic

There are three clinic days in a week held in the afternoons viz; Tuesdays, Wednesdays and Thursdays run respectively by firms I, III and II. They are manned by consultants, senior registrars, senior house officers, nurses, medical and nursing students.

Patients are referred from casualty, gynaecological wards, other clinics in the hospital and others are from health facilities in Nairobi and elsewhere in the country.

The commonest problem dealt with at the clinic is infertility, accounting for two thirds of all cases seen. Other problems commonly dealt with are disorders of menstruation, uterine fibroids and other pelvic masses.

A detailed medical history is sought from the patients at the clinic. A thorough physical examination is done. Relevant investigations are requested for. These include hormonal profiles, semenalysis, hysterosalpingography, ultrasonography and pap smears for cytology. Others are high vaginal swabs, urinalysis, hemogram and renal function tests. These are repeated whenever necessary. There is also a colposcopy room where colposcopy is done when indicated.

Cold Admissions

Cold cases are admitted through gynaecology clinics or transferred from the emergency gynaecology ward. Those admitted from gynaecology outpatient clinic have their diagnoses well established with adequate laboratory workups. In-patients are re-clerked and prepared for elective operations on specific theatre days. Cytotoxic chemotherapy for gynaecological malignancies are given in the cold gynaecological wards when indicated.

Emergency Gynaecological Ward

This is ward 1D located on the first floor of the tower block. It has a capacity of 32 beds, but usually the patients are more than the number of beds. The ward has a high patient turnover.

Among the commonest diagnoses at admission are abortions, acute pelvic infections, pelvic infections, ectopic cyesis, abnormal uterine bleeding, bartholin's abscess/cyst, translocated intrauterine contraceptive devices, carcinoma of the cervix, genital injuries, and twisted ovarian cysts . Most of the emergencies are admitted through casualty and are operated on usually within 24 hours of admission. Cases of cervical cancer are examined and staged weekly under anaesthesia.

laparascopy

Laparoscopy, which used to be done in laparoscopy clinic 66, is currently carried out in main theatre. Previously this was performed at the Rahimtulla Wing. Patients are referred from the gynaecology clinic or family planning clinic for diagnostic laparoscopy or laparascopic tubal ligation. Interval and postpartum tubal ligation using the minilap technique is also perfomed in this clinic.

GYNAECOLOGICAL OPERATIONS

Emergency operations like uterine evacuations for incomplete abortion and endometrial curettage for abnormal uterine bleeding are performed in the procedure room attached to the emergency ward using the manual vacuum aspiration Karman technique without anaesthesia. Patients are adequately counseled before the procedure and informed consent obtained.

The operations that are performed in main theatre as emergencies include laparotomy for ruptured and unruptured ectopic gestations, drainage of pelvic abscess, laparotomy of twisted ovarian cysts and tubo-ovarian masses. Others are marsupialization, dilatation and curettage, fractional curettage, removal of translocated intrauterine contraceptive devices, secondary suture of burst abdomen and tubal ligations for patients with previous scars where operation under local anaesthesia is difficult.

Most of the major operations such as vesicovaginal fistula repair, Wertheim's hysterectomy, radical vulvectomies, routine hysterectomies whether vaginal or abdominal are carried out in the elective theatre list for the different firms. Details of these operations are described in the cases presented except for total abdominal hysterectomy.

Total Abdominal Hysterectomy

The abdomen is opened via the lower midline incision in most cases. A self retaining retraction is applied to the edges of the incision and opened to expose the pelvis. The gut is packed with abdominal packs. The round ligaments are identified and are divided between two straight long artery forceps close to the uterus and doubly ligated with number 0 or 1 chromic catgut sutures.

An incision is made on the visceral peritoneum just above the bladder and extended laterally upwards through the anterior leaf of the broad ligament to reach the incised broad ligament. The bladder is then mobilized downwards from the uterus all the way down to the cervix. The posterior leaf of the broad ligament is perforated using a knife or finger to make a window beneath the fallopian tubes, utero-ovarian ligaments and ovarian vessels. These are doubly clamped close to the uterus, cut and tied with a transfixing chromic catgut suture No 2. This procedure preserves the ovaries. The posterior leaf of the broad ligament is then divided inferiorly towards the uterosacral ligaments and the peritoneum of the utero-rectal pouch down with a "swab on a stick".

The uterine vessels on either side are identified near their origin and are doubly clamped using Kocher's forceps, divided and tied with a transfixing chromic catgut No.2. The cardinal ligaments are similarly divided and tied. The anterior vaginal wall is opened after identifying the lower limit of the cervix.

Using 2 tissue forceps about 1cm apart, the upper vaginal portion is opened by an incision between them. The incision is extended laterally around the cervix using a curved scissors. This is "circumcising" the cervix.

The vaginal vault is supported with 4 long straight artery forceps and the whole of the uterus; cervix and part of the adnexa are removed. The vaginal vault is closed with mattress-interrupted sutures using chromic catgut then the pelvis is peritonealized. The abdominal packs are removed, count of swabs and instruments is made to confirm with the earlier count, after which the abdomen is closed in layers.

OBSTETRIC SHORT CASES

CASE NO. 1 : SEVERE ANAEMIA IN TERM PREGNANCY-LIVE BABY

NAME: B.O D.O.A 20/3/2002

AGE: 19yrs D.O.D 30/3/2002

IP NO: 0819334 PARITY 0+0

Presenting Complaints

She presented with one-week history of easy fatigability, palpitations, headache and dizziness. There was however no chest congestion or cough.

History of Presenting Complaints

She presented to the casualty with the above complaints, which had progressively worsened one week prior to admission. She also had low abdominal pain and backache for two days. There was no history of similar illness outside pregnancy. There was no history of blood loss, fever, cough or other recent illness. She had no history of recent travel outside Nairobi and had not received any treatment prior to admission.

Obsterics and Gynaecologic History

She was a primigravida; her menarche was at 16 yrs of age, her periods occurred regularly every 28 days with a moderate flow lasting 3 days. Her last menstrual period was on 14/6/01 and the expected date of delivery was 21/3/02, giving a gestation of 39+ weeks. She attended antenatal care at a private clinic but could not avail the antenatal records. She gave no history of contraceptive use.

OBSTETRIC SHORT CASES

CASE NO. 1 : SEVERE ANAEMIA IN TERM PREGNANCY-LIVE BABY

NAME: B.O D.O.A 20/3/2002

AGE: 19yrs D.O.D 30/3/2002

IP NO: 0819334 PARITY 0+0

Presenting Complaints

She presented with one-week history of easy fatigability, palpitations, headache and dizziness. There was however no chest congestion or cough.

History of Presenting Complaints

She presented to the casualty with the above complaints, which had progressively worsened one week prior to admission. She also had low abdominal pain and backache for two days. There was no history of similar illness outside pregnancy. There was no history of blood loss, fever, cough or other recent illness. She had no history of recent travel outside Nairobi and had not received any treatment prior to admission.

Obsterics and Gynaecologic History

She was a primigravida; her menarche was at 16 yrs of age, her periods occurred regularly every 28 days with a moderate flow lasting 3 days. Her last menstrual period was on 14/6/01 and the expected date of delivery was 21/3/02, giving a gestation of 39+ weeks. She attended antenatal care at a private clinic but could not avail the antenatal records. She gave no history of contraceptive use.

Past Medical History

This was not significant.

Family and Social History

She was married and living with her husband in Nairobi. Both parents were alive and well. She was a fourth born in a family of 8 children who were alive and well. Her grandmother was diabetic; her stepmother had history of twins. She neither took alcohol nor smoked cigarettes. There was no history of other major illness.

General Examination

She was a young lady of fair nutritional status and general condition. She was clinically very pale, had mild bilateral pedal pitting oedema, no jaundice, no lymphadenopathy and no cyanosis. Her temperature was 36.8^{o}C and the respiratory rate was 24/minutes.

Cardiovascular System

The pulse was regular and of good volume with a rate of 100 beats/minute. The blood pressure was 100/70 mm Hg. The jugular veinous pressure was not raised and the praecordium was not active. Both heart sounds I and II were heard and were normal. There was a systolic flow murmur at the apex which was in the 5th intercostals space along the medial axilliary line.

Respiratory System

There was good air entry bilaterally. No basal crepitations were auscultated

Abdominal Examination

The abdomen was uniformly distended with a fundal height corresponding to a 36 weeks gestation. The fetus was in a longitudinal lie and cephalic presentation and the fetal heart tones were heard and regular with a rate of 148/minute. Both liver and spleen were not palpable. No uterine contractions were elucidated.

Central nervous system

This was essentially normal.

Diagnosis

Severe anaemia at a term pregnancy.

Investigations

Haemogram:

Haemoglobin	-	5.7 g/dl
WBC	-	$5.4 \times 10^9/1$
RBC	-	$1.33 \times 10^{12}/1$
MCV	-	99.9 fl.
Platelets	-	$270 \times 10^9/1$

Periferal blood film:

RBC	-	Showed anisocytosis, hypochromia, Polychromasia and macrocytosis
WBC	-	PMN 67%, Lym 32%
Haemoglobin Electrophoresis	-	HBAS
Blood slide for malaria parasites	-	Negative
Stool ova/cyst: occult blood	-	Negative
Urinalysis	-	Normal
VDRL	-	Negative
Blood group	-	"O" Rhesus positive
P24 markers	-	Negative

Management

The patient was admitted to the antenatal ward and started on Ranferone; a presumptive treatment for malaria with 3 tabs of fansidar was given since she had none prior to admission. With the haemoglobin level of 5.7 g/dl at 39 weeks gestation, a decision was made to transfuse her 3 units of blood each with 40 mg intravenous lasics bolus. Each unit was transfused over a period of 8 hrs under close observation of vital signs. The transfusion went on well without any complications. Meanwhile, she was continued on hematinics while awaiting labour. A repeat haemogram done on 27/3/2002 showed a haemoglobin level of 9.5g/dl. The patient had no complaints. On 29/3/2002at 5 a.m, she reported lower abdominal pain and backaches for two hours. She was now at a gestation of 41 weeks. The patient was taken to the labour ward where she was reviewed and the abdominal findings were: Cephalic presentation, longitudinal lie, fetal head was 3/5 up and fetal heart rate was 142/minute, regular. Two moderate uterine contractions each lasting 20 to 30 seconds were palpable every 10 minutes. Pelvic examination showed normal external genital with show at the introitus. The cervix was 5cm dilated; membranes bulging, no cord felt. Amniotony was done and clear liquor was obtained presenting part vertex, station zero, left occipital anterior position . Blood was taken for grouping and cross matching and two units were requested; she was started on a partogram and planned for vaginal delivery.

At 7.30 a.m, she had 3 strong uterine contractions and expressed a "Desire to push". Vaginal examination confirmed second stage labour. Patient was taken to delivery room and encouraged to push. A live male baby weighing 2600 grams was delivered; apgar score was 9 in 1 minute and 10 in 5 minutes. The placenta was delivered by controlled cord traction; it was inspected and found to be complete; it weighed 400 grams. The cervix and the perineum were explored and found to be intact; the uterus was well contracted. Total blood loss was estimated to be 150mls. Immediate postpartum vital signs were: Pulse 84/minutes, respiratory rate 22/minute, temperature 36.6^{o} and blood pressure was 100/60 mmHg. She was observed in the labour ward for 2 hours and remained stable. She was then transferred back to the lying-in ward

Review after 24 hrs was done; the patient had no complaints and the baby was well. The vial signs were normal, the uterus was well contracted and the lochia loss was normal. She was restarted on Ranferone and discharged home on this treatment to be reviewed in the postnatal clinic after two weeks and for repeat of haemogram. Meanwhile, she was counselled on the need for malaria prophylaxis; she was adviced to take three tablets of fansidar after 2 weeks.

Postnatal Clinic

She did not turn up on the scheduled appointment and was lost to follow up.

COMMENT

The patient was a 19 yr old primigravida who presented with severe anaemia at term, for which she was transfused 3 units of blood. She subsequently went into labour and delivered a live male infant weighing 2600gms with a good apgar score.

Anaemia is a significant maternal problem during pregnancy; a pregnant woman will lose blood during delivery and in the puerperium and an anemic woman is therefore at increased jeopardy. In practice, a haemoglobin level of 10.5 g/dl or less (equivalent to a haematocrit of 33%) is generally considered the criterion for diagnosing anaemia in pregnancy[1,2]. This takes into consideration the physiological expansion of the mother's blood volume, which consists of an increase in plasma volume (40-60%) and the red cell, mass by 20-50%[3]. The incidence of anaemia in pregnancy varies worldwide due to the different causative and associated factors; it is particularly high in tropical countries of Africa, South America and South East Asia[2,6].

The patient presented here was a teenage mother; she had no history of contraceptive use.

Anaemia occurring in pregnancy is most frequently due to deficiency of iron or folate or both; iron deficiency accounts for up to 95% of the cases, a reflection of the increased demand by the fetus, and postpartum blood loss.[1,2,3] This overwhelms most women's marginal iron stores, thus causing a deficiency. Many women already have depleted iron stores before conception, which aggravates the pregnancy.[1,3,11] Other anaemias occurring in pregnancy are aplastic anaemia, drug induced hemolytic anaemia and the

haemoglobinopathies (i.e sicle cell, thalassemias).[1,2,3] In tropical countries, other additional causative factors include illnesses[6]. In Kenya Mati[7] found that up to 32.8% of the anaemic patients had malaria while only 4.3% had ova of Ancylostoma duodenale in the stools.

The patient presented had anaemia which showed features of iron deficiency on the peripheral blood film i.e the RBCs showed anisocytosis with hypochromia and polychromasia. There was no avidence of malaria or hookworm infestation. Haemoglobin electrophoresis showed she had sickle cell trait; however, anti-malaria treatment was still given because sickle cell trait alone is rarely a cause of anaemia while malaria remains very common. However, it is well documented that patients with sickle cell trait (HbAS) have inherent resistance to P.falciparum malaria[1,2,3,6,9,11]

The pathophysiology of anaemia depends on the aetiology; deficiencies of iron and folate impair erythropoiesis hence causing production of abnormal red blood cells with reduced concentration of hemoglobin. Hookworm infestations or chronic blood loss lead to iron deficiency. Hemolysis occurs with chronic illnesses, bone marrow suppression is a result of the disease itself as well as the drug used in controlling the disease e.g in cancers, renal diseases etc[1,2,3]. The patient presented gave no history of any such possible aetiological factor.

Presentation of anaemia may be vague and non-specific, especially when it is of insidious onset. The symptoms when present, consist of undue fatigue, dyspnoea, headache, dizziness, palpitations, loss of appetite and digestive disturbances[1,2]. These symptoms are those of tissue hypoxia, those of the cardiovascular systems attempts to compensate for the anaemia or those due to an underlying diseases[8]. The signs include pallor and oedema of various degrees depending on severity of the anaemia. Jaundice may be present as an indicator of excessive hemolysis; others such as lymphadenopathy, hepatosplenomegaly, melaena stool (or frank hematochezia), glossitis, stomatitis and koilonychias are indicative of the possible causes of effects of the anaemia [1, 8]. Clinical situations associated with anaemia include multiple pregnancies, chronic renal and liver diseases, as well as chronic infections[8]. The patient presented already had symptoms of cardiovascular compensation (i.e tachycardia, palpitations); she had pallor as well as

padal pitting oedema. Other signs were not present. Anaemia itself is not a diagnosis; it is a sign of an underlying diseases process. The key issue in evaluation of a patient with anaemia is to find an underlying diseases process, keeping in mind that the underlying process may be of a grave consequence. The genetic implications are equally important [8]. The patient presented here had a genetic condition, a hemoglobinophathy. A thorough medical history and a full blood count with a reticulocyte count provides an important diagnostic tool[1,8]. Additional studies include basic ones such as blood slide for malaria parasites, stool microscopy and occult blood and urinalysis; others such as haemoglobin electropheresis, bone- marrow aspirate, sickling test and serum levels of hemopoietic elements can also be done[1, 2, 8].

The patient presented had a history and physical examination complemented by laboratory tests; the haemoglobin electrphoresis showed a blood disorder while the complete blood count showed severe anaemia with a haemoglobin level of 5,7g/dl. The peripheral blood film showed features of iron deficiency.

The management of anaemia in pregnancy depends on the cause, the severity and the gestation. With a haemoglobin level that is not below 6g/dl in the first and second trimester, hematinics are usually sufficient. With levels below 6 g/dl transfusion is required irrespective of gestation [9].Other indications of transfusion include evidence of cardiac failure due to anaemia, and anaemic patients in the last four weeks of pregnancy or in labour [1,6,9]. This patient had a haemoglobin level of 5.7g/dl at 39 weeks gestation thus transfusion was indicated. The dangers of blood transfusion should bone in mind in such patients due to cardiac decompensation; the aim is to transfuse with parked cells or give a patient intravenous diuretic to avoid fluid overload. The technique of exchange transfusion has been used successfully. A maternal mortality rate of 1.3% was reported in patients who had exchange transfusion as compared to 19.6% in those who underwent direct transfusion; such patients should labour while propped up in bed, examined and delivered very aseptically and planned for assisted second stage delivery (vacuum or low forceps); care should be taken to avoid postpartum haemorrhage[9]. In the patient presented, blood was taken for grouping and cross matching.

Complications associated with anaemia in pregnancy are maternal and fetal. Maternal complications include cardiac failure and maternal death, worsening of concurrent illnesses, increase incidence of sepsis, and increased thromboembolic phenomena[9]. Those undergoing caesarean delivery have poor wound healing and wound dehiscence is not unusual. Poor myometrial contraction leads to more prostpartum haemorrhage. The patient described here did not develop any of these complications; the total blood loss at delivery was 150mls.

Maternal anaemia is known to cause intrauterine fetal growth retardation, fetal death and low birth weight. Placental gigantism (over 900 grams) has been reported. There is a high incidence of late abortions, preterm labour and perinatal deaths[6,9,11]. The fetal outcome in this patient was a 2600 gram baby, fairly small for gestational age; the placental weight was within normal.

Prevention of anaemia in pregnancy revolves around identifying the risk factors in the population; this involves nutritional education, prophylaxis against malaria and hookworm, screening for haemoglobinopathies and routine hematinic supplementation[6,9,11].The serious effect of malaria on the course of pregnancy makes efficient prophylaxis mandatory. Presumptine treatment of malaria in antenatal period has been used successfully with sulphodoxine-pyrimethamine drugs For the success of this programme, good antenatal care is vital for satisfactory maternal and fetal outcome.

REFERENCES

1. Biswas M., Perloff D. Haematologic disorders in pregnancy. Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th Ed. Appleton and Lange/McGraw Hill NEW York pg 448-460, 2003.

2. Whitefield C.R. Blood disorders in pregnancy In: Dewhurst's Textbook of obstetric and Gynaecology forpostgraduates 4th ed. Blackwell Scientific Publications, 1986.

3. Duffy T.D. Haematologic aspects of pregnancy In: Haematology; Basis, Principles and Practise 2nd ed.Pp 2117. Churchill Livingstone 1995.

4. Sinei S.K., Mati J.K.G., Mungai J. et al. Prevalence of anaemia of pregnancy and the role of malaria in its aetiology in rural Kenya.J. Obstet. Gynaecol. East Cent.Afr. 3:119, 1984.

5. Formulu J.N. 2 year retrospective controlled survey of maternal and fetal prognosis in anaemia of pregnancy at Kenyatta Nation Hospital. M.med Thesis, University of Nairobi, 1981.

6. Cunningham F. G., Gant N. F. Haematological disorders in pregnancy. Williams Obstetrics.21st Ed. Appleton and Lange/McGraw Hill New York pg1307-10, 2001.

7. Mati J.K.G. The importance of anaemia of pregnancy in Nairobi, Kenya, with special reference to the role of malaria in the aetiology of megaloblastic Anaemia.M.R.C.O.G. Thesis, 1968.

8. Laros R.K. Jr. Haematological disorders in pregnancy. Medicine of the Fetus and Mother pp 1139-51. J.B. Lippincott Company, 1992.

9. Harrison K.A. Anaemia, Malaria and Sickle Cell disease. Clin. Obstet. Gynaecol. 9(3):445, 1982.

10. Beishcer N.A., Holsman M., Kitchen W.H. Relation of various forms of anaemia to placental weight. Am. J. Obstet. Gynaecol. 101:801, 1968.

11. Hibbard B.M. Hemopoietic function and dysfunction. Principles of Obstetrics. Butterworth and Company Ltd., 243-61, 1988.

CASE NO. 2: SICKLE CELL CRISIS IN PREGNANCY-TWIN GESTATION,MACERRATED STILL BIRTH

Name: J.A Parity 0 + 0

Age: 22 D.O.A 24/6/2002

IPNO: 0809879 D.O.D 19/7/2002

Presenting Complaints

She was admitted through causality with complaints of palpitations and generalized body and joint pains for two days.

History of Presenting Complaint

She was a known sickler since childhood and had been well a week prior to this admission. It all started with multiple body aches and swelling of joints mainly involving the hip, the knee and wrist joints. She would hardly walk or stand due to pain, which was not relieved by taking paracetamol. She gave a history of febrile illness one week prior to admission.

Obstetric and Gynaecologic History

She was para 0+0 gravida 1 and her last menstrual period was on 13.11.2001 with an expected date of delivery on 20.8.2002 thus giving her a gestation period of about 30 weeks. Her menarche was at 16 years following which she had regular menstrual cycles every 28 days and a flow for 3 to 5 days associated with spasmodic dysmenorrhea. She had no history of contraceptive use.

In this pregnancy, she was attending antenatal care at Dandora City Council Clinic and had no problems.

Past Medical History

She had been admitted with similar problems in Ngao Hospital many times previously and was on regular Folic acid and proguanil tablets which she had voluntarily stopped taking for two years.

