CASE REPORTS AND LONG COMMENTARIES IN OBSTETRICS AND GYNAECOLOGY

SUBMITTED BY

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DEDICATION

This work is dedicated to my wife Cecilia Wahinga, my daughter Angela Wanjiku, my late son Andrew Kimani for your continued love and care.

And...

To my dad Reuben K. Muni and my mother Irene Wanjiku for inspiring me to take a career in medicine. To my sister and brothers; Caroline Njambi, Benson Ngugi and Josiah Njore for your support and encouragement.

... with this course, hopefully, I have fulfilled a small bit of each one of your dreams.

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Finally I wish to thank all the special people in my private life for their kind support and love without who this work would perhaps not have seen the light of the day. God bless you all abundantly.

DECLARATION

This is to declare that the long commentaries and case records presented in this book are my original work and were managed by me under the supervision of the senior members of the Department of Obstetrics and Gynaecology, University of Nairobi and Kenyatta National Hospital.

The Obstetric and Gynaecology short and long commentaries in this book have not been presented for a degree in any other university.

DR. JOHN KARANI KIMANI, MB. ChB. (University of Nairobi)

SIGNATURE  .

DATE 09/12/05 .

CERTIFICATION

This is to certify that Dr. J.K. Kimani managed Obstetric cases Nos. 5,6,9,10,11 and 14 and, Gynaecology cases 1,2,4,6,8,11,12,13,14, and 15 under my supervision at Kenyatta National Hospital.

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Professor

Department of Obstetrics and Gynaecology

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SIGNATURE  .

DATE 13/12/005.

CERTIFICATION

This is to certify that Dr. J. K. Kimani managed obstetric cases Nos. 1,2,4,8, and 13, and Gynaecology cases Nos. 5, 9, and 10 under my supervision at Kenyatta National Hospital.

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Department of Obstetrics and Gynaecology
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SIGNATURE 

DATE 13.12.05.

CERTIFICATION

This is to certify that Dr. J. K. Kimani managed obstetric cases 3,7,12, and 15 and, Gynaecology cases Nos 3, and 7 under my guidance and supervision at Kenyatta National Hospital.

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SIGNATURE Wanjala

DATE 13/12/05

INTRODUCTION

KENYATTA NATIONAL HOSPITAL

Kenyatta National Hospital (KNH) is situated about 4 km on the western side of the central business district of the City of Nairobi. It was started as a small hospital called the Native civil Hospital in 1901 when Kenya was a British colony. It was changed to King George V Hospital and later in 1970 to its present name Kenyatta National Hospital. It was made a parastatal body in 1987 and has continued to serve as National Referral Hospital in the country; referrals are mainly received from all public and private health institutions in the country as well as from neighbouring countries. The hospital provides preventive, curative, promotive, specialised and rehabilitative services.

This hospital has been offering teaching facilities for the College of Health Sciences since the inception of the Faculty of Medicine in 1967. This is at both undergraduate and postgraduate levels. Research facilities are also found within the hospital complex mainly under the University of Nairobi's faculties of Medicine, Pharmacy and Dentistry. It also acts as a teaching facility for nurses and paramedical staff.

Obstetric services

The obstetric services in this hospital are provided in the antenatal clinic, maternity unit and the post-natal clinic.

The Antenatal Clinic (ANC)

The antenatal clinic at KNH caters for both patients in high risk category and for general antenatal mothers. These patients are booked into the clinic every Monday morning alternately by each of the three firms. The patients are first interviewed by the clinic midwives who record personal history, obstetric history, medical and surgical history. Blood pressure, weight, height measurements and urinalysis are done on all patients. Normally, about fifty patients and a few staff members are booked per session.

A detailed medical, obstetric, gynaecological, family and social history are taken for the patients who are booked for follow up. An initial thorough physical examination is performed. Subsequently, follow-up examinations to include uterine size, fetal lie, presentation and fetal heart tones are done. Blood for antenatal profile for haemoglobin estimation, blood group and rhesus and serology for syphilis is taken.

Voluntary confidential counselling is done to every client and testing test for HIV is encouraged and performed as part of antenatal profile test. Those who are found to be HIV infected are put on zidovudine from the 34th week of gestation to term. They are also advised on the options available of feeding the infant after delivery.