Family and Social History

She was a single lady, unemployed and living with her brother in Dandora. She was the fifth born in a family of two male and five female children. Two of her sisters had died of sickle cell disease and a similar history was present in the maternal family. She neither smoked cigarettes nor took alcohol. Her maternal auntie had a history of twin delivery.

Pysical Examination

She was a young lady, sicklooking, in pain; she had deep jaundice and a temperature of 39.2°C; there was no lymphadenopaty. Her pulse rate was 102 per minute, respiratory rate was 22 per minute and the blood pressure was 120/90mmHg. She had generalized tenderness of bony areas and joints but no obvious swellings were noted.

Abdominal Examination

The abdomen was uniformly distended and moved with respiration. The spleen was 6cm below the left subcostal area and the liver was tipped but not tender. The fundal height was corresponding to a 34 week gestation, the lie was longitudinal and the presentation was cephalic. Fetal heart sounds were heard at 142 per minute and regular. There was plenty of liquor.

Respiratory, Cardiovascular and Central Nervous Systems

These were all normal.

Pelvic Examination

This was not indicated at this stage.

Diagnosis

Sickle cell crisis with a febrile illness in pregnancy .

Management

She was admitted to the antenatal ward and given intramuscular pethidine 50mg six hourly, intravenous fluids normal saline 500ml 4 hourly, i.v crystalline penicillin 2 MU six hourly, i.v gentamycin 80mg 8 hourly and folic acid 5mg daily. Blood was taken for

full hemogram, blood slide for malaria parasites and renal function tests. Stool and urine were also taken. VDRL and hemoglobin electrophoresis were also requested. An obstetric scan was requested due to uterine size being larger than dates.

Results of Investigation

Blood Slide - Positive for Malaria parasites

Hemogram:

WBC - $39.0 \times 10^9/1$

Hb - 7.5g/dl

RBC - $2.76 \times 10^{12}/1$

MVC - 83.6 fl

Platelets - $856 \times 10^9/1$

Peripheral Blood film - marked poikilocytosis with target cells and sickle cells seen. Macrocytes and Howell- Jolly bodies also seen.

WBC differentials:

Polymorphs (neutrophils) - 60%

Lymphocytes - 27%

Monocytes - 4%

Myelocytes - 5%

Eosinophilis - 4%

Sickle cells were 20% of all cells

Platelets were increased

Urea/Electrolytes:

Na+ - 138 mmol/L

K+ - 4.1 mmol/L

Creatinine - 82umo/L

Urinalysis - Proteinuria $^+1$

Stool microscopy - normal

VDRL - negative.

Hb Electrophoresis - HbSS

Obstetric scan showed viable twin pregnancy, both babies in breech presentation and fetal cardiac activity demonstrated;

Gestation by BPD - 7.0cm = 26wks + 6 days

FL - 5.1cm = 27wks + 4 days

The placenta was fundo anterior. Following the blood slide result, she was put on injection Artenum 160mg stat then 80mg daily for 4 days after which the fever subsided and a repeat blood slide was negative for malarial parasites.The haematologist reviewed the patient and recommended transfusion (at least 3 units). She got one unit of blood. She recovered well from crisis, the pain subsided completely and she was able to walk around the ward on her own by the second week of hospitalization. Her diastolic blood pressure remained between 90 – 100mmHg with development of minimal pedal oedema.

On 10-7-02, she reported loss of fetal movements completely and on abdominal auscultation, no fetal heart sounds were elicited. An urgent Obstetric scan was requested for which showed:

Twin pregnancy in transverse lie with no fetal cardiac activity demonstrated.

Twin 1 - Spalding of skull bones

Twin 2 - Scalp oedema

Conclusion - Twin intrauterine fetal death.

This ultrasonographic report was explained to the patient and the plan to terminate the pregnancy communicated to her. A repeat hemogram and a coagulation screen were done; blood was also taken for grouping and cross-matching and two units were requested.

Results

Repeat Hemogram:

Hb	-	6.2g/dl
WBC	-	$6.4 \times 10^9/1$
RBC	-	$2.15 \times 10^{12}/1$
PCV	-	18.9
MCV	-	98,5Fl

Coagulatation Tests:

PT I	-	13 seconds
Control	-	14 seconds
KCCT	-	33 seconds

Control - 31 seconds

She was then transfused one unit of whole blood and while awaiting induction of labour, she went into spontaneous labour on 14.7.02 resulting in the birth of twin macerated still births.

- 1st twin was female in breech presentation weighing 750g with placenta weighing 150g.
- 2nd twin was male in cephalic presentation weighing 800g with placenta weighing 20g.

The total blood loss was 150mls; on the following day, she was transfused one more unit of blood. The immediate post-partum period was uneventful, her vital signs remained normal, the lochial loss was normal and the uterus remained well contracted; the breasts were not engorged. She was discharged home on folic acid 5 mg daily and proguanil tablets 100mg daily to be followed up in the hematology clinic. She was also put on tablets bromocriptine 2.5. mg twice daily for five days and a paludrin tablet weekly. She was then advised to come back to the postnatal family planning clinic for counseling after six weeks.

Follow-up

She had no complaints. She was in good general condition and moderately pale. The breasts were not active and the uterus was not palpable abdominally. She chose to abstain as she was single and was counseled on the fact that sickle cell disease is inherited and her offsprings were likely to have the disease. She was informed that subsequent pregnancies could aggrevate the disease process leading to increased perinatal/maternal morbidity/mortality

COMMENT

The patient presented was a 22 year old primigravida who had sickle cell disease at 30 weeks gestation and presented with sickle cell crisis for which she was managed but lost the pregnancy.

Sickle cell disease denotes a group of inherited blood disorders occurring mainly in people of African ancestry and characterized by a predominance of haemoglobin S (HbS) in the erythrocytes. The major variants included in this condition are homozygous sickle cell anaemia (HbSS), sickle cell trait (HbAS), sickle cell hemoglobin C disease (HbSC), sickle cell B- Thalassemia (HbS/B –Thal) and a combination of sickle cell and hereditary persistence of fetal hemoglobin (HbS/HPFH)[1]. The disease results from abnormal synthesis of the Beta chain of the hemoglobin molecule which results in erythrocytes assuming a sickle shape when exposed to hypoxic or acidotic environment.

Sickle cell disease is widely distributed throughout the world; in Britain, the carrier rates among Negroes is about 10% while in Tanzania, HbSS occurs in about 1% of the population. In Jamaica HbSS occurs once in every 300 births[2,3]. Among the blacks in the United States, 10% have sickle cell traits while 1 in 500 has sickle cell anaemia[1]. In Kenya sickle cell disease is mostly encountered in the "sickle belt" i.e Coast, Western and Nyanza provinces, where the incidence is abut 1%.

Few studies have been done on the overall incidences of pregnancy and its complications among sicklers worldwide. There is a worldwide increase in the cases of sickle cell aneamia in pregnancy due to increasing survival of children with sickle cell towards adulthood and motherhood. The median age at death is 42 years for men and 48 years for women[1].This patient was 22 years old.

Sickle cell disease is characterized by chronic hemolytic anaemia and intermittent crises of variable frequency and severity. The disease results from an autosomal recessive disorder of hemoglobin synthesis resulting in abnormal hemoglobin molecular structure which influences its properties which are essential for function[1,3]. In sickle cell disease, there is a genetic substitution of valine for glutamic acid in the sixth position of the beta chain resulting in unstable hemoglobin molecules that lead to a sickle shaped red cell in low oxygen tension. These unstable erthrocytes undergo circulation trauma leading to shortened life span through hemolysis while intravascular sickling in the microcirculation leads to vaso-occlusion and infarction of various organs leading to the different crises.

The inheritance pattern affects both sexes equally. Patients homozygous for haemoglobin S gene manifest with sickle cell anaemia (HbSS)[3,4]. Those carrying the S-gene in a heterozygous state have sickle cell trait and develop symptoms only under significant low oxygen tension. They have twice as many UTI, as normal women.

This patient has the gene in homozygous state hence had sickle cell anaemia. Pregnancy in patients with sickle cell disease remains a substantial clinical problem as evidenced by poor fetal-maternal outcome; the overall status of the disease fluctuates but tends to be exacerbated by pregnancy. Crisis occur more frequently and severely and anaemia may have sudden deterioration; cardiac failure, pulmonary complication. Generalized infections and pre-eclampsia also tend to occur with higher frequency[3,4,5]. Detrimental effects on the fetus include abortions, stillbirths, preterm delivery and intrauterine growth retardation; this fetal wastage results from placental insufficiency due to maternal placental bed thromboses, which explains a cumulative probability of fetal death of 25% by the end of first trimester[5].

Due to the chronicity of the disease most patients tend to be asymptomatic; hemolytic anaemia is chronic with hemoglobin usually around 8g/dl and an associated tinge of jaundice is common. The anaemia worsens in pregnancy due to normal increased physiological requirements; it also tends to get worse during the various crisis due to increased sequestration and red cell breakdown[3,4]. This patient presented with hemoglobin of 7.5g/dl and had deep jaundice; the hemoglobin was likely lowered to this level by the concurrent sickling crisis and twin gestation hence the drop to 6.2g/dl by the end of crisis.

Splenomegaly is a common feature due to increased reticuloendotheliali activity. Crises of variable frequency and severity occurs and normally have a triggering factor. Painful crises include severe pain and swelling in bones and joints usually precipitated by dehydration, acidosis or infection; common infections are Malaria, Pneumonia and urinary tract infection which may be a trigger as well as a sequelae of the crisis[3,4,6]. The patient was admitted in crisis with a fever and the blood slide was positive for Malaria parasites; a dipstix urine test had shown a proteinuria $^{+}1$ but a culture was not done.

Aplastic crisis are characterized by rapidly developing anaemia when the hemoglobin can suddenly drop to 2g/dl; it results from cessation of bone red cell production. An acute splenic sequestration crisis is a consequence of sudden massive sequestration of red cells in the splenic sinusoids resulting in severe anaemia and hypovolemic shock[3,4]. These two

crises can be rapidly fatal. The patient did not develop these life threatening conditions. Pseudotoxemia, whereby there is systolic hypertension and albuminuria also occurs during a painful crisis and is rapidly fatal[6]. It was not looked for in the patient though she had a blood pressure of 120/90mmHg and protenuria $^{+}1$ on admission which were ascribed mainly to effects of multiple gestation rather than to the sickling crisis.

In any management of a pregnant sickle cell anaemia patient presenting with acute abdomen, differential diagnosis should also consider abruptio placenta, acute pyelonepihritis and if in early pregnancy, ectopic gestation should be ruled out[(7)]. Thorough and comprehensive laboratory investigations should be routinely carried out in patients who present in crisis. Full hemogram and peripheral blood film will show evidence of anaemia, sickling, a left shift in red cell maturation with a high number of reticurocytes and myelocytes as well as signs of infection as shown by elevated white cell counts. Urine analysis for urinary tract infection (which is fairly common) is an important test during management. Screening tests for the sickling phenomenon include the sodium metabisulphite and the sickledex tests which will pick all sicklers while electrophoresis will determine their genotype character[4]. This patient had most of these tests done on admission; her genotype was noted to be HbSS; her hemogram showed anaemia and the peripheral blood film showed sickle cell and target cells; while her blood slide was positive for malaria parasites.

Other tests may be done for obstetric indications as happened in this patient; ultrasonography was done and confirmed twin gestation and later to confirm intrauterine fetal death; it can also be done to assess for intrauterine growth retardation and for placental compromise.

Management of sickle cell disease in pregnancy requires a multidisciplinary approach involving the hematologist, obstetrician, neonatologist and good laboratory backup; it has been observed that in high prevalence areas, all medical personnel should be well acquainted with the inherent complications[6,7]. Prenatal genetic counseling is vital and should encompass both males and females in the affected couples[4,6,7]. Good prenatal care and treatment of antecedent complications will provide good outcome. Individualized patient care should be encouraged. Anaemia is a chronic problem and usually of folic acid deficiency type due to high bone marrow activity. Regular folic acid supplements are recommended with transfusion of blood if hemoglobin drops to below 8g/dl or hematrocrit below 25%[4,5]. Malarial prophylaxis should be provided throughout

pregnancy and even prenatally as infection can be a cause of profound anaemia[6]. Blood transfusion should also be considered in cases of repeated crisis, or in symptomatic cardiovascular compromise; Aluoch[7] noted that in cases of hemolytic crisis, transfusion of packed cells is mandatory. The patient presented needed transfusion and was given two units of blood when hemoglobin dropped to 6.2g/dl. Management of sickle cell pregnancy is no different from that in other patients[7]. Adequate rehydration is essential; it is recommended that normal saline infusion to alternate with 5% dextrose at a rate not less than 6ml/kg/hr; pethidine is given intramuscularly as 50mg not more than 3 hourly till the pain subsides after which paracetamol may be continued [2, 4, 7]. Concurrent malaria and other infections are treated as appropriate. Avoidance of acidosis and hypoxia further improves recovery[2, 3, 4]. All the above management principles were utilized in our patient.

Delivery should always aim for the vaginal route as surgery and general anaesthesia predispose to crisis hence they should be avoided. It is recommended that labour be managed similar to that in cardiac patients[2]. Sequestration has been noted to occur substantially during labour, delivery and early puerperium hence any fall in hemoglobin below 6g/dl or at the rate 2g per 24 hours will require exchange transfusion with donor red cells containing only HBA[2].

Prophylactic caesarean section in cases where necrosis of the femoral head and decompressed lumbosacral bodies compromise pelvic diameters is considered not justified[7]. The role of prophylactic partial exchange transfusion (PPET) has been debated and some studies found it beneficial with resultant higher fetal salvage and better birth weights as compared to cases where transfusion was done[5, 8]. The rationale for transfusion therapy to above 10g/dl was to suppress erythropoesis thus minimizing endogenous production of HBSS. This checks crisis episodes thus reducing placental insufficiency thereby decreasing fetal wastage and also improving the general status of the mother.

Prognosis in pregnancies complicated by concurrent sickle cell disease has become favourable; studies[5, 6, 8] have shown improved maternal and fetal outcome with decreased incidence of maternal mortality. The causes of mortality include infections, sudden deaths during massive crisis and embolism and obstetric associated mishaps[4].

Fetal wastage remains high as happened in this patient in whom the chances of pregnancy loss were heightened by concurrent multiple pregnancy. Surviving babies tend to be

smaller than in normal women; with prophylactic transfusion, fetal salvage rates were found over 75% in sickle cell anaemia with 30% of babies being over 2500g as compared to wastage of up to 80% without transfusion[5, 7, 8]. This patient lost both babies who were delivered vaginally as macerated still births.

Inducing of nonsickling red blood cells from bone marrow has been considered.Recombinant erythropoietin and hydroxyuria have been used together recently with elevation of haemoglobin F. More recently intravenous arginine butyrate has been used with equal success.[4]

These patients should be offered reliable contraception; combined oral contraception is contraindicated due to increased risk of thromboembolism while intrauterine devices predispose to infections[2, 4, 7]. Sterilization should be considered if maternal complications become life threatening[2, 4]. Progestin only contraceptives can be used safely in these patients. The patient described was single and chose abstinence.

Women with sickle cell anaemia or trait, planning to have children should have their partners tested for the sickle cell disease or trait for appropriate genetic counseling.[1,2]

REFERENCES

1. BiswasM.K., Dorothea P. Sickle cell disease. Current Obstetric and Gynaecologic Diagnosis and Treatment 9th Ed. Appleton and Lange/Mc Graw Hill New York Pg. 412-414,2003.

2. Cunningham F.G., MacDonald P.C., Grant N.F. Sickle cell haemoglobinopathies.Williams obstetrics 21st Ed. Appleton and Lange Mc Graw Hill New York Pg.1316-1320, 2001.

3. Hibbard B.M.Hemoglobinopathies In: Principles of Obstetrics Butterworths International Edition, 253-61, 1988.

4. Kelton J.H., Crickshank M. Haematological disoders of pregnancy.Medical Complications during pregnancy 3rd ed. Pg 67-94 1988.

5. El-Shafei A.M., Dhaliwal J.K., Sandhu A.K. Prgnancy in sickle cell disease in Bahrain. Bri. J Obstet. Gynaecol. 99: 101. 1992.

6. Ndugwa C. M. Pregnancy in sickle cell anaemia in Uganda (1971-1980). East Afr. Med. J. 59(5):320, 1982.

7. Aluoch J.R. Sickle Cell Disease in Pregnancy: Principles of Management J.Obstet. Gynecol. East Cent. Afri.6(2):95, 1987.

8. Charache S., Scott J. Management of Sickle Cell Disease in Pregnancy patients. Obstet. Gynecol. 55(4): 407-10, 1980.

CASE NO 3: GRAND MULTIPARITY – LIVE BABY

NAME: G.M D.O.D 15/4/2002

IPNO.:

AGE: 42 years L.M.P. 5/7/2001

D.O.A: 13/4/2002 E.D.D. 12/4/2002

Presenting History

The patient was admitted to the labour ward at 3 p.m. on 13/4/2002 with a history of drainage of liquor and abdominal pain for 3 hours prior to admission.

History of Presenting Complaint

She had noticed a sudden gush of liquor flow down her thighs; this was accompanied by lower abdominal pains which increased in intensity and duration. She had not noticed any vaginal bleeding.

Obstetric and Gynaecology History

She could not recall her menarche. The patient was Para 7+0. All deliveries were spontaneous vertex between 1980 and 1991. In the last pregnancy, she had delivered twins prematurely at 7 months gestation. They all lasted less than 12 hours with baby birth weights ranging between 3.9kg to2.3kg. All children were alive and well. She had been using Depo-Provera for contraception but had stopped attending Family Planning Clinic thinking that she could no longer conceive. Her menses had been irregular for a long time but she did not feel that she required medical attention. Her last menstrual period was on 5/7/2001 giving a gestation by dates of 40 weeks. She had attended antenatal clinic at a Nairobi City Council health centre when she felt quickening at 4½ months. The antenatal period had been uneventful with fundal height corresponding to dates throughout the antenatal period. Antenatal Profile: blood group-A ,Rhesus positive;VDRL and HIV negative;Hb-12.5gm/dl; proteinuria-nil throughout the antenatal period. She had been normotensive.

Past Medical History

This was not significant.

Family and Social History.

She was a housewife and her husband was a smale scale farmer on the outskirts of Nairobi. There was no history of any chronic illness. She did not smoke cigarettes nor take alcoholic drinks.

Physical Examination

Her general condition was fair. She was not pale and pulse was regular with a good volume. The blood pressure was 110/80 and she had no oedema.

Abdominal examination

The abdomen was uniformly distented, pendulus with striae gravidarum.
She was getting 2 moderate contractions every 10 minutes each lasting about 30 seconds. The fundal height was term, the lie was longitudinal, cephalic presentation with left occipital position. The head was 5/5 up. The foetal heart tones were heard at a rate of 136 per minute and were regular.

Vaginal Examination

The external genitalia was normal. The cervix was soft and was 6cm dilated. The membranes were not felt and she was draining clear liquor. The presenting part was vertex in left occipital anterior position, at station minus 2. There was no caput or moulding and the cord was not felt. There was show on the examination fingers.

Respiratory, cardiovascular and Central Nervous Systems

These were essentially normal.

Diagnosis

A diagnosis of 42 year old grand multipara in active phase of labour was made.

Management

A partogram was started to monitor the progress of her labour. Four hours later, she was reviewed again. She now had three moderate contractions lasting about 30 seconds. The head was 3/5 up and the foetal heart rate was 144 beats per minute and regular. Per vagina, the cervix was still 6cm dilated and there was no caput or moulding. The liquor was still clear. An impression of poor progress of labour was made; she was started on

intravenous oxytocin drip 2.5 units in 500 mls 5% dextrose starting at 10 drops per minute; this was escalated by 10 drops every ½ hours; at 30 drops per minute, she had attained 3 strong uterine contractions every 10 minutes each lasting about 40 seconds. She was closely monitored and the oxytocin flow rate was maintained at 30 drops per minute. During the intrapartum period, the pulse rate remained around 86 beats/minute, the contractions remained 3 in 10 minutes, and there was no bundle's ring or bleeding per vagina. Blood for crossmatching was taken. The foetal heart rate remained at about 140 beats/minute with physiological deceleration during each contraction, recovering within 20 seconds after the contraction. Labour progressed well and at 11 p.m. the cervix was fully dilated. She was transferred to the delivery room. She delivered a female infant who weighed 3300g; the APGAR scores were 8 and 10 at 1 minute and 5 minutes respectively. The placenta was delivered complete by controlled cord traction; it weighed 400g. The estimated blood loss was 150mls. The uterus was well contracted and the vagina and the perineum were intact. She was then transferred to the resting room where syntocinon was continued for 4hours, during which period the uterus remained well contracted and she had changed only one vaginal pad. She was then taken to the postnatal ward.

She was counseled on family planning and at this time she revealed that she had already opted and signed for sterilization. Tubal ligation was done the following morning by minilaporatomy then she was discharged home. The patient was given an appointment to be seen in the postnatal clinic after one week but opted to seen at the nearest health centre.

COMMENT

Obstetric complications associated with high parity were first pointed out by Solomons in 1934. He reported that from the fifth pregnancy, the rates of maternal mortality and morbidity increase steadily until, for those bearing 10 children or more, these rates are as many as five times higher than all other child bearing women [1].

The term grandmultipara commonly, and in our setup designates a woman who has had 5 or more viable pregnancies[3,9,10]. Earlier, it had been used for those who had 8 or more viable pregnancies[2]. The patient presented had 7 viable pregnancies prior to the present delivery. In modern Israel, where the national policy, religious and cultural norms encourage big families, the term grandmultipara has been used to designate 7 or more viable pregnancies[7,8].

In Kenya high fertility rates persists despite the natural family planning program initiated in 1967[2]. Moreover about 60% of all births take place at home[2]. The situation in Sudan has been described that grandmultipara usually seen in the maternity ward is the one who presents to hospital in the event of serious complication[5]. The patient presented here had attended the antenatal clinic since the time of quickening and presented to hospital on time after spontancous rupture of membranes at home.

Antenatal complications that occur more commonly in the grandmultipara, than in women of lower parity include:

(i) Iron deficiency anaemia despite iron supplementation during the antenatal period[2,3,4,10].

(ii) Medical disorders such as hypertensive disease of pregnancy and diabetes mellitus[3,4,5,8,10].

(iii) Antepartum haemorrhage from both abruptio placenta and placenta praevia [3,4,5,9,10]

The patient presented had none of these complications. During labour malpresentations, malpositions (such as occipitio-posterior positions) and sublaxation of 5th lumbar vertebra predispose to poor perinatal outcome and operative deliveries; these increase the rates of morbidity and mortality in the postpartum period[3,5]. Postpartum haemorrhage due to uterine inertia or complicating operative deliveries result in increased need for blood transfusion [3,5,6]. Induction and augmentation of labour with oxytocin has been a

controversial subject in these patients, however, oxytocin if needed should be used judiciously[3,9] Augumentation was done in this patient with no adverse effects.

Perinatal outcome is dependent mainly on antepartum complications in the mother. Early intervention due to medical complications is associated with increased rates of preterm deliveries and hence poor perinatal outcome[3,5,9]. Moreover, the incidence of multiple pregnancy also increase the poor perinatal outcome[4]. This patient had delivered twins in her seventh pregnancy at 7 months gestation and both had survived. In situations where family planning is accessible, obstetric and neonatal services well distributed and general socio-economic status fairly good, management of grandmultipara has improved. In our setup, these patients are taken as high risk and are followed up regularly in the high risk antenatal clinics. If this is not possible in peripheral health facilities, these patients are referred to high level of obstetric care.

The patient presented here was followed up in a health centre; has had a fairly normal antenatal period. During antenatal care, such patients should be well counseled on the need for permanent or long term contraception. This patient had decided and consented to undergo tubal ligation; this was done the day after delivery.

REFERENCES

1. Solomon B.The Dangerous Multipara.
   Lancet, 2:8, 1934.

2. Singinda 1.The persistence of high fertility in Kenya.
   Soc. Sci. Med., 20:71, 1985.

3. Baskett T.F.Granmultiparity – A continuing threat: A 6 year review.
   Can. Med Ass. J., 116:1001-4, 1977.

4. Chang A.The Obstetric Performance of the Grandmultipara.
   Med. J. austr., 1:330-2, 1977

5. Aziz F.A.Pregnancy and Labour of Grandmultiparaous Sudanese women.
   Int. J. Gynaecol. Obstet., 18:144-146, 1980.

6. Arulkumaran S.The effect of grantmultiparity on labour. Gynaecol., 91:843-848, 1984.

7. Fruchs K.The "Grandmultipara" is it a problem? A review of 5785 cases.
   Int. j. Gynaecol. Obstet., 23:321-25.

8. Seidman D.S.Does Grandmultiparity affect Foetal outcome?
Int. J. Gynaecol. Obstet., 25:1-7, 1987.

9. Tanbo T.G. The Grandmultipara-Maternal and Neonatal Complications.
Acta Obstet. Gynaecol. Scadin., 66:53-6.1997.

10. Mwambingu F.T.The problem of Grandmultiparity in Current Obstetric Practice.
Int. J. Gynaecol. Obstet. 355-9, 1988.

CASE NO 4: POST TERM PREGNANCY – INDUCTION OF LABOUR - LIVE BABY

NAME:	F.A.	D.O.A	4/2/2002
AGE:	20 years	D.O.D	6/2/2002
IPNO:	0819012	PARITY	0+0

Presenting History

She was admitted through casualty as a referral from a private clinic with postdatism. Her last menstrual period was on 14/4/2001 and her expected date of delivery was on 21/1/2002 thus the gestation maturity was 42 weeks by dates. She had her quickening in early September, which extrapolated to 42 weeks or more.

Obstetric and Gynecologic History

She had her menarche at 13 years of age; her menstrual periods occurred regularly every 28 days and the flow lasted 3 days. She had never used contraceptives. She had regularly attended antenatal clinic in a city council clinic since 6 months of gestation; this had been uneventful.Ante natal profile done showed an Hb of 11gm/dl, blood group B positive, VDRL –negative and Elisa for HIV negative. Obstetric ultra sound at 16 weeks compared well with the dates.Quickening was at 20 weeks by dates.

Past Medical History

This was not significant.

Family and Social History

She was a housewife living with her husband in Nairobi. She never smoked nor drank alcohol. There was history of twins in the maternal family. There was no history of chronic illness in the family.

Physical Examination

She was in good general condition, not pale, afebrile, and no oedema. The pulse rate was 76 per minute regular and of good volume. The blood pressure was 140/70mmHg and her temperature was 36°C.

Central Nervous, Respiratory and Cardiovascular Systems

These were essentially normal.

Abdominal Examination

The abdomen was uniformly distended and moved with respiration. The fundal height was corresponding to a term gestation with the fetus in longitudinal lie and cephalic presentation. The head was 4/5 up. The liquor felt scanty, fetal mobility on palpation was minimal and fetal parts were easily palpable. The fetal heart tones were heard at 142 beats per minute and were regular. There were no uterine contractions palpable.

Pelvic Examination

The external genitalia was normal. The cervix was about 2cm long, posteriorly placed, of firm consistency and the os was tightly closed. The fetal head was above the level of the ischial spines. There was no blood or discharge on the examination fingers. The Bishop's score was 0.

Diagnosis

Postdatism with unfavourable Bishop's score.

Investigations

Bio-physical profile:

Single fetus in cephalic presentation; fetal cardiac activity demonstrated and the rate was 144 per minute; normal fetal body tone and breathing movements demonstrated. Quantity of liquor appears adequate.Decreased fetal movements. Cervix appeared normal and the os was closed. Placenta was fundo-posterior with a Resistive index of 0.88 which is high.

Fetal Biometrics

B.P.D.	- 108mm corresponding to 40 weeks plus five days
F.L.	- 72mm corresponding to over 40 weeks
A.C.	- 348mm corresponding to over 40 weeks.
Mean maturity	- Over 40 weeks.
Gestation Age	- Over 40 weeks

Estimated fetal -

Weight - 3900g

Conclusion

1) Post-term fetus

2) Biophysical profile score (BPS) is 8/10 due to decreased fetal movements; there is placental insufficiency.

Management

The condition was explained to the patient and she was planned for induction of labour. The following day, 50ug of cytotec was inserted into the posterior vaginal fornix and the patient kept under close observation; six hours later, a repeat vaginal examination was done and this revealed that the cervix was still posterior, medium consistency, about one cm long and the os was still closed giving a Bishop's score of 2; a second prostaglandin tablet was inserted then. Review done the following day showed that the Bishop's score had now improved, further the cervix was posterior, soft, 0.5cm long and admitting a finger tip giving a Bishop's score of 4. A third tablet was now inserted. A review this time showed that the fetal head was 4/5 up she had weak uterine contractions, and the cervix was 3 to 4cm dilated, soft, mid-position and less than 0.5cm long. Artificial rupture of membranes was done at 8a.m obtaining clear liquor, she was commenced on intravenous oxytocin 5iu in 500ml 5% dextrose starting at 10 drops per minute and escalating half hourly by 10 drops until 3 strong contractions each lasting 40 seconds were attained. Fetal heart tones were observed half hourly while head descent and cervical dilatation was assessed four hourly. She progressed well and at 10p.m, she delivered by vacuum extraction due to delayed second stage to a live male baby who weighed 3750g with an Apgar score of 8 in 1 minute and 10 in 5 minutes. The placenta was delivered by controlled cord traction, weighed 700g and appeared grossly normal. The estimated blood loss was 200mls. She was observed of vital signs and then transferred to the postnatal ward. The baby was reviewed by the neonatologist due to cephlohaemotoma.

Post-partum

The post-partum period was uneventful. The following day, she had started lactating actively, the baby was well and the cephalohaematoma had subsided; her uterus was well

contracted and the lochia loss was normal. The baby was immunized and they were discharges home on the third day for follow up in the postnatal clinic at the nearest health centre after 6weeks.

COMMENT

This was a primigravida who presented with prolonged pregnancy; induction of labour was successfully done and a live baby was delivered. The duration of pregnancy in human beings averages 260 days from conception or 280 days form the first day of the last normal menstrual period of a 28+5 day circle. A pregnancy that exceeds 294 days (42 weeks) from the last menstrual period is said to be post term while postdate pregnancies last longer than 40 weeks.[1] The incidence of prolonged pregnancy is about 10% but generally varies between 3% and 12% worldwide[2]. This is because of the varied available methods of confirming postdatism.

The aetiology of postdatism is largely unknown though certain conditions have been associated with this anomaly. These include anencephaly, absence of fetal pituitary, placental sulphatase deficiency, and fetal adrenal hypoplasia[3]. The patient presented had none of these associated fetal aetiological factors, detected at the time she went home. Women who present with post-dates can be identified into three groups. The first group consists of those whose baby matures slowly thus only reaches maturity at 42 weeks or more. The other group is those whose babies show evidence of postmaturity and are at risk of dying in utero; these are the ones who account for the notable increase in perinatal mortality – that occurs after 42 weeks of gestation. The third group are those who have wrong dates of their last normal menstrual period[1,2]. Features of post maturity are seen only in babies of those in the second group. These features include those resulting from intrauterine malnutrition (scanty sub-cutaneous fat, dry scaly skin) as well as hardening of fetal skull with narrow sutures, small fontanelles, and well developed genitalia[1,2,3]. With prolonged gestation, some fetuses continue to grow and achieve large sizes. The baby delivered by this mother was 3750g and had no grossly notable features of post-maturity.

In making a diagnosis of post-datism, confirmation of gestation age is important; when the menstrual history is a accurately known, this can be calculated using Naegle's rule of adding seven to the first day of the last period and subtracting 3 from (or adding 9 to) the month, in a woman with a 28 day cycle [1,3]. This can be further complemented by the time of quickening, sequential fundal measurements, early pregnancy tests, fetal heart tones and ultrasonography[3]. The value of ultrasonography is stressed because fetal anomaly is looked for and biophysical profile can be done to assess fetal well being as

well evaluate for placental insufficiency. This was done in our patient and features of post maturity confirmed; placental insufficiency was also reported. The patient was sure of her dates and time of quickening hence the diagnosis was made based on the findings. Post dates pregnancy has been associated with increased fetal and neonatal morbidity and mortality. Fetal jeopardy increases progressively after 41 weeks onwards as marked by the higher rate of intrapartum fetal distress and primary caesarean deliveries[2,4]. This fetal compromise is attributed to oligohydrmnios, umbilical cord compression and placental insufficiency. Cord compression acting through vagal mechanisms is thought to stimulate fetal heart rate decelerations and peristalsis; these mechanisms explain the chronic fetal distress and passage of meconium which is of high incidence in post date babies. Oligohydramnios results from decrease in amniotic fluid as pregnancy goes beyond term and is thought to be another sequelae of placental insufficiency. This placental compromise leads to small for gestational age babies with intrapartum fetal distress that necessitates more caesarean delivery. Aging of the fetal bones in utero is associated with reduced moulding of the fetal skull necessary for normal vaginal delivery; difficult labour and traumatic delivery has been noted[4,5].

The patient described had placental insufficiency confirmed by ultasonography (biophyisical profile) there was no meconium staining of liquor but she had a difficult second stage delivery leading to vacuum extraction; a fetal cephalhematoma occurred as a result but the baby was generally well.

Just as the diagnosis of postdatism is controversial, the obstetric management of the condition is equally shrouded in controversy. Some authors advocate elective termination of the pregnancy at 42 weeks to avoid fetal death and post maturity syndrome. Others recommend that since routine induction of labour is associated with increased need for caesarean delivery, such post dates pregnancies should be managed conservatively[2,3,5]. With conservative management and proper anatepartum fetal surveillance, over 30% of such pregnancies will go into spontaneous labour with better outcome than those who are induced[2]. In Kenyatta National Hospital, the practice is to perform induction of labour after 41weeks. Perinatal outcome has to be balanced against the risk of induction of labour where the cervix is unfavourable; studies have reported a high incidence of poor Bishop's score which consequently implies a high incidence of failed induction[6]. Use of intravaginal prostaglandin gel or tablets has been advocated followed by amniotomy and oxytocin augmentation to achieve good results[2,6]. This was

done in our patient and she picked up good contractions; three prostaglandin E2 pessaries had to be used due to a very poor initial cervical score. Those with a favourable score only require amniotomy and oxytocin augmentation.

Recent studies have shown that fetal fibronectin found in the choriodecidual interface is usually found in cervicovaginal secretions 1-2 weeks before delivery. The combination of a negative fibronectin test and unfavourable cervix at 39 weeks gestation may be additive in a patient remaining undelivered at 41 weeks. One study found that serial membrane sweeping in these patients resulted in earlier delivery compared to controls[7]

REFERENCES

1. Alexander J.M., Mc Intire D.D., Leveno K.J. Forty weeks and beyond;Pregnancy outcomes by weeks of gestation. Int. J Obstet Gynaecol 96 (2):291-4, Aug. 2000.

2. Crowley P. Interventions for preventing or improving the outcome of delivery at or beyond term. Cochrane database syst. Rev 2000; CD 000170 (midline)

3. Pernoll M.L. Untimely Termination of Pregnancy. Current Obstetric and Gynecologic Diagnosis and Treatment. Appleton and Lange/Mc Graw HillNew York. pg 309, 2003.

4. Eden R.D., Seifert L.S., Winegar A. Perinatal characteristics of uncomplicated postdate pregnancy. Int. J. Obstet. Gynaecol. 69 :296 (1987).

5. Hannah M.E., Hannah W.J., Hellmann J. Induction of labour as compared with serial antenatal monitoring in post term pregnancy. A randomized controlled trial group. N Engl.J. med 326 (24); 1587-92,1992.

6. Cassis A., Mazor M., Leiberman J.R. at al. Management of postdate Pregnancy; A case control study. Obstet. Gynecol. 78(1): 19, 1991

7. Cunningham E.F., Grant N.F., Levelo K.J. Induction and augmentation of labour. Williams Obstetrics 21st Ed. Mc Craw-Hill publishers pg 469-481 (2001).

CASE NO 5: ADOLESCENT PREGNANCY – VAGINAL DELIVERY – LIVE BABY

NAME:	P.M	PARITY	0+0
AGE:	15years	D.O.A	22/03/2002
IPNO:	0819777	D.O.D.	23/03/2002

Presenting Complaints

She complained of labour pains for six hours prior to admission.

History of Presenting Complaint

She had been well till the morning of admission date when she developed low abdominal and back pains of increasing intensity and frequency; there was no associated vaginal bleeding or drainage of liquor. When the pains intensified, she requested neighbours to accompany her to hospital where she was seen and admitted to the labour ward through casualty.

Obstetric and Gynecologic History

She was a primigravida who had her menarche at 13 years of age; her monthly periods occurred every 28 days with a flow lasting four days associated with dysmenorrhea and the volume was normal. Her last menstrual period was on 28/6/01 and the expected date of delivery was on 5/4/02 giving a gestation of 38+ weeks by the time of admission. She had attended antenatal care four times since 7 months gestation at Waithaka City Council Clinic, which was uneventful. She never used any contraceptives.

Past Medical History

This was not significant

Family and Social History

She was a single lady, first born in a family of three children all alive and well. She dropped out of primary school for lack of school fees and was currently unemployed.
Her mother, who died in August 1997 of Tuberculosis of the spine, had been separated from her father. She neither smoked cigarettes nor took alcohol.

Examination

She was a young lady in fair general condition, not pale, jaundiced or oedematous; she had no lympadenopathy. Her BP was 100/60mmHg, respiratory rate 24 per minute, pulse rate 82 per minute and temperature 36.4°C.

Central Nervous, Cardiovascular and Respiratory Systems

These were essentially normal.

Abdomen Examination

The abdomen was uniformly distended with a fungal height corresponding to a term gestation; the lie was longitudinal with the fetus in cephalic presentation and the head was 2/5 up. The fetal heart was heard and was 136 beats per minute and regular. She was having three moderate contractions every 10 minutes each lasting 20 to 40 secondas.

Pelvic Examination

The external genitalia was normal; the cervix was anterior, soft, effaced and was 6cm dilated; amniotomy was done obtaining clear liquor. There was no caput or moulding and no umbilical cord was felt. The pelvis was clinically adequate. Show was seen on the examination fingers.

Diagnosis

Active labour in primigravid adolescent.

Management

The diagnosis of active labour was explained to the patient and also the plan of management; consent for surgery was also obtained from the accompanying adults in case of necessity for surgical intervention. She was started on routine partograph where the number and nature of uterine contractions in 10 minutes were charted; fetal heart rate recorded half hourly and vaginal examination done 4 hourly. She progressed uneventfully and was due for review 4 hours after initial assessment. During the next

review, the cervix was found to be fully dilated and the head crowning. She was taken to the delivery room where a live male baby was delivered weighing 3200g and had an Apgar score of 8 in one minute and 10 in five minutes; the placenta weighed 450g and blood loss was estimated to be 100mls. The cervix and vulva were inspected and found intact and the episiotomy was repaired. Post delivery observations were normal; she was allowed to rest in the delivery room after which she was taken to the post-natal ward.

The following morning, the patient had no complaints, the breasts were lactating, the uterus was contracted to 16 weeks size and the lochia loss was normal. The baby was actively breastfeeding. Both mother and baby being well, they were discharged home and advised to attend the adolescent counselling clinic after one week and to take the baby to the nearest health facility for immunization.

Follow up

She was seen in the adolescent clinic and counselling was done. She chose to come back after 6 weeks for Norplant insertion as a method of contraception.

COMMENT

This patient was a 15 year adolescent who presented at term in active labour ending with a normal vaginal delivery. The 1974 World Health Organization (WHO) meeting on pregnancy and abortion in adolescence defined adolescence as the period during which:

(i) The individual progresses from the point of appearance of secondary sexual characteristics to sexual maturity.

(ii) The psychological processes and patterns of identity develop from those of a child to those of an adult.

(iii) A transition is made from a state of total socio-economic dependence to one of relative independence[1].

Throughout the world, pregnancy and childbearing are increasingly occurring among adolescents than in the past. These are school age girls not psychologically and socially

prepared for childbearing and motherhood. In Kenya, up to 10,000 school girls have been reported to drop out of school each year due to pregnancy which is acquired in unwanted circumstances; up to 16% are actually expelled from schools while only 19% of all ever resume their education[2]. Generally the incidence of adolescent pregnancy countrywide ranges from 11-19% in urban areas and 10% in rural areas. Muraya[3] found an incidence of 21% in a rural area in Kenya.

Generally, pregnancy at either extremes of reproductive life increases the risks of maternal mobirdity and mortality, the risks being lowest at 20 and 30 years age group. Socioeconomic factors play a big role in adolescent pregnancy as they affect biological development and general behaviour among young girls. Those factors that determine increased fertility include general sexuality of the contemporary society, early sexual maturation, breakdown in cultural bonds, lack of parental guidance and peer pressure[2]. The age of menarche among Kenyan girls has been noted at a mean of 13+2.69 years[4] which parallels that in other parts of the world. The patient presented here had her menarche at 13 years of age; she had grown up under care of a single mother, separated from her husband. Her mother eventually succumbed to a chronic illness leaving her and other siblings under no parental care. Such socioeconomic circumstances are typical fertile grounds for adolescent pregnancy; this patient is under no parental guidance and has to fend for herself. Poor education is typical of pregnant adolescents; they either never went to school or dropped out of school when young. This patient dropped out of primary school and proceeded to seek for employement. Muraya[3] found 53.3% married adolescents. The patient presented here was not married and had no gainful means of livelihood. Adolescent pregnancy is a focal area in obstetrics and gynecology due to its medical and social implications. Poor education leads to ignorance; this together with societal stigma leads to poor antenatal care[5].This is collaborated by a study in Nigeria which showed that the poor obstetric outcome of teenage pregnancies is related to non-utilisation of prenatal care rather than their biological age[7] . Our patient had attended antenatal care four times form 28 weeks of gestation, which was not adequate. Anemia in pregnancy is also common due to poor dietary habits. Ngoka[5] reported between 11.8% and 42.9% of some patients with some degree of anemia. Our patient was not clinically anaemic though hemoglobin estimation was not done.

Pregnancy itself is associated with certain complications; notable among these are first and third trimester bleeding, pre-eclampsia and eclampsia, and premature rupture of

membranes. Muraya[3] reported hypertensive disease of pregnancy rate of 2.5% among teenagers compared to 0.5% among the women above 20 years of age. This patient did not develop any of these pregnancy related complications. Pregnancy outcome varies widely; fetal wastage is rampant among the adolescents due to the fact that the pregnancy is unwanted. In Kenyatta National Hospital, up to 18% of abortions have been noted to be in the 12 to 19 years age group with 76% of them being unwanted pregnancies[6]. There is an increased incidence of low birth weight, stillbirths and perinatal mortality due to higher rates of preterm labour and delivery. It has been noted that labour related complications are common[1]. Ngoka[5] noted a higher rate of caeserian section and a lower rate of vaginal delivery than in the general population.