Other tests relevant to individual patients e.g. random blood sugar, indirect Coomb's test (ICT), obstetric scan, etcetera, are requested as appropriate. The patients are then given appointments for subsequent visits; those for immediate admission are sent to the antenatal wards through the labour ward.

Follow up

The focused antenatal care method is being introduced where the clients visit the clinic for follow-up at least 4 times in there entire gestation. However, the traditional method is also used where clients are seen every four weeks up to 28 weeks gestation, two weekly up to 36 weeks gestation and then weekly thereafter until delivery. Patients may be seen more or less often depending on individual needs. During each visit, the following are recorded:

- Weight.
- Blood pressure measurement.
- Urinalysis for protein and sugar.

Complaints are also sought for form the client and management/treatment is provided as appropriate for individual cases. Abdominal examination is done during each visit and compared with previous findings and the calculated gestation. The fetal lie, presentation and heart tones are ascertained.

At 36 weeks gestation, a clinical pelvic assessment is done on all primigravida and at 37 weeks, radiological pelvimetry is done on patients with one previous Caesarean section of non-recurring indication. A clinical pelvic assessment may also be done instead of the radiological pelvimetry. For those planned for elective induction of labour or repeat Caesarean section, amniocentesis may done at 38 weeks gestation for assessment of fetal lung maturity.

During each visit, the clinic midwives provide health education sessions; patients are enlightened on regular clinic attendance, better nutrition and hygiene, preparation for labour and childbirth, postpartum care, family planning and STIs, HIV/AIDS.

The Maternity unit

The Maternity Unit is made up of the labour ward, three antenatal/postnatal wards, a maternity theatre, a private maternity wing, a mothers' hostel and the newborn unit. Over 6000 deliveries are conducted in the unit annually.

The Labour ward unit

The Labour Ward comprises of ten first stage cubicles each with two beds. There are two delivery suites each with two couches and a rescuscitaire. An acute room with capacity for three beds caters for very sick patients who need close observation; these include those with hypertensive disease in pregnancy, cardiac disease, severe infections, etcetera. There is an oxygen room where patients found to have fetal distress, or other conditions requiring oxygen administration by mask, are kept under observation.

Patients booked at antenatal clinic present directly to the labour ward admission desk while unbooked patients are admitted though casualty. The midwives receive the patients and records the vital signs. Clerking is done by the intern; and full physical examination by the registrar. Those patients who are not in labour and have no acute problems requiring very close monitoring are admitted to the antenatal ward. Those clients in labour or with an acute problem are admitted to the first stage area or the acute room.

Major ward rounds are done twice a day; at 8 am and 8 pm. These are attended by the consultant, senior registrar, registrar, internists and all the nursing staff on duty.

First stage of labour

Here, a record of the progress of labour is accurately kept by the doctors and the primary nursing midwife; for this purpose, a partogram is utilised. This partograph record consists of:

1. Patients identification, parity, and time of admission.
2. Date and time of rupture of membranes, whether artificially ruptured or spontaneous, and the colour of liquor.
3. Date and time of onset of labour.
4. Half hourly fetal heart rate monitoring.
5. Descent of the fetal head into the pelvis.

6. Cervical dilatation, recorded 4 hourly.
7. Uterine contractions each 10 minutes, their frequency and duration.
8. Use of oxytocin, its concentration and rate of infusion.
9. Other drugs and fluids used, dosage and time administered.
10. Maternal vital signs every 1/2 hour.
11. Urinary examinations done every 2 to 4 hourly.

All vaginal examinations are aseptically done after the bladder has been emptied. Such assessment is done every 4 hours or at shorter intervals where indicated. Artificial rupture of membranes (amniotomy) is done at a cervical dilatation of 4 cm or more if the HIV serostatus of the client is known. Active management of labour is then done aiming at delivery within 12 hours of onset of active labour. Analgesia is usually given early in the active phase of labour; intramuscular pethidine or tramadol is utilised.