The patient had no pregnancy or delivery related complications ending up with a normal baby weighing 3200g with good Apgar Score. Preventive measures to curb the increasing rate of adolescent pregnancy are surrounded by political, social, legal and religious controversies. Fear of contraception abounds among parents in the pretext that it will promote promiscuity and erode morality; the Catholic Church outrightly discourages contraception among its followers while not providing practical solutions to the menace. There are divided opinions among politicians, teachers and society at large on the role of teaching sex education in schools such that the issue remains unsolved. Parental guidance is a missing necessity in most families due to changing lifestyles, lack of role models and disintegration of cohesiveness of families. The legal aspects which bar legalisation of abortion should equally be revised probably to make abortion not legal as such but to decriminalize it. This will ensure morbidity and mortality associated with it is reduced as most of the girls will seek termination from qualified health providers. Antenatal care should delink adolescents form routine clinics providing adolescents clinics which will be more acceptable and improve attendance; this is already in practice in Kenyatta National Hospital.

REFERENCES

1. World Health Organisaiton, Pregnancy and Abortion in Adolescence: A report of WHO meeting Wld. Health. Org. Rep. 583, 27, 1975.

2. Mati J.K.G., Sanghvi H.C.G.Outcome of Pregnancy in Teenage Mothers in Nairobi Birth Survey. J. Obstet. Gyncol. E. Cent. Afri., 2:134, 1983.

3. Muraya G.N. Teenage Pregnancy in Rural Kenya. M. Med. Thesis, University of Nairobi, 1985.

4. Rogo K.O., Oniango R.K., Muruli R.A. Menarche in African Secondary School Girls in Kenya E. Afri. Med. J., 64(8), 511. 1987.

5. Ngoka W.M., Mati J.K.G. Obstetric aspects of Adolescent Pregnancy E.Afr. Med. J., 57(2) 124, 1980.

6. Omuga B.O.O.Presenting of Abortion and its Preventive Problems at Kenyatta National Hospital. M.Med. Dissertation, University of Nairobi, 1988.

7. Loto O. M., Ezechi O.C., Kalu B. K. et. al. Poor obstetric performance of teenagers: is it age or quality of care–related? J. Obstet. Gynaecol. 24(4)395-8, 2004.

8. Estambale B., Njagi J.K., Magnussen P. et. al. Prevention of anaemia in pregnancy using insecticide treated bednets (ITN) and sulfadoxine-pyrimethamine (SP) in a highly malarious area of Kenya: a randomized trial. Trans. R. Soc. Trop. Med. Hyg.97(3):277-82. 2003

CASE NO. 6 ABRUPTIO PLACENTA WITH FOETAL DISTRESS: EMERGENCY CAESAREAN SECTION LIVE BABY

NAME: P.A. PARITY 3+0

AGE: 28 D.O.A 9/4/02

IPNO: 0819600 D.O.D 17/4/02

Presenting Complaint

The patient was admitted to the labour ward with a 3 hour history of per vaginal bleeding. Vaginal bleeding was spontaneous in onset; she had changed blood soaked pads twice before she came to hospital. The bleeding was accompanied by lower abdominal pain but no drainage of liquor. There was no history of spotting during this pregnancy.

Obstetric and Gynaecologic History

She attained her menarche at the age of 16 years; her periods were regular lasting 4 days in a 28 cycle. Her last menstrual period was on 12/8/2001 and the expected date of delivery was on 19/5/2002 thus, she was at a gestation of 34 weeks. She had attended antenatal care at a private clinic during which period she had no problems. VDRL was negative, blood group A Rhesus positive and Hb 11gm/dl. She was not screened for HIV. All her previous three deliveries were spontaneous vertex and the babies were alive and well. Her last delivery was in 1996. She had used the intrauterine contraceptive device for between 1996 and 2000.

Past Medical History

She had no history of major illness.

Family and Social History

She was married and worked as a clerk. She did not smoke cigarettes or take alcoholic drinks. There was no family history of major chronic illnesses, nor history of twins.

Physical Examination

The patient was a young lady in good general condition. She was not pale or febrile. There was slight pedal oedema. The blood pressure was 120/80mmHg, the pulse rate was 84 per minute and the respiratory rate was 22 per minute.

Abdominal Examination

The abdomen was uniformly distended and the fundus was corresponding to a 36 weeks gestation. The uterus was not tonic but had marked tenderness in the fundal region. She was getting mild contractions at a rate of 2 every 10 minutes each lasting about 20 seconds. The lie was longitudinal with cephalic presentation and the head was 4/5 up. The fetal heart rate was 114 per minute and was irregular.

Cardiovascular, Respiratory and Central Nervous Systems.

These were within normal limits.

Speculum Examination

The external genitalia was normal. The external cervical os was 3cm open and there was blood from within the cervical canal. There were no clots in the vagina and she was not draining liquour.

Digital examination was deferred.

Diagnosis

A diagnosis of anterpartum haemorrhage at 34 weeks gestation with premature labour and foetal distress was made.

Management

She was started on intravenous fluids of 500mls 5% dextrose; blood was taken for grouping and cross-matching and two units were requested for. The diagnosis was explained to her and consent was obtained from her for examination under anaesthesia (EUA) and possibly caesarean section. She was nursed in the left lateral position and

oxygen was administered by mask. She was shaved then premidicated with intramuscular atropine 0.6mg stat then wheeled to theatre.

In theatre, she was put under general anaesthesia and placed in lithotimy position. The vulva and perineum were cleaned and draped. The bladder was aseptically catheterized of clear urine.She was repositioned in supine position and the abdomen cleaned and draped. Through a Pfannenstiel incision, the abdomen was opened in layers. The lower segment was identified and the uterovesical peritoneum incised. After retracting the bladder anteriorly a lower segment caesarean section was done. The liquor was found to be meconium stained grade II. A female infant weighing 2300g was delivered and the APGAR score were 8 and 10 in 1 and 5 minutes respectively. The placenta was fundal and was manually extracted. It weighed 350g. There was a large retroplacental clot of about 400mls in volume. The total estimated blood loss was 800mls.

Post-operatively she was observed routinely as described elsewhere in this book. The check haemoglobin was done in the third post-operative day and it was 10.2g/dl. She had a normal post-operative period and was discharged home on the 4th day to be seen at the postnatal clinic in 6 weeks.

Follow-up

She was seen at the postnatal clinic as scheduled. She was not pale and the uterus was fully involuted. The baby was breast feeding well and continuing with immunization. She was referred to the Family Planning Clinic for contraception and advised to come to the antenatal clinic in a subsequent pregnancy.

COMMENT

The patient presented was a 28 year old, Para3+0 who presented with abruptio placenta at 34 weeks gestation. She was in early premature labour; foetal distress was diagnosed by an irregular fetal heart rate. She was therefore delivered by emergency caesarean section with good fetal outcome.

Abruptio placenta occurs when a normally implanted placenta separates from its uterine attachment before delivery of the baby. This happens in about 1% of deliveries and in about 60% of cases, it leads to preterm delivery. It is associated with high rates of

maternal morbidity and mortality as well as perinatal deaths[1,2,3,4,5,6,7]. It is postulated that defective placental vasculature is a primary causative factor in abruptio placenta. Conditions in which the incidence of abruptio placenta is increased include maternal trauma, sudden uterine decompression (as following rupture of membranes in polyhydramnios), hypertensive disease, maternal folate deficiency, high parity and advanced maternal age. Unusually short umbilical cord had been associated with placental abruption. Patients with a previous history of abruptio placenta are at higher risk in a subsequent pregnancy of nearly 10% recurrence[1,2,4,5].

Cigarette smoking and alcohol ingestion have also been implicated[1,5]. Primary risk factors are absent in about 40% of the cases. Chorioamnionitis has been reported to be a contributing factor where no other cause is found[2]. The patient presented had none of these identifiable factors. Folate deficiency or cord abnormality was however not looked for and neither was chorioamnionitis detected.

Abruptio placenta can be concealed or revealed. When bleeding occurs per vagina, it is revealed haemorrhage. It is concealed in about 20% of the cases. It is also classified in grades 0-3: [4]

Grade 0- The patient is asymptomatic but a retroplacental clot is noted after delivery.

Grade 1- The patient has vaginal bleeding along with uterine tenderness. Neither mother nor baby shows any signs of distress.

Grade 2- The patient experiences uterine tenderness and tetany with or without external evidence of bleeding. The mother is not in shock but there is some evidence of fetal distress.

Grade3- Uterine tetany is severe; the mother is in haemorrhagic shock (>1000mls) The bleeding may or may not be revealed and the foetus is probably dead.

The patient presented had grade 2 placenta abruption with revealed haemorrhage. When it is concealed, the uterine size may be bigger than dates; severe haemorrhage in such cases may occur leading to major maternal morbidity or mortality and almost certain foetal death[1,3,4,5,7].

Clinical presentation includes vaginal bleeding, uterine irritability and hypertonicity, uterine tenderness and back pain. Abnormalities of fetal heart rate (late deceleration or bradycardia) usually occur[1,2,3,4,5]. The patient frequently goes into spontanieous labour due to uterine irritability. Hemodynamic changes that occur result in various degrees of

shock and are a good indication of maternal blood loss; this may not be obvious in cases of concealed haemorrhage and the patient may only present with preterm labour or fetal distress whose cause may not be clear[2]. The patient presented had preterm labour and fetal heart irregularity; she had mild uterine tenderness. Her hemodynamics were well withing normal.

In the management of these patients good history supplemented by the clinical picture will usually lead to diagnosis of abruptio placenta. Early diagnosis is vital for good maternal and fetal outcome as happened in the patient presented here. Intravenous fluids are instituted to expand plasma volume; blood is taken for grouping and cross-matching of fresh blood. A packed cell volume (PCV) and coagulation studies should be done; and increase in fibrinogen degradation products (FDPs) is highly suggestive of placental abruption[1,4,5]. An obstetric scan will help in confirming the diagnosis and rule out placenta praevia. Using real time ultrasonography, severe abruptio is diagnosed by separation seen around the placental edges, placental thickness beyond 5.5 cm and bulging to assume a spherical shape. A negative scan does not rule out abruptio especially where haemorrhage is revealed[1,6]. These confirmatory tests were not done in our patient. Active management includes urgent delivery of the patient after adequate resusciatation. At Kenyatta National Hospital, examination under anaesthesia is done to rule out placenta praevia. The decision on vaginal or abdominal delivery is determined by fetal viability, severity of the abruptio and the condition of the mother. Expectant management has been done in cases where the abruptio is confirmed to be mild, the mother is hemodynamically stable and there is not evidence of fetal compromise[6]. These patients require very close monitoring with daily repeat scanning and twice weekly non-stress test hence it should be done where adequate facilities are available.

Where the mother is hemodynamically stable, the cervix is inducible, no fetal comprise exists and there is no contraindications to vaginal delivery, amniotomy is done and oxytocin augumentation will suffice. This is also recommended when the fetus is found to be dead. Caesarean delivery must be considered in all cases where the fetus is still alive to save the fetus from further compromise. Individualization of patient management is however paramount[1,2,5,6]. Maternal complications of abruptio placenta include shock which is life threatening and must be vigorously treated with intravenous fluids and massive blood transfusion to guard against renal complications. Urine output must be monitored to ensure at least 30mls per hour. Coagulation failure and acute

pituitary necrosis may also occur with serious consequences[1,2,3,5,7]. In the post partum period, anaemia with concurrent poor wound healing and infection are not unusual [3]. Rhesus isoimmunisation may occur in a rhesus negative mother.

The patient presented was delivered by caesarean section; she was hemodynamically stable but had evident fetal distress. She did not develop any postnatal complications and recovered well from the operation. Perinatal morbidity and mortality is usually high even in fully equipped centers. Timely delivery and good neonatal care improves fetal outcome[2,7]. This patient had timely intervention with good fetal outcome. Although fetal distress was noted on admission, the APGAR score was good and no emergency neonatal unit admission was warranted.

Maternal mortality is usually as a result of late patient presentation, poor resuscitation measures or to renal and coagulation complications that follow. All women must be educated on detection of antepartum haemorrhage especially those who have the associated risk factors. They should be encouraged to attend clinic early and report to hospital any time they notice abnormal vaginal bleeding especially in late pregnancy.

REFERENCES

1. Nimrod C.A, Oppenheimer L.W. Third trimester haemorrhage. Medicine of the Foetus and Mother. 2nd Ed. Lippincott-Raven publishers pg 1497-1505 (1999).

2. Claydon C.S., Martin L., Pernoll. Third trimester vaginal bleeding. Current Obstetric and Gynaecologic Diagnosis and Treatment,9th Ed. Appleton and Lange , McGraw Hill New York, pg 357-362, 2003.

3. Cunningham F.G., MacDonald P.C., Grant N.F. Obstetric haemorrhage. Williams Obstetrics, 21st Ed. Appleton and Lange McGraw Hill New York, pg 621-630, 2001.

4. Nancy J. Collins. Abruptio placenta.Home medical education. Press Nursing ceu. Com.

5. Niswander K.R. Third Timester Bleeding In: Manual of Obstetric 3rd ed. 1989.

6. Riviera-Asma M.E. The use of Ultrasound in the Management of Abruption Placenta. Am. J. Obstet. Gynaecol. 146:924, 1983.

7. Barren S.L. Abruptio placenta. Turnbull's Obstetrics 2nd Ed. Churchill Livingstone publishers pg 319-325 1996

CASE NO.7; DELAYED SECOND STAGE OF LABOUR; VACUUM EXTRACTION – LIVE BABY

NAME:	J.N	PARITY:	0+0
AGE:	18 Years	D.O.A:	15/4/2002
IP NO:	0819610	D.O.D:	17/4/2002

Presenting Complaints

The patient was admitted to the labour ward through the teenage antenatal clinic with history of labour pains for about 12 hours.

History Presenting Illness

The patient attained her menarche at the age of 13 years. Her periods were regular and lasted for 3 days with a cycle of 30 days. She was a primigravida. Her last menstrual period was on 1/7/01 and the expected date of delivery was on 8/4/02. She was at a gestation of 41 weeks at the time of admission. She had attended the teenage antenatal clinic at Kenyatta National Hospital and seen for a total of 12 times, since the gestation period of 16 weeks. The antenatal period had been uneventful. Investigations done at the clinic included:

Haemoglobin	-	12.2g/dl
Blood group	-	"O" Rhesus Positive
VDRL	-	Negative

At 36 weeks, clinical pelvic assessment had been done. The external genitalia was normal. The sacral promontory was not tipped, the ischial spines were not prominent, and the intertuberous distance could accommodate four knuckles. Thus, the pelvis was clinically adequate for normal vaginal delivery.

Past Medical History

This was not significant.

Family and Social History

She was single and unemployed; she lived with her parents in Nairobi. She had attended school up to form four. She was the second born in a family of four; other siblings were alive and well. There was no family history of chronic illness or twins. She did not take alcohol or smoke cigarettes.

Physical Examination

The patient was in good general condition; she had no pallor, jaundice or oedema. Her pulse rate was 80 per minute, the blood pressure was 110/60mmHg, and she was afebrile.

Abdominal Examination

The abdomen was uniformly distended, the fundal height was term, and the lie was longitudinal with cephalic presentation. The head was 4/5 up. The foetal heart was heard at a rate of a 148 per minute and was regular. She was getting 2 moderate contractions in 10 minutes each lasting 30 seconds.

Respiratory, cardiovascular and Central Nervous Systems

These were essentially normal.

Vaginal Examination

The external genitalia was normal. The cervix was 50% effaced, in the mid position, soft and 4cm dilated. The umbilical cord was not felt. Artificial rupture of foetal membranes was done and clear liquor was obtained. The foetal head was in left occiput anterior position and the pelvis was adequate.

Diagnosis

An impression of primigravida at 41 weeks gestation in active labour was made.

Management

She was admitted to the first stage of labour at 3cm cervical dilatation and a partogram was started to chart the progress of the labour. She was reviewed four hours later; at this time, she was getting 3 strong contractions every 10 minutes each lasting 40 seconds. The foetal heart rate was regular at 144 beats per minute. The head was 3/5 up. Vaginal examination revealed that the cervix was now fully effaced, and was 7cm dilated. The liquor was clear. There was no caput or moulding. She was to continue and to be reviewed after 3 hours.

In the next review, she had three very strong contractions, the foetal heart was 148 per minute with irregularity at the peak of a uterine contraction but returning to normal after the contraction waned off. The foetal head was now 1/5 up and she expressed the urge to

bear down with each contraction. Vaginal examination revealed that the cervix was fully dilated thus, in second stage of labour. She was transferred to the delivery room. In the delivery room, she was put in dorsal lithotomy position, vulvo-vagina toilet was done and she was draped. She was encouraged to bear down with each uterine contraction but the head remained high and would not distend the vulva. After several such efforts, the patient was noted to be exhausted and had poor effort with successive uterine contractions. An intravenous drip of 500mls 5% dextrose was started while a vacuum extractor was prepared to expedite the delivery. She was now put in full lithotomy position and the bladder was catheterized. Twenty milliliters of 2% lignocaine was infiltrated into the left posterolateral aspect of the perineum and a mediolateral episiotomy was made. A 50mm cap was applied to the occiput. Negative pressures of $0.2kg/cm^2$ were created till $0.8kg/cm^2$ was attained. Intermittent traction perpendicular to the cup surface was applied at the height of the uterine contraction. With the second application of traction, the head descended, distended the perineum and was delivered. The pressure was slowly released and the cup came of the head. The nose and the mouth were wiped clean with gauze. Intramuscular ergometrine 0.5mg was given on the birth of the anterior shoulder. The delivery of the rest of the body soon followed then the cord was double clamped and cut. The baby was a male infant who weighed 3000g and had an APGAR score of 8 and 9 at 5 and 10 minutes respectively.

The placenta was delivered by controlled cord traction; it was complete, healthy and weighed 400g. The uterus was well contracted. Repair of the episiotomy was done using chronic catgut number 2/0. The estimated blood loss was 300mls. During the immediate postpartum period she was taught how to care for the episiotomy and how to breast feed her baby. She was discharged home on the second post delivery day to be seen in the postnatal clinic after 6 weeks. She did not turn up as scheduled and was lost to follow up.

COMMENTS

The patient presented was a primigravida who had good progress of labour but got a delayed second stage due to poor maternal effort. Assisted vaginal delivery by vacuum extraction was done with good foetal outcome.

Vacuum extraction is the method mainly utilized at Kenyatta National Hospital for operative vaginal delivery. Obstetric forceps are not used. The common vacuum extractor consists of a specially designed metal cup whose diameter at the rim is smaller than at the surface. The cups are in 3 sizes of diameters 40mm, 50mm and 60mm. A hose pipe is connected to the cup and a chain leading to a crossbar is threaded. This is connected by another hose pipe to a trap bottle to which a monometer is attached[1,3]. This is the original Malmstrom's vacuum extractor. Modifications of this apparatus have been made; Birds modifications have the chain permanently fixed onto the surface of the cup and the hose pipe is attached to a different point[3]. Soft plastic cups have also been introduced which simplifies the procedure as well as reducing trauma to both mother and baby because the cup is malleable and foetal scalp is not sucked in[4,5]. At Kenyatta National Hospital both plastic and metal vacuum extractor are in use. The Malmstrom's apparatus was used in the patient presented.

The main indication for vacuum extraction is to expedite the second stage of labour. Maternal indication for this includes cardiac disease, hypertensive disease, uterine inertia and maternal exhaustion. Foetal indications include foetal distress and malposition when the patient is in second stage. Rarely, it may be used to rotate the fetus from an occipito-transverse or occipito posterior position to the anterior position. Traction alone may sometimes lead to autorotation[1,2]. In this patient there was maternal exhaustion and a high presenting part. During the procedure, a generous mediolateral episiotomy is recommended; the largest and easiest fitting cup is the one applied on the occiput in such a way that the sagittal and lambdoidal sutures are symmetrical at the rim of the cup. Care should be taken not to include vaginal and cervical tissue in the cup; the cup should never be applied on the anterior fontanelle. Increasing the negative pressures gradually ensures even and complete filling of the cup with foetal scalp hence making it less likely for it to slip off during traction[1,2,3,7].

Traction is made intermittently and coincidentally with uterine contractions. This should be done perpendicular to the cup to avoid slipping off. The cup should not be applied for

more than 30 minutes and the procedure should be abandoned if there is no descent of head after three tractions to avoid cepholohematomas, intracranial haemorrhage and skull fractures[1,2,7,8]. These complications need not occur if vacuum extraction is carefully done on selected patients. It is contraindicated in non-vertex presentations, prematurity and cephalopelvic disproportion[1,2]. No immediate foetal and maternal complications were noted in the patient presented here. The procedure ideally should be performed where delivery by caesarean section is possible without delay as the latter is generally associated with poor neontatal outcome[5,7,9].

Application of the vacuum extractor is not a commitment to vaginal delivery; the test of success is the outcome of both mother and baby. Failure is said to occur when avoidable injury had been inflicted [5,9].

REFERENCES

1. Ralph W., Hale R. W. Operative Delivery In: Current Obstetric and Gynaecology Diagnosis and Treatment. 9th Ed. Appleton and Lange/McGraw Hill New York Pg 543, 2003.

2. Cunningham F.G., MacDonald P.C., Grant N.F. Vacuum extractor. Williams Obstetrics, 21st Ed. Appleton and Lange/McGraw Hill New York pg 490-492 (2001)

3. Bird G.C. Modification of Malmstrom's Extractor. Br. Med. J., 3:526, 1969.

4. Berkus M.D. Cohort Study of Silastic Obstetric Cup Delivery: Safety of the Instrument. Obstet. Gynaecol., 66:503, 1985.

5. Combs C.A. Risk factors for Third and Fourth Degree Perineal Lacerations in Forceps and Vacuum deliveries. Am. J. Obstet. Gynaecol., 163:100-4, 1990.

6. Lowe B. Fear of Failure: A place for Trial of Instrument Delivery. Br. Obstet. Gynaecol., 94:60-6, 1987.

7. Roberton P.A. Neonatal and Maternal Outcome in low Pelvic and Midline Operative Deliveries. Am J. Obstet. Gynaecol., 162:1436-42, 1990.

8. Ritchie J.W.A. Obstetric Operations and Procedures in: Dewhurst's Textbook of Obstetrics and Gynaecology for Post graduates 4th ed. Blackwell Scientific Publications, 428, 1988.

9. Chamberlain G. Operative vaginal delivery. Turnbull's Obstetrics, 2nd Ed. Churchill Livingstone publishers pg 695-715 (1996)

CASE NO 8: RETAINED PLACENTA MANUAL REMOVAL

NAME:	J.A	PARITY	1+0
AGE:	22	D.O.A	27/7/02
IP NO:	0820130	D.O.D	28/7/02

Presenting Complaints

This patient was referred from a City Council Clinic with a diagnosis of retained placenta.

History of Presenting Complaints.

She had developed labour pains one day prior to admission; she delivered on arrival at the City Council Clinic, a normal baby who cried immediately after birth. Attemps to deliver the placenta by controlled cord traction. The time elapsed from delivery to the arrival at Kenyatta National Hospital labour was about 3 hours.

Obstetric and Gynaecologic History

The patient was a para 1+0 following this delivery. Her menarche was at the age of 14 years following which her periods were regular every 30 days and lasting for 4 days. Her last menstrual period was on 12/10/01 hence her expected date of delivery was on 19/7/02. The gestation at delivery was 41 weeks. She had attended antenatal care in a private clinic in the estates; this had been uneventful. There was no history of contraceptive use.

Past Medical History

This was not significant.

Family and Social History.

She was a married lady and lived with her husband in Nairobi. She did not smoke cigarettes nor take alcohol. There was neither history of chronic illness in her family nor history of twins.

Examination

She was in good general condition. There was no pallor, jaundice or fever. Her pulse rate was 86 per minute, the blood pressure was 110/70mmHg and the respiratory rate was 20 per minute.

Abdominal Examination

The uterus was firm non-tender and the fundal height was corresponding to 24 weeks gestation. The bladder was not full. The liver and the spleen were not palpable.

Respiratory, Cardiovascular and Central Nervous Systems

These were within normal limits.

Vaginal Examination

The external genitalia was normal. The umbilical cord was distally ligated with a string and was dangling freely from the introitus. She was having minimal bleeding. Digital examination revealed the cervix was open and attempts to remove the placenta by controlled cord traction were unsuccessful.

Diagnosis

Retained placenta.

Management

An intravenous line was established and blood taken for grouping and cross-matching. Two units of blood were made available on request. She was started on 40 units of oxytocin in the drip of 500mls 5% dextrose. The diagnosis was explained to her and the consent for manual removal of the placenta under general anaesthesia was obtained. Intramuscular atropine o.6mg was given and the patient wheeled to theatre. In theatre, she was put under general anaesthesia and in lithorimy position vulvo-vaginal toilet was done and then she was draped. She was aseptically catheterized obtaining clear urine. Examination revealed a fundal height corresponding to a 20 week gestation; the vaginal and cervix were intact. The right hand was inserted into the uterus and the fundoposterior position of the placenta identified. The left hand was used to steady the uterus abdominally while the right hand was used to shear off the placenta using the ulna aspect.

Shearing was done starting from one edge until the whole placenta was extracted; it weighed 500g and was inspected and found to be complete. Uterine message was done and ergometrine 0.5mg given intramuscularly. Having ensured that there was no abnormal bleeding, she was repositioned in supine position and reversed from general anaesthesia.

Post-operative Management

Intravenous oxytocin infusion was continued. Her vital signs were observed ½ hourly till she was fully awake. Vaginal bleeding remained normal as expected for lochia loss. In the postnatal ward, she remained stable; she was discharged home on antibiotics after 24 hours and advised to attend maternal child health clinic at the nearest health facility.

COMMENT

The patient was a primigravida prior to this delivery. She was admitted with retained placenta for which manual removal was done under general anaesthesia. Failure of the placenta to deliver spontaneously is a major cause of postpartum haemorrhage. If the placenta is not expelled within 30 minutes after completion of the second stage of labour, this is considered abnormal[1]. There is generally no clear cut time before manual removal of placenta is undertaken. A third stage longer than 10 minutes is considered abnormal by some, while others advocate expectant management for as long as 30 minutes or even up to 2 hours[2,3]. However it has been shown that there is no major risk until 30 minutes have elapsed following which time active intervention should be contemplated[4]. The patient presented had retained placenta for about 3 hours.

The cause of placenta retention is often not identified. Possible causes include a mismanaged third stage, uterine atony and abnormally adherent placenta[1,4]. Uterine atony may be due to prolonged labour, multiparity, augumented labour or following induction of labour. Abnormal placental adherence may follow previous abortion or uterine scars[1,4,5]. The patient did not have any clear associated aetiological factors. Under normal circumstances, spontaneous placental separation follows soon after delivery of the baby. This occurs after the uterus has contracted and expelled the placenta through the internal segment and into the vagina. This is marked by a sudden gush of blood and lengthening of umbilical cord; the uterus seems then to elevate into the abdomen and becomes firmly globular[1,5,6].

In our maternity unit, active management of third stage is usually done using either ergomerine or oxytocin. A combination of both drugs (syntometrine) can be used but is usually not available. In addition, active management is done by gently lifting the uterus cephalad to avoid acute inversion of the uterus while applying firm traction on the cord. This is termed controlled cord traction [6]. It had been attempted on this patient without success.

Retained placenta will usually lead to postpartum haemorrhage. Blood loss may be severe enough to cause hypovolemic shock. Necessary resuscitation should be done promptly and oxytocin drip kept running. This will reverse the shock and encourage uterine evacuation[5]. This patient did not have postpartum haemorrhage; she was started on oxytocin infusion while blood was requested. She was soon taken in for manual removal.

The procedure for manual removal should be performed under general anaesthesia explained in the introduction of this book. Other methods of managing a retained placenta include attempts at infusing oxytocin through the umbilical vessels. This has been tried with some success while some studies report no success [2,8,9]. Complications of manual removal include postpartum haemorrhage, especillay with a morbidily adherent placenta (accrete,increta,percreta); uterine rupture may occur, uterine inversion and infection[1,5,6]. None of these complications were noted in this patient. She was put on prophylactic antibiotics to prevent any possible infection.

REFERENCES

1. Loeffler F. Postpartum haemorrhae and abnormalities of third stage of labour. Turnbull's Obstetrics 2nd Ed. Churchill livingstone publishers pg 729-734 (1996).

2. Craigo S. B., Kapernick P. S. Postpartum haemorrhage and the abnormal peurperium. Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th Ed. Appleton and Lange/McGraw Hill New York pg 568-574 (2003)

3. Bayer-Zwirello L. A., Veronikis D. K. The third stage. Operative Obstetrics,1st Ed. William and Willkins pg 488-509 (1995).

4. Combs C.A., Lares E.K. Prolonged Third Stage Labour: Morbidity and Risk Factors. Obstet. Gynaecol., 77(6), 1991.

5. Ratnam S.S., Rauff M. Postartum Haemorrhage and Abnormalities of the Third Stage of Labour in Obstetrics. Churchill Livingstone, Edinburg, 1989

6. Cunningham F.G., MacDonald P.C., Grant N.F. Conduct of Normal Labour and Delivery. Williams Obstetrics 21st Ed. Appleton and Lange/McGraw Hill New York pg 309-331, 2001.

7. Elbourne D., Prediville W., Chalmers I. Choice of Oxytocin Preparation for Routine in Management of the Third Stage Labour: An Overview of the Evidence form a Controlled Trial. Br.J. Obstet. Gynaecol., 95:17, 1988.

CASE NO. 9: VAGINAL BIRTH AFTER CAESAREAN SECTION

Name:	H.A	Parity:	1+1
Age:	23yrs	DOA:	12.1.2002
Unit:	0818400	DOD:	14.1.2002
LMP:	10.4.2001	EDD:	17.1.2001

Presenting complaints

She was admitted from home complaining of labour pain six hours before admission. There was no history of drainage of liquor or vaginal bleeding.

Past medical history

Not contributory

Family and social history

She was a housewife. The husband was a technician with Ministry of Works. She was educated up to standard five. She neither drank alcohol nor smoked cigarettes. There was no family history of chronic illness.

Obstetric and gynaecologic history

She was para 1+1. The first pregnancy ended in a spontaneous abortion at four months. No evacuation was done. The second pregnancy was in 1999 and she was delivered by caeserean section due to failed induction for high blood pressure. The birth weight of the child, a male, was 2.5Kg and was alive and well.The wound healed well and peurperium was uneventful.

Present pregnancy

Her last menstrual period was on 10.4.01. Her expected date of confinement was 17.1.02. She was sure of her dates. She had not used any contraceptives. Her cycles were regular every 28-30 days with a flow lasting four days. She was booked at KNH antenatal clinic on 13.10.01 at a gestational age of 26 weeks because of the previous scar. She had attended nine times. All was well. Her height was 5 feet 5 inches.

Haemoglobin = 12.6g/dl

Blood group = B Rhesus 'D' Positive

HIV Test - Negative

Serology for syphilis: Negative

A clinical pelvic assessment was done at 36 weeks, and the pelvis was adequate. (The sacral promontory was not reached, the ischael spines were not prominent and the sacral curve was concave. The subpubic angle was wide and could accommodate two fingers. The intertuberous distance could accommodate 4 knuckles.) She was to have a trial of scar when she went into labour.

She was last seen at the antenatal clinic on 30.12.95 when she was at a gestation of 41+ weeks. She was informed about the need to deliver her by an elective caesarean section as she was getting to post term. She was only 3 days before she was 42 weeks. She declined to be admitted on that day and said she would report to labour ward after the 3 days if she would not have gone into labour.

However she never showed up till the time she was in labour. Her gestation at admission was now 43+ weeks.

Physical Examination

She was in fair general condition, not pale, afebrile and there was no pedal edema. Her vital signs were as follows:

Blood pressure: 110/80 mmHg, Pulse: 76/Minute, Temperature: $36.^{80}C$, Respiration: 22/minute. The cardiovascular, respiratory and the central nervous system were normal.

Abdominal Examination

She had a midline subumbilical scar. The fundal height was term with fetal estimated weight was 3kg. The fetal lie was longitudinal and its presentation cephalic. The head was engaged 4/5 up. There were only mild contractions felt, 1-2 every 10 minutes lasting less than 20 seconds. The fetal heart tones were auscultated and found to be 140/minute and regular.

Vaginal Examination

The vulva was normal. The cervix was partially effaced and soft, the os admitted one finger. A pelvic assessment was done and confirmed the antenatal findings.

Diagnosis

An impression of one previous caesarean scar at term in latent phase of labour was made.

MANAGEMENT

She was for trial scar. An intravenous dextrose drip was started after taking blood for grouping and cross-matching. Two units were to be ready on request. Half hourly observations of blood pressure, pulse rate, contractions and fetal heart rate was done.

After 4 hours she was reviewed and found to have picked good contractions. Her blood pressure was 110/80mmHg. The pulse was 80/minute and of good volume. She had 3 contractions in 10 minutes lasting 20 – 30 seconds. The head was 3/5 up. The fetal heard rate was 136/min and regular. The cervix was now fully effaced and dilated 3cm. The membranes were intact. The lower uterine segment was swept and the membranes were buldging. No cord was felt. Artificial rupture of the membranes was done. The liquor was clear. There was no caput or moulding. The position was left occipito anterior.

This was followed by rapid and progressive labour with strong contractions. Four hours later the head was only one fifth above the pelvic brim, and the cervix was 8cm dilated. She was fully dilated two hours later and was in second stage.

She was taken to the delivery room. The pelvis was roomy and the perineum was lax. She had a spontaneous vertex delivery ten minutes later to a female baby whose weight was 2800gm. The apgar score was 8 in one minute and 10 in five minutes. The placenta was delivered by controlled cord traction and was complete. The uterus was well contracted. The perineum did not have any tears. The blood loss was normal.She was transferred to the cold wards for observations.Routine postpartum observations were normal and she established lactation quite fast. She was discharged the following day.

COMMENT

Craigin's dictum "once a section, always a section" that was appropriate for a previous time should now be replaced by the axiom "once a section, often vaginal birth after caesarean" [1].

The safety of such vaginal birth has been documented for most women. Recent data indicate that a trial of labour is successful in 60 – 80% of patients who had lower transverse uterine incision for the previous delivery and who were candidates for vaginal births for subsequent pregnancies .[1, 2, 3]

At Kenyatta National Hospital (KNH) Walton [4] obtained a successful trial in 73.9% of his patients. Noting that upto 50% of caesarean sections are performed solely because of prior section [3,5] a successful vaginal delivery would have substantial benefits in eliminating operative and post operative complications, reduction of length of hospital stay and also save money.

The patient presented underwent a successful vaginal birth after one previous caesarean section and was discharged home on the following day.

In selecting the patients for such trial the American College of Obstetrics and Gynaecology (ACOG) [2] recommend the following points to be reflected in each management protocol:-

- In the absence of a contraindication, a woman with one previous caesarean delivery with a lower segment incision should be counseled and encouraged to undergo a trial of labour in her current pregnancy.
- A woman who has had two or more previous caesarean deliveries with low uterine incisions and who wishes to attempt vaginal birth should not be discouraged from doing so in the absence of contraindications.
- Efforts should be made to document the type of previous uterine incision. If unsuccessful a judgment must be made as to the advisability of a trial of labour, since classical incisions continue to be used especially for patients who undergo caesarian sections for extreme prematurity or transverse lie.
- A previous low vertical incision or a foetus with an estimated weight of more than 4000 g are not contraindications for a trial of labour
- A previous classical uterine incision is associated with a rate of rapture of upto 12%. A trial of labour should be discouraged in such cases.

- A trial of labour and delivery should occur in a hospital setting that has the professional resources to respond to acute intrapartum obstetric emergencies such as performing a caeserian delivery within 30 minutes from the time the decision is made until surgical procedure is begun.

This protocol is similar to the one recommended by Canadian obstetrician [6] except that a low vertical incision is a contra-indication for a trial of scar for the Canadians.

Waltons recommendations[4] include performing pelvimetry and trying women whose pelvic dimensions show a true conjugate of a least 10.5cm. Ogutu[7] and Githiru[8] found that clinical pelvimetry alone is enough to predict a successful trial of scar. In the United Kingdom 42% of the obstetricians use clinical pelvimentry[6]. The American and Canadian protocols do not emphasize the use of pelvimentry. [2, 6]

Suspected macrosomia does not appear to be a contraindication to labour after a caesarean section.[1,3] Trial of labour has been shown to be successful in upto 70% of women in whom the indication for the primary section was " cephalopelvic disproportion".[2] However such women should be individualized based on actual estimate of fetal weight and maternal pelvimentry [1]

The patient presented had one previous caesarean scar, the estimated fetal weight was 3Kg and clinically, the pelvis was adequate.

Trial of labour with more than one previous caesarean scar has not been shown to be associated with an increased risk of uterine rupture, [9] although there is paucity of studies to confirm this. In our institution all patients with more than one previous section undergo an elective caesarean.

Oxytocin use for either induction or augmentation of labour is not contraindicated in women with previous caesarean scars undergoing trial of labour. However optimal monitoring of contractions with the use of intrauterine pressure catheter, where feasible, is recommended. In the UK, there is a general agreement that oxytocin is not contraindicated. [6] In our institution we are yet to begin using oxytocin for that purpose due to lack of monitoring facilities.

Studies done at KNH on multiparity with one previous scar concluded that these patients can have a successful trial of scar [10, 11]

Intapartum management of patients undergoing a trial of scar should include the following:-

- Intravenous access on admission
- Obtaining blood count, type and crossmatch with two units of blood available
- Continous electronic fetal monitoring
- External tocodynamometry, with internal uterine monitoring of contractions when feasible.
- Nothing by mouth during labour [1]

The signs of uterine rupture which should be looked for during the trial include:

- Signs of fetal distress. This is the most common finding, reported to occur in 50 – 70% of all uterine ruptures [1] and presents with abnormal fetal heart rate patterns with variable decelerations and fetal bradycardia.
- Uterine pain, which continues between contractions usually located in the area of the previous incision.
- Intrapartum haemorrhage and hematuria. Other signs include loss of uterine contractions, recession of the presenting part, and fetal death [1]

Our patient had progressive cervical dilation, the fetal heart rate remained regular and contractions progressively increased in intensity until she had a vaginal delivery. None of the said clinical features of uterine rupture were present.

REFERENCES

1. Pridjian G. Labour after prior caesarean section. Clin. Obstet & Gynaecol 35:3: 445, 1992.

2. ACOG Committee opinion on Obstetric Practice. Vaginal delivery after a previous caesarean birth. Int. J. Gynaecol & Obstet 48 (1995) 127 – 129.

3. Ralph W., Hale. Operative delivery. Current Obstetric & Gynaecologic diagnosis & treatment. Appleton & Lange/McGraw Hill New York. 9th Ed. (2003) Pp 570-571.

4. Walton S.M. The antenatal and intrapartum management of patients with previous caesarean section scars.East Afri. Med.J. 33:47,1987

5. Karanja J.G. A review of caesarean section deliveries at Kenyatta National Hospital in 1980. M. Med Thesis U.o.N, 1983

6. Roberts L.J., Beardworth S.A., Trew G. Labour following caesarean section: Current practice in the United Kingdom. Brit. J. Obstet and Gynaecol Vol. 101 pp 153-155, Feb, 1994.

7. Ogutu G.W. A prospective study of X-ray pelvimetry and trial of scar and mode of delivery at Kenyatta National Hospital. M.Med Thesis U.o.N., 1985.

8. Githiru K., Kamau R.K. et al. Value of erect lateral pelvimetry in management of patients with one previous scar. J.Obstet.Gynae. East Cent. Afr. 10:1:13,1992.

9. Phelan J.P., Ahn M.O., Diaz F., Brar H.S., RodriguesM.H. Twice a caesarian always a caesarean? Obstet gynaecol 1989:73:161.

10. Miano J.R. Grand Multipara – A review of 200 cases. M. Med Thesis UoN, 1977

11. Nyamu J.M. The management of Grand Multiparous patients previously delivered by caesarean Section at Pumwani Maternity Hospital and Kenyatta National Hospital. M.Med Thesis, UoN, 1989.

CASE NO. 10: PRETERM PREMATURE RUPTURE OF MEMBRANES SUCCESFUL DELIVERY

Name:	A.N.	DOA:	27.6.2002
Age:	23 years	DOD:	5.7.2002
IP No:	0821340	Parity:	1+0
LMP:	6.11.2001	EDD:	13.8.2002

Presentation Complaints

Patient was admitted through casualty into labour ward with complaints of draining liquor for 1 day and labour pains for 6 hours.

History of Presenting Illness

Patient had been well till 1 day before admission when she developed draining of liquor, which ran down her feet on standing. She did not seek help till late at night when she developed labour like pains. She was then taken to a nearby nursing home that referred her to Kenyatta National Hospital.

History of Presenting Pregnancy

Her last menstrual period was on 6.11.2001 thus she was at 34 weeks gestation at the time of admission. She had attended antenatal clinic in Mathare North starting at 28 weeks gestation. She only attended twice. During her antenatal visit there was no problem noted. Antenatal profile:

Blood group 0 rhesus (+) ve, Hb 12.6 gm/dl , VDRL (-)ve , HIV (-)ve.

Past medical and surgical history

This was not significant.

Past Obstetric and Gynaecologic history

She was a para 1+0. Her last delivery was in 2000 at term by spontaneous vertex delivery to a female baby who weighed 2.5Kg and was alive and well. She had no complications during that pregnancy or the puerperium. She attained her menarche at age 14 years. Her cycles were regular with menses lasting 3 days and a cycle of 28-30 days. She had never used any contraceptive method.

Family and social history

She was a housewife. She lived with her husband and daughter in Huruma estate. She did not drink alcohol or smoke cigarettes. There was no history of chronic illness in the family.

Examination

She was in good general condition, not pale, not jaundiced nor febrile. She had no edema or lymph node enlargement.

Respiratory system

The respiratory rate was 20 breaths per minute. The air entry was good bilaterally. There were no added sounds.

Cardiovascular System

Her pulse rate was 86 beats per minute and of good volume. The blood pressure was 110/70mmHg. The first and second heart sounds were heard and there were no murmurs.

Abdomen

The abdomen was uniformely distended and was moving with respirations. The uterus corresponded to 34 weeks gestation. The fetus was in longitudinal lie and cephalic presentation. The head was two fifths above the pelvic brim. She was having 3 contractions in 10 minutes that lasted between 30 to 40 seconds. The fetal heart was heard and the rate was regular at 138 beats per minute.

Pelvic Examination

The external genitalia was normal, the cervix was fully effaced and the os was six centimetres dilated. There was no cord felt, caput or molding of the fetal head. She was draining clear liquor, which had no offensive smell.

Impression

Preterm premature rupture of membranes at 34 weeks gestation in labour.

Management

The patient was in established labour.

She was allowed to progress and started on partogram monitoring. Due to prolonged rupture of membranes (close to 24 hours) she was given erythromycin 500mg orally 8 hourly for 5 days. She progressed well and delivered a live male baby who weighed 2200g. The placenta weighed 500g and was complete and healthy. The baby was admitted into the neonatal unit where he stayed for 7 days. On day 3, he developed jaundice for which he was treated with phototherapy for 3 days. He was discharged from the neonatal unit on the 7th day in good general condition.

The patient's stay in the ward was uneventful. She did not develop any puerperal fever and her lochia remained normal during her stay in the ward. The uterus was involuting well. She was discharged on 5.7.2002 to attend postnatal clinic in 5 weeks.

Follow up

The patient did not turn up for follow-up as advised.

COMMENT

The patient presented had preterm premature rupture of membranes. She delivered vaginally to a live male baby. Premature rupture of membranes (PROM) is defined as rupture of fetal membranes before labour. [1, 2] This may occur before 37 weeks gestation or after 37 weeks. The former is known as preterm premature rupture of membranes *(PPROM) and the latter is termed premature rupture of membranes (TPROM).* Our patient had preterm premature rupture of membranes at 34 weeks gestation.

The incidence of premature rupture of membranes ranges between 10-17% [1,3] with 20 – 46% of the PROM occurring in preterm patients [5] though others have given a lower figure of 5% .[1] In Kenya the incidence at Kenyatta National Hospital was given as 9.3% of all deliveries. [4] Of these 43.7% were preterm premature rupture of membranes. In his study, Wanjala [5] found 46.4% of the premature ruptures of membranes were in preterm pregnancies. The two figures are not significantly different.

The etiology of PROM is not known but there are several associated factors which include cervicitics, vagitinitis, incompetent cervix, cigarette smoking, prenatal diagnostic procedures, coitus, nutritional deficiency and cervical examinations. These are remediable. There are other risk factors that are not remediable, such as previous history of PROM, cervical surgery, placental pathology, Ehlers-Danlos syndrome and fetal sex (male). The time of the day has also been implicated with majority of PROM occurring at 2 – 4 o'clock in the morning. The recurrence rate of PROM is 21%. If a patient has had a prior preterm delivery with or without PROM the risk of a subsequent PROM doubles. [6]

These patients have a relatively low collagen types III and IV which contribute to the reduced resistance of the amnion to the collagenolytic activity of the enzymes.[7]

The patient had a male fetus, she had no previous history of PROM, abortions, preterm labour, she did not smoke cigarettes nor did she have any genital infection previously.

Premature rupture of membranes presents with drainage of liquor. This may be continous wetness of the vagina or a sudden gush that goes down the legs. The diagnosis is usually confirmed clinically by speculum examination which reveals drainage from the cervical os. This may be difficult to note even with straining in small or old leaks.[8] This can be confirmed using the following tests; cytological identification of lanugo and uric acid

crystals in vaginal fluid. Staining for lipids, identification of fetal cells, alteration of vaginal cytology, pH determination, carbonization and alpha fetoprotein [8]

The simplest method of determining pH is by use of the litmus paper. This shows an alkaline pH in the vagina which without rupture is acidic. The test has a 35% error. [9]

The most commonly used method is Nitrazine indicator paper which changes colour (to blue) at pH 6.4-6.8 which is taken to indicate evidence of ruptured membranes.[10] In our patient the diagnosis was made from clinical examination.

The management of preterm premature rupture of membranes is aimed at reducing the fetal and maternal morbidity and mortality. The patient is assessed to confirm rupture of membranes. The cervical dilation is assessed visually through a speculum and also noted whether there is cord prolapse or prolapse of fetal parts.[11] If patient is not in labour, amniocentesis may be done transabdominally, or, 1ml of vaginal fluid taken to assess the fetal lung maturity using the lecithin/sphingomyelin ratio or the phospatidylglycerol for the vaginal sample. If the patient is in labour she is allowed to progress and deliver vaginally unless obstetrical indications for caesarian section are present. That not in labour would be managed expectantly. For patients in whom expectant management has been selected, the patient must have bed rest, weekly white blood cell counts, daily temperature and pulse rate monitoring. Clinical evaluation should be done daily for early detection of infection. In those that one uses prophylactic antibiotics, erythromycin or cephalosporin is preferred. This only helps to reduce frequency of post partum endometritis.[2] The patient presented had 34 weeks gestation and was given prophylactic intravenous ampicillin.

The complications of PPROM include prematurity, Respiratory Distress Syndrome (RDS). Hyaline membrane disease, intraventricular haemorrhage, pulmonary hypoplasia, positional deformities, fetopathy of prematurity, cerebral palsy, necrotising enterocotis, neonatal sepsis, thermal instability, hypoglycaemia, hyperbilirubianemia and fluid and electrolyte imbalance .[11]

To prevent premature rupture of membranes may not be possible at present but some of the known remediable courses can be avoided such as enumerated earlier. Vaginal examination antenatally should be avoided unless totally indicated. However this has not been associated with PROM or choriomnionitis in a recent study .[1]

REFERENCES

1. Pernoll M.l. Late pregnancy complications. Pernoll M.L. ed. Current obstetric and gynaecologic diagnosis and treatment. Appleton and Lange/McGraw Hill New York. 9th Edition, 2003, pp 332.

2. Polzin, W.J., Brady K. Mechanical factors in the aetiology of premature rupture of the membranes. Clin. Obstet. Gynaecol. 1991; 34(4): 702.

3. Trap, R., Helm P, Lidegard, O; et al. Premature rupture of the fetal membrane the phases of the moon and barometer Readings. Gynaecol. Obstet. Invest. 1989, 28:14

4. Otieno, J.A. Spontaneous premature rupture of the membranes. A retrospective analysis of 80 cases. M.Med Thesis, University of Nairobi, 1980.

5. Wanjala, S.M.H. Premature rupture of membranes. Maternal and fetal morbidity and mortality. M.Med. Thesis, University of Nairobi, 1980.

6. Herge J.H., Hsing A.W., Tuomala R.E. et al. Risk factors for preterm premature rupture of fetal membranes. A multicentre case control study. Am. J. Obstet. Gynaecol 1990 : 163: 130

7. Victoria H. Amniotic membranes collagen content and type distribution in women with PPROM in pregnancy. Bri. J. Obstet. Gynaecol. Vol.104 No.9 pg1087, 1997.

8. Davidson, K.M. Detection of premature rupture of the membranes Clin. Obstet. Gynaecol. 1991; 34:715

9. King A.G. The determination of rupture of membranes. Am. J. Obstet. Gynaecol. 1985:30:860

10. Abe T. The detection of rupture of fetal membranes with nitrazine indicator. Am. J. Obstet. Gynaecol 1940; 39:400

11. Munson L.A. Jr., Graham A., Koos B.T. et al. Is there a need for digital examination in patients with spontaneous rupture of the membranes? Am. J. Obstet. Gynaecol. 1985;153:562.9 Abe T.

12. Harlass F.E. The rupture of membranes in patients with preterm labour. Clin. Obstet. Gynaecol. 1991, (34) (4); 751.

13. Greenberg R.T., Harnkins, G.D.V. Antibiotic therapy in preterm premature rupture of membranes. Clin. Obstet. Gynaecol, 1991; 34 (4): 742

14. Seo K., McGregor J.A., French J.I. Preterm birth is associated with increased risk of maternal and neonatal infection. Obstet. Gynaecol, 1992; 79 (1): 75

CASE NO. 11: UNSENSITISED RHESUS D-NEGATIVE MOTHER, ANTI-D AFTER DELIVERY

Name:	L.A	Age:	22 years
Parity:	0+0	Unit No:	0873413
L.M.P:	22.4.2002	EDD:	29.1.2003
DOA:	11.1.2003	Gestation on Admission: 37^{+} weeks	
DOD:	13.1.2003		

Presenting Complaint

The patient was admitted from home complaining of labour pains since early morning of 11.194. She had passed show, was not draining liquor and had no PV bleeding.

Past medical history

Not significant

Social and family history

She was a housewife; the husband worked with Kenya Power and Lighting Company as an engineer. There was no family history of twins, tuberculosis, diabetes or hypertension. She neither smoked nor drunk alcohol.

Obsteric and Gynaecological History

She was a primigravida with a gestational age on admission of 37^{+} weeks. Antenatally she was booked in Kenyatta National Hospital because she was a primigravida and her gestation at her first visit was 19 weeks.

Follow up Antanatally

Her Hb was 12.1g/dl, Bp was 110/70 mmHg. VDRL was negative, blood group O Rhesus 'D' negative. HIV test was negative; husband's blood group was B Rhesus positive.

She was seen seven times in the antenatal clinic. On the third visit an obstetric ultrasound was requested for because her fundal height was found to be 30/52 although she was 28/52 by dates. The report showed a single live fetus assessed with biparietal diameter and femoral length to be about 29 weeks mature.

This was consistent with her dates.

Indirect coomb's test was done and found to be negative.

Throughout her follow up, she had been relatively well and on her last visit she was to be seen again after one week for the reports but she went into labour before the appointment date.

Her pelvis was assessed at 36/52 and found to be clinically adequate.

She was counseled on Rhesus negative problem and advised to buy Anti-D immunoglobulin in the event she goes into labour since it was out of stock in the hospital.

General Examination

She was in good general condition. She was not pale, had no edema or jaundice. She was afebrile. Vital signs; Bp 110/70 mmHg, Pulse 74/min and RR 22/min. Her respiratory, cardiovascular and nervous systems were normal.

Abdominal Examination

The abdomen was uniformly distented and moving with respiration. The fundal height corresponded to term pregnancy, longitudinal lie, cephalic presentation 4/5 above the pelvic brim.Fetal heart rate was 136/minute, regular.

Vaginal Examination

She had a normal external genitalia, the vaginal walls were normal. The cervix was soft, partially effaced and the dilation was 2cm. The membranes were intact. The pelvis was assessed and found to be adequate.

Diagnosis

Unsensitised Rh Negative mother at term in latent phase of labour.

MANAGEMENT

She was initially given pethidine 100mg by intramuscular injection and she was to be reviewed again after 4 hours. This was at 10.45 am.

A second review at 2.30 pm was done and was not found to be in active labour and trasfered to the cold antenatal wards. She was transferred back to labour ward at 11.45pm in active labour. This time she was contracting moderately. The station of the presenting part was 3/5 up and the cervix was 4cm dilated. Artificial rupture of membranes was done and clear liquor was obtained. The lower uterine segment was stripped of the fetal membranes. The labour progressed well and the mother was in second stage at 5.30 am and delivered a live male infant who scored 8/1 and 10/5 whose weight was 3600 gms. The placenta was delivered by controlled cord traction. It was normal. The blood loss was insignificant.

The baby was taken to nursery for further observations. The mother was given 300mgm Anti D immunoglobulin intramuscular on the 12.1.2003.

Progress

The baby developed jaundice on the second post delivery day. Her blood group was B Rhesus 'D' Positive Direct coombs test – Negative, Hb – 16g/dl. This was determined before the mother was given the anti-D immunoglobulin. The bilirubin level was 46 and 40 umol/l for total and direct levels respectively. This was on the higher side of the normal levels. She was put on phototherapy for 2 days and the jaundice cleared. This was thought to have been physiological. The mother was discharged on the 13.1.2003 while the baby was discharged on the 15.1.2003 in good general condition.

DISCUSSION

This case is that of an unsensitised Rh D-negative mother who received anti-D IgG immunoglobulin after delivery of a baby who developed physiological jaundice which cleared on phototherapy. In the erythrocytes blood group systems, there are about 400 recognized antigenic factors of which the most common are the ABO, Rhesus, MNs, P, Kell and Lewis. Others are Lutheran, Duffy and Kidd.[1]

The most important of these is the Rhesus (Rh) factor, so named because the antigen was first recognized in the red cells of the rhesus monkey. The rhesus antigens are grouped in 3 pairs: Dd, Cc, and Ee. The terminology "Rh positive" or "Rh negative" refers to the presence or absence of the 'D' gene. A woman, who lacks the Rhesus factor, Rh (D), may carry a Rh-positive fetus. If fetal blood cells pass into the mother's circulation in sufficient numbers, maternal antibodies to the Rh-positive antigen may develop and cross the placenta, causing haemolysis of fetal blood cells. The degree of haemolysis varies from causing mild anaemia to a severity which results in cardiac failure and fetal death[1, 2]. Furthermore, haemolysis continues after birth until the maternal antibodies have been eliminated. The excess blood pigments cannot be disposed off readily by the fetus so that jaundice sometimes of severe degree may develop [1].

There are considerable racial variations in the distribution of Rh groups. The Basque population has the highest incidence of Rh-Negativity (30-35%). Caucasians have an incidence of 15-16%, Finland 10-12%, Blacks in the USA have a rate of 8%, African Blacks 4%; Indonesians have a low incidence of 2% while the American Indians have a rate of 1%. The incidence among mongoloid races is nil [2].

In Nairobi the incidence among pregnant women attending antenatal clinics is 5% [3]. At Kenyatta National Hospital (KNH), the prevalence is 4.1% and 2.6% for antenatal mothers and patients with incomplete abortions respectively .[4, 5].

The initial response of a Rh-negative individual to injection of Rh positive cells, is the formation of IgM ("Complete or saline") antibodies. These are of large molecular weight and do not cross the placenta. IgG ("incomplete or albumin") and IgC (7C immunoglobulin) antibodies develop subsequently (within 6 weeks to 6 months) and these can cross the placenta to the detriment of the fetus [1, 2]. Maternal levels of IgM fall fairly rapidly but IgG antibodies decrease only slightly over a period of years. Further injection of Rh positive cells produce a rapid and stronger IgG response. The degree of

initial sensitization may be so low that it is undetectable by normal laboratory testing (sensibilization), but such patients will develop a strong and rapid response to further stimuli [1,2].

Upon crossing the placenta, maternal antibodies get attached to the corresponding erythrocyte antigens. The antibody antigen complex will be recognized and subsequently phagocytosed and destroyed by fetal macrophages in the reticuloendothelial system, mainly in the fetal spleen. This results in a decreased life span of the fetal erythrocytes and a compensatory increase in fetal erythropoiesis. Fetal anaemia will ensure if the increased erythropoiesis cannot compensate for the destruction of red blood cells. The degradation of fetal blood cells results in the production of bilirubin which is secreted into the urine and subsequently into the amniotic fluid. In severe cases the fetal anaemia will lead to hypoxia, acidosis impaired liver function, skin edema, ascites, pericardial effusion and eventually impaired cardiac function and fetal demise. The principle mechanism was first recognized in 1941 by Levin et al [6].

The stimulus to Rh (D) antibody, production in a Rhesus negative woman is either the presence of Rh (D) positive fetal cells which have traversed the placenta, or incompatible blood transfusions, which provides a powerful stimulus to antibody formation. It has also been suggested that a Rh negative fetus might be sensitized by trasplacental passage of maternal Rh positive cells which may account for antibodies which are sometimes detected in a first pregnancy [1,2.]

Fetomaternal haemorrhage may occur during pregnancy or at delivery. With no apparent predisposing factors, fetal red cells have been detected in maternal blood in 6.7% of women during the first trimester, 15.9% in the second trimesters, and 28.9% in the third trimester [2].

Kizza [7] found higher values than in the Caucasian population in a study done at Kenyatta National Hospital. His study found a fetomaternal haemorrhage of 15.4%, 29.5% and 38% in the first, second and third trimester respectively.

Predisposing factors to fetomaternal haemorrhage include spontaneous or induced abortion, abdominal trauma including external cephalic version, placenta praevia, fetal death, multiple pregnancy, manual removal of placenta and caesarean section [2].

Unless there has been any previous stimulus, the first Rh incompatible pregnancy rarely results in the development of antibodies before delivery, but a rapid antibody response can occur in the next incompatible pregnancy.

The patient discussed was a primigravida and she remained unsensitized till delivery. Sensitization occurs in only about 1 in 20 pegnancies in which there is feto maternal Rh incompatibility [1].

Factors influencing the development of Rh antibodies in a Rh negative women include:-

- The fetal blood group – The fetus must have inherited a "D" gene form the father.
- Transplacental passage of fetal cells into the maternal circulation. Although the exact number of Rh-Positive cells necessary to cause isoimmunization is unknown, as little as 0.1mls of Rh positive cells will cause sensitization.
- Natural protective mechanisms. ABO incompatibility results in the destruction or inactivation of transfused fetal cells before they have the opportunity to induce Rh antibody formation.
- Variability of response to the stimulus. About 30% of Rh-Negative persons never become sensitized (non-responders) when given Rh-positive cells. The factors influencing this individual variation is not clear but is thought to be genetically determined [1, 2]. The patient's fetus was blood group O Rhesus positive. Also since this was her first pregnancy and she was not sensitized the problem of haemolytic disease of the newborn was not expected.

Prevention of Rhesus iso-immunisation is the mainstay of management of the rhesus negative unsensitised, pregnant woman. On the first booking visit, the antenatal mother should be screened for blood group isoantigens (ABO and Rh). They should also undergo antibody screening (indirect coomb's test) at 28 weeks. If the woman is Rh negative, testing for paternal ABO and Rh blood groups may be useful, more so where the woman is sensitized to decide whether there is a possibility for the fetus being affected; and also for counseling on future pregnancies [6]. This patient was discovered to be Rh negative during routine antenatal profile in our clinic.

The administration of IgG antiD Immunoglobulin to the mother at the time of exposure to the antigen will inhibit the synthesis of IgM, most likely by blocking the antigen determinants on the fetal erthrocytes i.e the D antigen. This prophylaxis will only be effective if the amount of Anti D immonoglobulin is enough to cover all fetal red blood cells that have passed the placental to the mother. It must also be given before a primary IgM, response [6].

Routine antenatal anti D immunoglobulin given at 28 and 34 weeks have been advocated to reduce the number of sensitizations. However in our unit prophylaxis is given only

when there is a predisposing factor to transplacental haemorrhage as stated earlier. This is because of the cost of anti D immunoglobulin. The majority of fetomaternal transfusions occur during the third trimester and at the time of delivery. The risk for immunization also increases at obstetric procedures such as manual removal of placenta where the transfused blood volume can be larger than 25 mls [8].

A standard dose of 300ug anti-D immunoglobulin is regarded as sufficient for fetomaternal transfusions upto 25mls. This dose will also reduce the number of isoimmunisations involving larger transfusions by upto 50% [6]. The amount of fetal blood in the maternal circulation can be assessed by testing maternal blood according to the Kleihauer-Betke method. An additional amount of 300ug anti-D immunoglobulin should be given for every additional 25mls of fetal blood found in the mother .[9]

In North America, a single dose of 1500 units of Anti-D globulin given at 28 weeks gestation has been found to be protective against the sensitization that occur in late pregnancy .[1]

Anti-D globulin is normally given within 72 hours of birth but appears effective if given as long as 28 days postpartum.[1] It is commonly given by the intramuscular route principally because this can be undertaken by the midwife. However, the intravenous route is reported to be more effective.[1] The patient discussed was given 300mgm of antigen-causing antibody production in the pregnant woman. Chorionic villus sampling will enable phenotype of fetal red blood cells to be determined. Where the risk of carrying a severely affected fetus is unacceptable appropriate action can be advised.

Serial ultrasound apart from accurate determination of gestational age early in pregnancy may reveal features of a severely affected fetus such as ascites, pericardial and pleural effusions, and skin oedema (hydrops fetalis). Other features include enlarged placenta, dilated umbilical vein and hydramnios.

Doppler studies – blood flow velocity studies on fetal circulation provide information on the fetal adaptation to anaemia and hypoxaemia and redistribution of blood flow. In anaemic fetus, there is increased umbilical artery resistance, intrahepatic umbilical vein blood flow is increased and dilated umbilical vein is seen.

Amniocentesis – analysis of bilirubin concentration in amniotic fluid is performed by spectrophotemetry. The optical density (OD) at 450mm is then determined. The concenctration of bilirubin is inversely proportional to the haemoglin concentration in the fetal blood. The deviation of the OD from the baseline is then compared to the

evaluation chart, originally constructed by Liley. Delivery depends on the degree of affection and gestation of the fetus.

Fetal blood sampling may be done to determine the degree of fetal anaemia and intra-uterine tranfusion may be done [1, 2, 6]

During delivery, the haematologist and paediatricians are involved. Blood is obtained from the umbilical cord and the ABO and Rh group of the fetus are determined. Other investigations are Coomb's test, haemoglobin concentration, and serum bilirubin estimation. The haemoglobin and bilirubin estimations should be repeated six hourly for the first 36 hours and less frequently as indicated[1] Indications for early exchange transfusion are:-

Haemoglobin less than 15.5g/dl, positive Coomb's test, cord serum bilirubin more than 85 umol/l and previous history of severely affected infant plus positive Coomb's test, irrespective of other findings[1]

The baby was taken to nursery and developed jaundice on the second day. The investigations showed this haemolysis to be mild and because the direct coomb's test was negative and the baby had a high haemoglobin level 16g/dl, and since this was the first child, the jaundice was thought to be physiological in origin. Indeed it disappeared in a 2 days on phototherapy. No exchange transfusion was indicated.

REFERENCES

1) Cunningham., McDonald. Foetal abnormalities; inherited and acquired. Williams obstetrics 21st Ed. Appleton and Lange/McGraw Hill New York pg 1057-1068,2001.

2) Ashley S., Pernoll M. L. Rh isoimmunization and other blood group incompatibility. Current obstetric and gynaecologic Diagnosis and treatment. 9th Ed. Appleton & Lange/McGraw Hill New York pg 295-299, 2003.

3) Mati J.K., Agarrwal V.P., Sanghvi H.C.G., Lucas S. et. al. Nairobi Birth Survey II: Antenatal care. J.Obstet. Gynaecol. East Central Afr. 2(1): 1, 1983.

4) Mulandi, T.N. A two year prospective study to show the effectiveness of anti-D gamma globulin in preventing Rhesus negative pregnant women at Kenyatta National Hospital. M. Med Thesis U.o.N. 1985.

5) Muroki, F.K. The Rhesus problem after abortion in Kenyatta National Hospital M.Med Thesis, U.o.N. (1987)

6) Magnus S., Anders S., Goran L. et. al. Rhesus immunization: New perspective in maternal fetal medicine. Obstetrical and gynaecological survey 48, 4: 189, 1991.

7) Kizza A.P., Rogo K.O. Fetomaternal haemorrhage in Kenya. E.Afri. Med. J. 1990; 67:810

8) Bowman, J.M. Controversies in Rh prophylaxis: Who needs Rh immunoglobulin and when should it be given? Am. J. Obstet. Gynaecol. 151, 3, 1985.

9) Li T.C., Bromham D.R.., Balmer B.M. Fetomaternal microtransfusion in the Yorkshire region prevalence and obstetric factors. Br. J. Obstet. Gynaecol. 95:1144,1988.

CASE NO. 12: MALARIA IN PREGNANCY: PRETERM LABOUR – LIVE BIRTH

Name:	L.S	L.M.P:	31/12/2002
IP:	0900214	E.D.D:	7/10/2003
Age:	30 yrs	D.O.A:	23/8/2003
Parity:	2+0	D.O.D:	12/9/2003

Presenting Complaints

The patient was admitted through casualty with complaints of fever, headache, joint pains, general malaise, nausea and vomiting.

History of Presenting Illness

She was well until ten days prior to admission, when she started having headache and fever. This was followed by joint pains and general malaise.She took metakelfin and was temporarily relieved before the symptoms recurred together with nausea and vomiting.

Obstetrical and Gynaecological History

She was para 2+0. Her last delivery was in 1999 by SVD at Pumwani Maternity Hospital. Her menarche was at 14 years. She had Norplant inserted following the last delivery until October 2002 when it was removed to have a planned pregnancy.

History of Present Pregnancy

She was not on any antenatal follow-up.

Past Medical History

This was not significant

Family and Social History

She was a single lady. Prior to her illness she had been working as a tea picker in Nandi district. She did not smoke and did not drink alcohol. There was no history of major illness in the family.

Systemic enquiry

This was not contributory.

General Examination

She was ill-looking. She was febrile with a temperature of 39°c. She had mild jaundice and was pale. She was not dehydrated. The pulse was 120 per minute, regular and of good volume. The blood pressure was 120/80 mmHg and the respiratory rate was 36 per minute.

Central Nervous and Cardiovascular systems.

These were essentially normal.

Respiratory Systems

She was tachypnoeic with a respiratory rate of 26/minute. Breath sounds were vesicular and normal. There were no crepitations.

Cardiovascular System

She had a tachycardia of 120/minute. Pulse was of good volume, regular and non-collapsing. The first and second heart sounds were heard and normal.

Abdominal Examination

The abdomen was uniformly distended and moved with respiration. The liver and the spleen were not palpable. The fundal height corresponded to a 34 week gestation. The fetus was in longitudinal lie with cephalic presentation. The head was 5 fifths above the pelvic brim. The fetal heart sounds were heard at 138 per minute and were regular. There were no uterine contractions.

Vaginal Examination

This was not indicated at this stage.

Impression

Malaria and severe anaemia in pregnancy .

Systemic enquiry

This was not contributory.

General Examination

She was ill-looking. She was febrile with a temperature of 39°c. She had mild jaundice and was pale. She was not dehydrated. The pulse was 120 per minute, regular and of good volume. The blood pressure was 120/80 mmHg and the respiratory rate was 36 per minute.

Central Nervous and Cardiovascular systems.

These were essentially normal.

Respiratory Systems

She was tachypnoeic with a respiratory rate of 26/minute. Breath sounds were vesicular and normal. There were no crepitations.

Cardiovascular System

She had a tachycardia of 120/minute. Pulse was of good volume, regular and non-collapsing. The first and second heart sounds were heard and normal.

Abdominal Examination

The abdomen was uniformly distended and moved with respiration. The liver and the spleen were not palpable. The fundal height corresponded to a 34 week gestation. The fetus was in longitudinal lie with cephalic presentation. The head was 5 fifths above the pelvic brim. The fetal heart sounds were heard at 138 per minute and were regular. There were no uterine contractions.

Vaginal Examination

This was not indicated at this stage.

Impression

Malaria and severe anaemia in pregnancy .

Management

She was admitted to the antenatal ward and before commencing treatment a blood slide was taken for malaria parasites and a sample of blood for full haemogram taken. She was put on injection Artenum 160mg start to be followed by 80mg daily for four days. She was put on paracetamol two tablets 8 hourly. Other investigations were carried out to include blood group, urinalysis, urea and electrolytes, liver function tests and VDRL.

Results for Investigations

1. Blood Slide - Positive for malaria parasites. There was no report on degree of parasitaemia.
2. Haemogram - Haemoglobin – 5.3gm/dl
   RBC – Anisocytosis, moderate Polychromasia, Immature RBC, Heavy parasitaemia of plasmodium falciparum
   WBC – 14.3 x 10^9/L, N=82%, Lymph=12%, M=3%, b=1%
3. Stool - No ova and cysts seen
4. Ultrasonography- Single live fetus, fetal cardiac activity demonstrated. The BPD was 8.4cm corresponding to 34 weeks, placenta was fundo-posterior.
5. Blood group - O' Rhesus 'D' positive
6. VDRL - Negative
7. Urinalysis - NAD
8. LFTs

-Total protein	54g/L
-Albumin	21g/L
-Total bilirubin	43umol/L
-ALT	9u/L
-AST	35u/L
- ALP	63u/L

After 3 days of treatment the fever seemed to abate. She was started on Ranferone and transfused one unit of blood. On 31/8/2003, a spike of fever was noted and a blood slide indicated that she had moderate parasitaemia. An impression of recurrence was made. She was started on oral Quinine 600mg 6 hourly to take for 10 days.

The same day the patient started complaining of lower abdominal pains and on review she was found to be in labour and transferred to labour ward. In labour ward she was started on i.v. drip of 5% Dextrose, she was put on oxygen by mask and nursed on the left lateral position. The partogram was strictly observed. The first stage of labour progressed uneventufully and after 8 hours she was transferred to delivery room and a female baby was delivered with an apgar score of 9 at one minute and 9 at 5 minutes, weighing 2000gms. The baby was admitted to nursery for observations due to prematurity.She did not show any signs of congenital malaria. The placenta and the membranes were delivered by controlled cord traction. They were complete and weighed 500gms. The estimated blood loss was 150mls. Post delivery the mother was continued on oral quinine and hematinics. She was transfused 2 units of whole blood. On 4th postpartum day, repeat blood slide for malarial parasites was negative. The baby was discharged from NBU to join the mother. The check haemoglobin was found to be 8.9gm/dl after 10 days post partum. She was discharged on the 11th day on haematinics. She was to be seen in the postnatal clinic in 6 weeks.

FOLLOW UP

She did not turn up for follow up

COMMENTS

This was a 30 year old para 2+0 lady who presented with malaria in pregnancy in third trimester. The infection was complicated by anaemia and she went on to have preterm delivery.

Malaria is an infection characterized by relapsing fever, rigors, splenomegaly and anaemia. It is caused by any of the four plasmodium species transmitted from human to human by bite of female anopheles mosquitoes. These species include plasmodium falciparum, malarie, vivax and ovale. The patient presented had a typical clinical presentation of malaria with fever, headaches, rigors and anaemia. She was infected with plasmodium falciparum.In this country, plasmodium falciparum is the commonest malaria infection in pregnancy and it accounts for 98% of all cases [1].It causes acute life threatening disease. Other symptoms and signs are likely to be those of complications. [2]

Malaria is an endemic disease in many parts of tropical and subtropical Africa, Asia and Central and South America, where environmental features including temperature, humidity, bodies of water and agriculture, support the breeding of the mosquitos' vectors and encourage frequent contact between mosquitoes and man. It is a priority disease in Kenya accounting for 30% of outpatient hospital attendance.There are approximately 4000 instances per year of low birth weight babies, which is a single greatest risk factor for neonatal death(3)

In edemic areas infection first occurs in early childhood. Persistence of parasites and repeated inoculation of parasites produce humoral and cell mediated immune responses. After 5 to 10 years of residence in an endemic area, a person gets enough immunity to be asymptomatic for malaria.This is mostly undetected but associated with placental parasitation.Due to the resultant placental infarction, there is uteroplacental insufficiency leading to abortion, preterm lobour, stillbirths and low birthweight infants. Disruption of the host parasite balance by malnutrition, pregnancy or introduction of new strains of parasite may precipitate recrudescent or more severe clinical attack.[2, 4]

This patient's immunity is likely to have been disrupted by the pregnancy and probably explained the severe disease. Worldwide, it is reported that over 92 million people are infected with malaria every year.[3] The prevalence of malaria in pregnancy varies from place to place depending on the epidemicity of the disease. In a study in Coast Province of Kenya, Rukaria found prevalence of 21% among pregnant women.[5] Rukaria reported

that 45.9% of malaria infections were resistant to chloroquine in vivo with levels RI, RII and RIII being 36.1%, 8.2% and 1.6% respectively

Pregnancy exerts a damping effect on the immunity of malaria in non-immune women. In semi-immune women in endemic areas, parity appears to influence susceptibility to an important degree with primigravid women being at an increased risk of complication (6). Rukaria's study concurred with this finding and further reported malaria to be more severe in primigravidas and diminishing severity with higher parity.

The morphology of parasite is characteristic and helpful in identification of the infecting species. Diagnosis of the active infection depends on careful examination of thick and thin blood films for the parasite. Serologic tests are of help in chronically infected patients with unexplained fever.[1] This patient had plasmodium falciparum species diagnosed after a thick and thin film.

The severity of the clinical attack is related to the level of parasitaemia.[1, 2, 4] The merozoites of plasmodium falciparum will invade erthyrocytes of any age. There is decreased deformability of the paratised red blood cells. High parasite concentrations and diminished red blood cells compliance produce reductions in capillary flow with resulting tissue hypoxia. The brain, kidneys and placenta are especially vulnerable. Severe hemolysis, renal failure, coma, pulmonary oedema and intrauterine fetal death may be the eventual sequelae [1, 2] This patient had marked haemolysis as evidenced by the low haemoglobin, reticulocytosis and hyper-bilirubinaemia.

The mechanisms of anaemia causation are multifactorial. This include hemolysis, bone marrow dyserrythropoiesis and folate deficiency.[1, 2] Malaria produces haemolysis when parasited erthyocytes rupture, and other cells are constantly removed from circulation by the lymphoid macrophage system. Nevertheless, this may not be sufficient to account for the degree of anaemia seen. Auto-immune haemolysis is thought to be responsible for the accelerated haemolysis and subsequent anaemia .[1, 7, 8]

Plasmodium falciparum infected pregnant women are at a particular risk of developing hypoglycaemia. Treatment with quinine will exacerbate hypoglycaemia.[1, 2, 8] Falciparum malaria commonly induces uterine contractions which may lead to preterm labour or abortion. Pyrexia is also implicated in the genesis of uterine contractions [2, 7, 8] Other complications include pulmonary oedema and increased susceptibility to bacteria infections. This patient had haemolytic anaemia, pyrexia and went into preterm labour.

The principle of management involves treating the acute infection and detection and treatment of the complications of the plasmodium infection. This patient received Artenum chemotherapy but went on to have a pre-term delivery. She had a relapse in about one week of treatment. Drug resistance in malaria has been defined as the ability of a parasite strain to survive and /or multiply despite the administration and absorption of a drug given in doses equal to or higher than those initially recommended but within the limits of tolerance of the subjects.[8] This patient had resistance as evidenced by the fact that she received full course of Artenum as recommended but continued to have parasitaemia. The levels of resitstance RI, RII, and RIII is based on serial response of parasites to a schizonticidal drug.[10] The type of resistance for this patient was not known as sequential parasite counts was not undertaken.

Quinine is the treatment of choice where resistant infection is encountered. The patient received quinine 600mg 8 hourly for 10 days which cleared the infection. She was transfused 3 units of whole blood. The baby was admitted to nursery and did not develop signs of congenital infection.

REFERENCES

1. Cunningham F.G. et al.Infections in Williams Obstetrics 21st Ed. Appleton and Lange New York pg. 1477,2001.

2. Nathwani D., Currie P.F., Douglas J.G. et al. Plasmodium falciparum malaria in pregnancy: A review. Br. J. Obstet. Gynecol 99:118; 1992.

3. Ministry Of Health. Medical conditions in pregnancy.Essential Obstetric care manual for Health Service providers in Kenya,Ministry of Health, Nairobi.148-152,2002

4. Shulman C., Dorman E. Malaria in pregnancy.Africa Health, 22(6):26, 2000.

5. Rukaria R.M. In vivo and in vitro response of plasmodium falciparum to chloroquine in pregnant women in Kilifi District, Kenya. M.Med thesis, U.o.N. 1990.

6. Report of Director General WHO 1998

7. Bakri Y.N., Martan A., Amri A. Pregnancy complicated by malaria, precipitate labour and uterine rupture. Intern. J. Gynaecol.Obstet. 38(3):231,1992.

8. Wilfer J.D. Malaria in Principles and practices of infectious diseases. 3rd Edition. Churchill Livingstone (Publishers, Edinburgh), 1990

CASE NO. 13: CARDIAC DISEASE IN PREGNANCY
VACUUM DELIVERY – LIVE BABY

Name: D.M.	D.O.A: 9/2/2003
I.P: 0850787	D.O.D: 14/7/2003
Age: 30 years	L.M.P: 14/9/2002
Parity: 2+1	E.D.D: 21/6/2003

Present History

The patient was referred from the cardiac clinic and was admitted through the antenatal clinic for cardiac assessment and subsequent antenatal care as an in-patient until delivery. She had complaints of dyspnoea on performing ordinary domestic chores.

Obstetric and Gynaecologic History

She was para 2+1. Her first delivery was in 1997 by assisted vacuum delivery, with a birth weight of 2.8kg. Her second delivery was a macerated still birth at 7 months in 2002 after intrauterine fetal death. In 2000, she had a spontaneous abortion at 5 months. Her last menstrual period was 14/9/2002 and hence expected date of delivery was 21/6/2003. Her periods had been regular occurring every 28 days and lasting 3 days. She was at 20 weeks of gestation on admission. She had not previously used any contraceptive method but indicated a desire for tubal ligation after successful completion of the present pregnancy.

Past Medical and Surgical History

She was a known case of cardiac disease since 1993 and was being followed up in cardiac clinic with Rheumatic heart disease. The valvular lesions were mitral stenosis and incompetence. She had suffered a thromboembolic cerebravascular accident in 1994 which resulted in left hemiplegia for two weeks which has left hemiparesis to date. In 1995, she had been admitted due to cardiac failure. She was stable before this pregnancy with only mild limitation of physical activity. She had been on Digoxin 0.25mg once daily and monthly Benthazine penicillin 1.2MU.

Family and Social History

She was married and worked as a clerk. She did not smoke or take alcohol. There was no family history of any chronic illness.

Examination

She was in good general condition. She was not pale, not febrile and had no oedema. She did not have jaundice, lymphadenopathy, finger clubbling or splinter haemorrhages. Her pulse rate was 80 per minute regular and of good volume. The blood pressure was 110/70mmHg.

Central Nervous System

She was fully conscious and was well oriented in time, space and person. She had left sided hemiparesis.

Cardiovascular system

The pulse was 80 per minute, regular and of good volume. The jugular venous pressure was not raised. The praecordium was not hyperactive. The apex beat was in the 5^{th} intercoastal space at the midclavicular line. First and second heart sounds were heard. There was a pansystolic murmur and a mid-diastolic murmur best heard at the mitral area.

Respiratory System

There was normal chest expansion bilaterally. There was bilateral vesicular breathing and no crepitations.

Abdominal Examination

The abdomen was uniformly distended and moved with respiration. The liver and the spleen were not palpable. The uterine fundus was responding to a 22 week gestation. Fetal movements were felt.

Diagnosis

Cardiac disease grade IV in pregnancy at 20 weeks gestation.

Antenatal Management

She was admitted in antenatal ward and managed by an Obstetrician in consultation with the cardiologist. Routine antenatal profile to include blood group, VDRL and haemoglobin were done. She was advised on minimal exercises and adequate bed rest. She was maintained on Digoxin 0.25mg orally once daily. She continued on her monthly benzathine penicillin 1.2MU given intramuscularly. A full haemogram and urea and electrolytes were repeated weekly and two weekly respectively.

The vital signs were monitored recording the pulse, temperature and pressure. The chest was also examined daily for any basal crepitations. Echocardiogram and electrocardigram were obtained as part of cardiovascular assessment. Contraception was discussed and the patient opted for bilateral tubal ligation postpartum. At 36 weeks of gestation clinical pelvimetry was undertaken and this revealed an adequate pelvis. She was to await spontaneous labour and assisted vacuum delivery.

Results of investigation.

Haemogram	-	repeated severally and remained normal
VDRL	-	Negative
Blood group	-	A rhesus 'D' positive
Urinalysis	-	Normal – repeated severally
Urea/Electrolytes	-	remained normal on several repeated specimens
Chest X-ray	-	No evidence of cardiomegally
	-	Lung fields were normal
Echo Cardiogram	-	Thickened mitral valve apparatus. Large left atrium and Left ventricular cavities with good left ventricular function.
Doppler	-	Mitral stenosis with mitral regurgitation. Left atrium Measurement – 4cm.
Electrocardiogram	-	A notched prolonged 'P' wave with a prominent negative Deflection – features suggestion of left atrial enlargement.

Intrapartum Management

She was transferred to labour ward on 1/7/93 at 3 p.m. She had been in labour for 3 hours

before notifying the nurses. She was not draining and had no vaginal bleeding. On examination she was in good general condition, the pulse was 82 per minute, it was regular and of good volume. The blood pressure was 120/80mmHg. The chest was clear with a respiratory rate of 22 per minute. The fundal height corresponded to a term gestation, the fetus was in longitudinal lie and was in cephalic presentation with only 3/5 of the head above the pelvic brim. The fetal heart was heard at 138 per minute and was regular. She was having 3 strong contractions in 10 minutes lasting 30 – 40 seconds. The cervix was 5 cm dilated and fully effaced. Artificial rupture of membranes was done obtaining clear liquor. The position was left occiput anterior. She was placed in the semi Fowler's position. She was given 2gm of ampicillin and 80mg of gentamycin intramuscularly as prophylaxis against bacterial endocarditis. She was put on partogram. Oxygen was given by mask and she received a 100mg of pethidine intramuscularly.
She progressed well and at 7 p.m. She had full cervical dilation. She was placed in the delivery couch in the semi-recumbent position and advised not to bear down. A left mediolateral episiotomy was made after infiltrating the site with 2% lignocaine. A medium sized cap was applied on the vertex and connected to the vacuum apparatus. Vacuum was gently created and gently applied with every contraction. In the second contration after applying the cap, an easy assisted vacuum delivery was achieved. She was given 80mg of frusemide intravenously. The placenta was delivered by controlled cord traction and the uterus contracted spontaneously. The cervix and the vagina were inspected and noted to be intact. The episiotomy was repaired; the estimated blood loss was 200mls.There was no immediate post partum complication. The pulse was 88 per minute, the respiratory rate was 24 per minute and she was not dyspnoiec. She was transferred to labour ward acute room for observations.

Post-Partum Management

She remained stable for 24 hours where vital signs were observed half hourly after which she was transferred from acute room to the post natal ward.
Vital signs were observed 4 hourly, monitoring closely for any signs of infections. She was put on prophylactic ampicillin 500mg orally given 6 hourly for 5 days. Post-partum haemoglobin was 11.6g,/dl. On the 10^{th} postnatal day she was reviewed by a cardiologist and found to be stable. She remained in the wards for 14 days, where her post-partum observation chart remained normal including uterine involution and lochia loss.

Due to unavailability of theatre, the patient was discharged for tubal-ligation as an outpatient.

Post Natal Follow-up

The patient was seen in the postnatal clinic 6 weeks post partum. She had no complaints. The uterus was well involuted. The breasts were active and not engorged. She had a booking for minilaparatomy for tubal ligation. She was referred back to the cardiac clinic for life time follow-up.

COMMENT

The patient presented had cardiac disease grade IV according to the New York Heart Association (NYHA) classification. She had an assisted vacuum delivery with good outcome.

A varying incidence of cardiac disease in pregnancy has been reported in various parts of the world. It is said to complicate about 1% of pregnancies.[1] A study done in Kenyatta National Hospital showed an incidence of 0.66%.[2] A similar incidence has been noted in other developing countries.[3] This patient had rheumatic heart disease. Rheumatic heart disease is the commonest heart disease seen in pregnancy in many parts of the World. In Ngotho's series at Kenyatta National Hospital, 80% of the patients with cardiac disease in pregnancy had rheumatic heart disease, congenital heart disease comprised of 12.9%.[2] In the developed countries, the incidence of rheumatic heart disease has declined. One of the principal reasons for the decline has been the availability of treatment modalities for strepotococcal pharyngitis; the other is improvement of overall living standards .[4]

Clinically important rheumatic cardiac lesions are predominantly valvular in nature. The mitral valve is, by far, the most commonly affected valve, followed by aortic valve. Mitral stenosis occurs in approximately 73.5% of patients and is the most dangerous condition. Mital regurgitation occurs in approximately 6.6% of patients.[2]

Rheumatic heart disease is the long-term sequaelae of rheumatic fever. The causative organism of rheumatic fever is the lancified group A streptococcus. Group A streptococcus produces substances that contribute to their pathogenicity. The theory is that rheumatic fever is an auto-immune disorder in which tissue damage is mediated by the host own hyperimmune response to the antecedent streptococcal infection .[1, 4]

Mitral stenosis is the most common lesion as well as the most important hemodynamically.[1] The primary physiological abnormality of blood flow from the left atrium to the left ventricle is that produced by a narrowed valve orifice. Elevated atrial pressure is accompanied by increases in pulmonary venous and capillary pressure thus reducing pulmonary compliance and causing exertional dyspnoea. The first episodes of dyspnoea usually are precipitated by clinical events that increase the rate of blood flow across the mitral orifice.[4]

During pregnancy widespread circulating changes occur and results in increase of the workload of the heart. Previously unrecognized cardiac lesions are diagnosed for the first time in pregnancy. Infact, 25% of women with mitral stenosis have cardiac failure for the first time during pregnancy.[1] Ojiambo and Sequira found that 30 out of 35 cases of heart disease patients who were pregnant at Kenyatta National Hospital were first diagnosed during pregnancy.[3]

The diagnosis of heart disease is made more difficult due to many physiological changes of normal pregnancy.[1] This patient was already a recognized case of cardiac disease having been on follow-up for heart disease and hence presented no diagnostic difficulty.

There is no clinically applicable test for accurately measuring functional capacity of the heart. The New York Heart Association (NWHA) has provided a clinical classification which is based on past and present disability. The NYHA classifies cardiac disease in pregnancy as follows[1, 4]:-

Class I:	Uncomprimised patients with cardiac disease and no limitation of Activity.
Class II	Slightly compromised patients with cardiac disease and slight limitation of physical activity.
Class III	Markedly compromised patients with cardiac disease and marked limitation of physical activity.
Class IV:	Patients with cardiac disease and inability to perform any physical activity without discomfort.

The principle symptom of mitral stenosis as was the case with this patient is dyspnoea, which reflected reduced pulmonary compliance and vital capacity.[4] In severe disease, orthopoea and paroxysmal noctural dyspnoea may supervene.[4] A diagnosis of cardiac disease is made when a harsh or loud systolic murmur is heard, when there is unequivocal cardiac enlargement or if serious arrhythmias are present.[1] The patient had dyspnoea on less than ordinary physical activity and on examination she had a loud pansystolic murmur best heard at the apex. She was classified as cardiac disease grade IV due to history of cardiac failure. Diagnostic aids to cardiac disease include: a chest x-ray, electrocardiogram, echocardiogram and cardiac catheterization.[4] In this patient the electrocardiogram revealed signs of mitral stenosis while echocardiography showed both mitral stenosis and regurgitation and enlarged left atrium.

Management of patient with cardiac disease in pregnancy should be started early in pregnancy. Early attendance of the antenatal clinic is vital for early detection of the nature of the cardiac lesion, assessment of severity of the disease and introduction of appropriate measures to prevent complications. Attention by both obstetrician and cardiologist is vital.[1, 4, 5, 6] At the time of initial obstetric visit, a patient with heart disease should be classified according to the functional classification of the New York Heart Association.[1, 4] This system serves as an accurate guide to maternal prognosis. The maternal mortality associated with heart disease increases directly with specific functional class.[4] This patient was functionally grade II but with the history of cardiac failure as stated earlier, she was classified as class IV.

Most patients who present at their initial visits with a diagnosis of rheumatic heart disease are already receiving antibiotic prophylaxis against recurrent rheumatic fever.[4] This patient had been receiving Benzathine penicillin monthly. The recommended regimens for antibiotic prophylaxis are Benzathine penicillin 1.2 mega units I.M every 4 weeks or penicilling G 200,000 units-twice daily. For patients allergic to penicillin, sulphadizine 1gm daily or Erythromycin 250mg twice daily suffices.[4]

One of the cornerstones of management during pregnancy is to restrict physical activity and thus reduce strain on the cardiovascular system.[1, 4, 5, 6] Other factors that predisposes to cardiac failure must be prevented, these include anaemia, infection, excess weight gain and sodium intake.[1, 4, 6]

Those patients in grade I and II may be managed as outpatients. They are seen fortnightly until 36 weeks of gestation when they should be admitted. Patients of grade

III and IV are admitted on the initial visits for more vigilant surveillance[1] This patient was admitted on first contact and remained in the hospital till delivery.

Patient with rheumatic heart disease should be allowed to enter spontaneous labour at term. Labour is only induced for obstetric indications. Delivery should be accomplished vaginally and caesarean sections reserved for obstetrical indications[1,4] For patients with rheumatic bacterial endocarditis at the time of delivery, the American Heart Association recommends Ampicillin 2.0g and gentamycin 1.5mg/kg IV given at least 30minutes before delivery and repeated 8 –12 hours later[1,4]For patients allergic to penicillin, Vancomycin 1gm IV is given 60 minutes before delivery and repeated 8-12 hours later[4] This patient received ampicillin and gentamycin and she was continued on ampicillin post natally for 5 days. Adequate intra-partum analgesia is important to relieve pain and apprehension which is known to elevate heart rate and predispose to failure. Intramuscullar injection of 15mg of morphine is recommended but our patient received 100mg of pethidine as morphine was not available. Lumbar epidural anaethesia is seldom used. Its risks include hypotension and increase in venous capacitance [1,4] This technique is not practiced in our unit.

During labour the mother should be kept in semi Fowlers position. Measurements of the pulse and the respiratory rates should be taken quarter hourly during the first stage of labour and every 10 minutes in the second stage. Oxygen given by mask has an advantage to both mother and the fetus. The progress should be diligently followed with vaginal examination every four hours to monitor progress of labour[1,4] Continuous electrocardiogram, central venous pressure and blood gas studies are in some centres a routine during labour for patient with cardiac disease.[4] The periods of maximum risk for patients with cardiac disease are second stage and the immediate puerperium. During these periods women must be monitored for signs of heart failure, hypertension and arrthmias. To expedite second stage a prompt forceps delivery or assisted vacuum delivery are indicated. With the delivery of the baby, a strong diuretic is given to circumvent circulatory overload. This patient was given frusemide 80mg intravenously. It is essential to prevent excessive blood loss in the third stage of labour. The placenta should be delivered by controlled cord traction; vigorous manual message of the uterus to achieve uterine contraction should be performed. If need be, oxytocin should be administered by continuous infusion rather than intravenous bolus in order to prevent abrupt increase in blood pressure. Ergot alkaloids should be shunned as they produce marked elevation of the central venous pressure and transient hypertension.[1,4] This

patient was given oxytocin and had only uterine massage to achieve contraction of the uterus and hemostasis.

Whereas it is well established that the women with cardiac disease who receive appropriate care rarely die during pregnancy or puerperium, the possibility causes obscure deleterious affects that ultimately shorten her life span. Nevertheless some studies indicate that if a woman survives pregnancy, there are no deleterious remote effect of the rheumatic heart disease.[1] In the consideration of the prognosis, it is noteworthy to state that as pregnancy progresses, the demands on the heart increases. The elevated cardiac output is maintained predominantly by an increase in the heart rate. This predisposes to cardiac arrhymias and hence failure and thrombeombolism.[4] This patient had suffered a thromboembolic cerebral vascular accident prior to starting her obstetric career. Although cerebral vascular accidents (CVA) are rare in otherwise healthy young women, the patient had rheumatic heart disease and this explains the CVA.

REFERENCES

1. Cunningham F.G., MacDonald P.C., and Grant N.F. Maternal adaptation to pregnancy and Cardiovascular disease. Williams Obstetrics 21st Ed. Appleton and Lange/McGraw Hill New York pg 1182-1203, 2001.

2. Ngotho D.K. Cardiac Disease in pregnancy at KNH. A 10 year retrospective study 1970 – 1979. M.Med Thesis, UoN 1982.

3. Sequiera R.F. and Ojiambo H.N.P. The Heart in Pregnancy East Africa Med. J. 46:18, 1969.

4. Brady K. and Duff Patric. Rheumatic Heart Disease in Pregnancy.Clinical Obstetrics and Gynaecology 32 (1):1989.

5. Whitfield C. R. Heart in pregnancy. Dewhurst's text book of obstetrics and gynaecology for postgraduate. 4th Ed. Blackwell scientific publications/ELBS London pg 242, 1986.

6. Biswas M, Perloff D. Cardiac, Haematologic, Pulmonary, Renal and Urinary Tract disorders in pregnancy. Current Obstetrics and Gynaecologic Diagnosis and Treatment. 9th Ed. Appleton and Lange/McGraw Hill New York pg 428 ,2003.

7. Smoke G.A., Cox S.M., Cunningam F.G. Cerebrovascular accident complicating pregnancy and the puerperium. Obstet. Gynaecol. 78 (1): 37, 1991.

CASE NO. 14: HIV INFECTION WITH FETAL DISTRESS : CAESAREAN SECTION – LIVE BABY

NAME:	E.M	PARITY:	2+0
AGE:	28	L.MP:	10.1.03
IP NO:	0931876	E.D.D:	17.10.03
D.O.A	29.10.03	Maturity:	41 weeks and 5 days
D.OD:	4.11.03	L.D:	2001

Presenting complaints

The patient was admitted from home at 8 a.m. on 29.10.03 with complaints of lower abdominal pains and lower backache since midnight.The pains were intermittent and progressing both in frequency and intensity. At the time of admission she reported two contractions in 10 minutes.

Past Obstetrical and Gynaecology History

She attained menarche at 14 years. Her menses were regular with flows of 4 days and cycles of 28 days. Her last delivery was in 2001 to a male infant who weighed 3.4 kg at birth and was alive and well. The last menstrual period was on 10.1.03 and the expected date of confinement was on 17.10.03, so she was at a gestation of 41 weeks and 5 days on admission.

She attended antenatal care at Kenyatta National Hospital where she was booked at 20 weeks. The follow up was uneventful with a weight gain from 55 kg at 20 weeks to 60 kg at 39+ weeks. The antenatal profiles were within normal. She was put on zidovudine300mg b.d from 34 weeks gestation up to the onset of labour.She used an IUCD after the last delivery upto the time she stopped to conceive the index pregnancy.

Past Medical and Surgical History

The patient underwent HIV testing in 2000 after her husband tested positive for HIV while undergoing treatment for pulmonary tuberculosis. However she had not suffered any major illness e.g. tuberculosis, pneumonia, herpes, prolonged fever or diarrhoea. She

had not been on anti-retroviral therapy at anytime. She had never been transfused blood or used intravenous illicit drugs.

Family and Social History

The lady was married and did business of selling clothes. The husband worked as a mechanic and they lived at Kayole. There was no history of promiscuity or multiple sexual partners before or in marriage. She was not very sure of her husband's sexual exposure and habits, however he drunk alcohol and smoked.

Physical Examination

The patient was in good general condition, afebrile, not pale, and had no oral thrush. The blood pressure was 110/70 mmHg, pulse rate of 88/min, respiratory rate of 24/min, temperature 36.8^{0}C. The central nervous, cardiovascular and respiratory systems were normal.

Abdominal Examination

There was normal abdominal fullness. The fundal height was term, in longitudinal lie and cephalic presentation. The head was four fifths above the pelvic brim with a fetal heart bradycardia and irregularity at 100-115/min.

Vaginal Findings

The external genitalia was normal with no warts or genitalia ulcers. The cervix was anterior, soft, thin and fully effaced with a cervical dilation of 4cm. Artificial rupture of membranes was not done at this stage due to HIV status of the patient.

Diagnosis

HIV infection with fetal distress.

Investigation done antenatally

Blood group – A+ve

Haemogram – 12.7gm/dl

VDRL - Negative

Management

The decision was made to deliver the patient by caesarean section, which was communicated to her and she gave consent. An intravenous line was established and 5% dextrose was started. Oxgen by mask was started, patient monitored with the tococardiography and placed in the left lateral position. Blood was drawn for cross-matching two compatible units of blood. She was pre-medicated with intramuscular atropine 0.6mg then wheeled to theatre.Niverapine 200mg was given orally.

Caesarean Section

The patient was placed in dorsal position on the operation table then vulvo-vaginal toilet done. She was draped then catheterized and 200mls of clear urine was obtained. She was repositioned in supine position and the abdomen cleared with savlon then betadine. She was draped and then anesthesia administered. A pfannenstiel incision was made through which the abdomen was opened in layers. The peritoneal cavity was entered then lower uterine segment was identified. A transverse incision into the utero-vesical peritoneum was made. With the aid of a swab on sponge holding-forces the urinary bladder was pushed down. At a point estimated to be 2cm above the reflection of the utero-vesical peritoneum an elliptical incision was made into the lower uterine segment, and then extended laterally adequate enough to allow easy delivery. The membranes were ruptured and quickly a male infant was delivered in a left occipital position, then immediately the amblical cord was clamped and divided. The baby was thoroughly cleaned off all secretions with diluted savlon. These steps were aimed at preventing maternal-to-fetal transmission. A male infant was delivered with an apgar score of 10 at 1 minute, 10 at 5 minutes with a birth weight of 3.5 kg from a meconium stained and normal cord vessels were delivered by controlled cord traction. The uterine cavity was cleaned and normal viscera, ovaries and tubes. The instruments, abdominal packs and gauze were counted and found to be correct then the abdomen was closed in layers with chronic catgut No. 1 for peritoneum, vicryl No. 1 for rectus, plain catgut No. 2-0 for fat layer and vicryl 2-0 for the skin. The wound was cleaned, dressed and anaesthesia reversed. Vulvo-vaginal toilet revealed that the catheter was draining clear urine and was removed. The total blood loss was estimated to be 500 mls.

Post Operative Care

The patient was transferred to the ward fully awake. The blood pressure, respiratory rate, pulse rate and temperature being observed four hourly. Hydration was by intravenous normal saline alternating with 5% dextroxe 500mls every four hours. The antibiotic cover was of intravenous gentamycin 80mg eight hourly and crystalline penicillin 2mu six hourly. Post operative analgesia was provided by intramuscular pethidine 100mg six hourly. On the second post operative day she was stable with normal vital signs, and restored bowel activities hence she was started on oral sips, oral amoxicillin 500mg eight hourly, oral Iboprofen 400mg eight hourly and Bromocriptine 2.5mg 12 hourly with IV fluids and crystalline penicillin being stopped. During the antenatal care she had been counseled against breast feeding and she was advised to perform cold towel breast massage in order to prevent breast engorgement. Throughout the post operative period she remained well with no evidence of sepsis or breast engorgement or mastitis. The wound was exposed on the fifth post operative day was clean and healing well. The sutures were trimmed on the 7th post operative day, the wound was ensured to be well healed. The baby did not receive (as the policy of the institution) BCG vaccine but got polio and was to get the rest of polio and DPT vaccines. The baby's blood was drawn on the second day of life and tested positive for HIV, this was to be repeated at 15 weeks of age during follow up. The mother and baby were discharged on 4.11.03 in good condition. She was advised on breast care and referred to patient support centre for HIV follow up in one week for the baby and herself.

Discussion

It was late 1985 when testing for the human immunodeficiency virus (HIV) first became widely available. Most obstetricians became aware of the problem only after that time. In the subsequent decade an enormous global effort has been mounted to combat the disease[1]

Different institutions or centers have recommended various modes of HIV testing of antenatal mothers. At our unit it is now a policy to screen all antenatal mothers using the rapid test after counselling. Similarly certain centers recommended testing to all women attending antenatal clinics. The patient presented was tested in 2000 as a spouse of an

HIV patient. This was by the opt-in system of testing where adequate information was provided and expert counselling for her as a high risk person[3]. There are other systems of counseling that would be used for example opt-out system (anonymous) where all pregnant women are tested unless they object[4] and the mandatory system of screening of all pregnant women[5].

Studies from Africa have shown reduced birth weight in the pregnancy of HIV infected women[1]. The patient presented did not have any complication and delivered a baby with a good apgar score and a birth weight of 3.5kg.

Increased focus of attending on the availability of HIV testing at antenatal clinics is based on the following facts; The fact that much vertical transmission may be prevented and that it accounts for vast majority of paediatric HIV infections. The estimated rate of vertical transmission varies from 15%-20% in Europe, 15%-30% in USA and 25%-35% in Africa[1]. It is widely believed that the likelihood of vertical transmission is related to viral load, and therefore it is greatest early and late in the natural history of infection[1]. There is no doubt that the fetus can be infected during pregnancy, though one large study suggests that, contrary to what has previously been described, early in utero HIV infection may be very uncommon[1]. There are now clear advantages for the fetus in knowing a woman's HIV status. The advantages for the woman may be more contentious but as evidence builds that combination therapy is effective, the benefits from her point of view increase. Women who want testing should therefore have easy access to the same[1,6].

As an estimated two-thirds of transmission occurs around the time of delivery in non-breastfeeding populations[7], elective caesarian section delivery and douching of the birth canal have been suggested as other approaches to reduce acquisition of HIV. However, there is conflicting evidence regarding the effectiveness of caesarian section delivery in reducing transmission. A Zambian randomized controlled trial showed that douching the birth canal only significantly reduced transmission when the time between rupture of membranes and delivery exceeded four hours [8]. In our patient the measures undertaken to prevent or reduce vertical transmission included rupturing membranes when the baby was

being delivered, rapid clamping of cord immediately the baby was delivered and cleaning the baby thoroughly with savlon off any secretions. This is recommended by Towers[9] . Ocular or pulmonary surfaces are other possible sites of entry of the virus. In the case of twin gestation, the first twin is much more likely than the second twin to be the one affected, suggesting that it is the degree of exposure to be infected maternal blood and cervical mucus during delivery which is the main determinant of transmission[1]. A recent US study shows a doubling of risk if the membranes are ruptured for more than four hours, strongly consistent with intrapartum transmission[12].

There is evidence that elective caesarean section reduces the risk of vertical transmission[1] Our patient had opted for vaginal delivery after being counselled and caeserian section was done due to fetal distress. At caesarean section meconium stained liquor grade 2 was found, however the baby had a good apgar score..

Breastfeeding approximately doubles the risk of transmission (adds another 7%-22% risk of infection), and HIV infected women are therefore advised to refrain from breastfeeding where safe alternatives are available[7]. Our patient opted not to breast feed the baby, instead she was provided with formula milk (Na).

Avoidance of breast feeding together with zidovudine(AZT) therapy during pregnancy, delivery and in the neonatal period have a major impact on mother-to-child transmission of HIV as shown in Zidovudine trial[10]. In this study women who were 14 to 34 weeks pregnant, who had CD_4 count more than 200×10^9/L, who had not taken AZT during pregnancy, and who did not have abnormal liver function test or HIV related illness were recruited. Treatment with oral Zidovudine 100mg five times daily or placebo was started between 14 and 34 weeks of pregnancy. Women also received Zidovune or placebo intravenously during labour 2mg/kg body weight over one hour period, followed by a continuous infusion of 1mg/kg until delivery and their infants received Zidovune syrup (2mg/kg four times daily for six weeks, beginning 8-12 hours after birth). In other centres all infected women were routinely prescribed combination anti-retroviral therapy compared with Zidovune monotherapy in delaying progression of diseases in HIV infectected adults[7].

The effect that a pregnancy has on a woman's HIV diseases state is an important consideration when counseling women infected with HIV about planned or future pregnancies. Although a woman's decision making process may be concerned with the effect on the baby, the effect a pregnancy may have on her own survival is a major factor[11]. In addition , in developing countries infant survival has been shown to be adversely affected by maternal death with infants being over three times more likely to die if their mothers had died. The patient presented had been counseled against pregnancy after testing positive for HIV but the next time she was seen she was at 20 weeks of gestation.

The contraception of choice will be the condom that will in addition prevent further infection with an increasing viral load and new strains. Our patient is currently being follow up in the HIV clinic.

REFFERENCES

1 Frank D., Johnstone D. Review: HIV in pregnancy.
Br.J. Obstet. Gynaecol., Vol.103: pp 1184-1190, Dec 1996.

2 Dept. of Health. Guidelines for offering voluntary named HIV antibody testing to women receiving antenatal care. London, HMSO, 1994.

3 Centres for Disease Control – US Public Health Service recommendation for human immunodeficiency virus counseling and voluntary testing for pregnant women. MMWR
44:1-15, 1995.

4 Boyd K.M. HIV infection : The ethics of anonymized testing of pregnant women. J. Med Ethics ; 16 : 173-178, 1990.

5 Wilfert C.M. Mandatory screening of pregnant women for the human immunodeficiency virus. Clin. Infect. Dis.; 19:664-666, 1994.

6 Duffy T.A., Wolfe C.D.A, Varden C. et. al.
Women's knowledge and attitudes, and the acceptability of voluntary antenatal HIV testing. Brit.J. Obstet. Gynaecol., Vol. 105: pp 849-854, August 1998.

7 Newell M.L. Therapeutic and other interventions to reduce the risk of mother-to-child transmission of HIV-I in Europe. Bri. J. Obstet. Gynaecol. Vol. 105: pp 704-709, July 1998.

8 Biggar R.J., Muotti PG., Taha T.E et al. Perinatal intervention trial in Africa: Effect of a birth canal cleansing intervention to prevent HIV transmission. Lancet; 347: 1647-165, 1996.

9 Towers C.V., Develks A., Astrat T et al. Role of "blood less caesarean section" to decrease maternal child HIV transmission: a pilot study [abstract]. IXth International conference on Aids. Abstract No: PO-C162978, Berlin, 1993.

10 Connor E.M., Sperling R.S., Gelber R. et al. Reduction of maternal-infant transmission of human immuno-deficiency virus type-I with Zidovune treatment.
N. Engl. J. Med ; 331 : 1173-1180, 1994.

11 Rebeccah Frenah, Peter B. The effect of pregnancy on survival in women infected with HIV: a systematic review of the literature and meta-analysis. Brit. J. Obstet. Gynaecol., Vol. 15: pp 827-835, August 1998.

12 Landesman SH., Kalish LA., Burns DN. et. al.
Obstetrical Factors and the transmission of Human Immunodeficiency Virus Type-I from mother to child. N. Engl Med ; 334: 1617-1623, 1996.