Second stage of labour

This is reached at full cervical dilatation when the patient may experience an urge to bear down. The patient is then transferred to the delivery room and is placed in semi-lithotomy position, vulvoperineal toilet done and is draped. The midwife or doctor encourages the patient to bear down during uterine contractions. If the perineum is tight, 10 ml of 2% lignocaine hydrochloride is infiltrated locally or a pudendal block done and a mediolateral episiotomy is made during crowning of the fetal head. A sanitary pad is used to support the perineum as the head is delivered. The baby's mouth, nostrils and eyes are wiped with sterile gauze then a finger passed round the neck to ensure the umbilical cord is not around it. If it is, the cord is clamped at two points about 3 cm apart and cut in between. Restitution is then allowed to occur; by supporting the head between the palms of the hands. Gentle downwards traction is applied to deliver the anterior shoulder while upward traction will deliver the posterior shoulder. The rest of the body quickly follows. The cord is clamped and cut. The baby is handed over to a receiving midwife or paediatrician for Apgar scoring, weighing and resuscitation (where necessary). Syntocinon 20 IU is given in a drip or intramuscularly at delivery of the anterior shoulder. This is for prophylaxis against postpartum haemorrhage. Ergometrine is not given routinely and its use is actually discouraged.

The third stage of labour

Separation of the placenta is awaited and is indicated by a sudden gush of blood from the introitus, lengthening of the umbilical cord, and contraction of the fundus with the uterus

rising into the abdomen as the placenta passes down into the lower uterine segment and vagina. The placenta and membranes are then delivered gently by controlled cord traction. This process may also be expedited by active management of third stage of labour where the placenta is delivered by controlled cord traction prior to spontaneous separation. The cervix, vaginal walls and perineum are explored for lacerations or tears which are repaired if present. The episiotomy is then repaired using chromic catgut No. 1, in three layers. The placenta is weighed and total blood loss recorded.

Post delivery care

The vital signs are recorded and observations continued half hourly for about 2 hours. The patient keeps her bladder empty and a vulval pad is monitored. If no complications are noted, she is transferred to the lying-in-ward to continue resting and observations. If no problems arise, and the baby is well, and has been immunised, they are discharged home after 24 hours to be followed up in a post-natal clinic after six weeks.

The new born unit (NBU, Nursery)

The neonatology team comprises consultants, senior house officers and trained neonatology nurses. The NBU comprises of four sections; Nurseries A, B, C, and D. Babies are placed in Nursery A on admission. As they stabilize and weight gain is satisfactory, they are sequentially transferred to other sections until D when they are able to room-in. There is one isolation room for babies with infective conditions. Neonates admitted here include those born at home, on the way to hospital or from other centres.

Mothers hostel

Mothers who are well after delivery and have their babies in NBU are accommodated in the mothers hostel. They visit the NBU every 3 hours for breast feeding.

The cold obstetric wards (antenatal and postnatal wards)

These consist of wards GFA, GFB and IA distributed between Firms II, I and III respectively. Each ward has 32 beds shared by antenatal, post-operative and other post-natal patients in the various cubicles. There are five cubicles of 6 beds each and two single bed isolation rooms. Ward procedures are done in a separate procedure room. Each ward is run by senior house officers who do daily ward rounds, with the assistance of the nursing staff. A major ward round is conducted once a week by the senior registrars and consultants. The major ward rounds are also teaching rounds.

The postnatal clinic

This clinic is held every Friday morning and is mainly for patients who had operative deliveries or other complications. Mothers who had normal deliveries attend the family planning and child immunisation clinic at their nearest health centres. In this clinic, vital signs are recorded, urinalysis done and patient weighed. Systemic examination is done with emphasis on breasts, abdomen and pelvic examination. Contraception is advised and patients referred to the Family Planning clinic for further counselling. Patients with medical or other conditions are referred to relevant clinics for follow up.

Perinatal mortality meetings

These are held monthly on every second Friday morning. Mortality and morbidity data and rates are presented. Discussions are aimed at gauging the quality of services offered and the easing of any problems encountered that may contribute to the observed mortality and morbidity.

Obstetric procedures

Operative vaginal delivery

The Malmstrom vacuum extractor is used in our unit when operative vaginal delivery is indicated. Obstetric forceps are not used. The main indications for vacuum delivery are vertex presentation with:

a) Cardiac disease in second stage of labour.
b) Prolonged second stage of labour with poor maternal effort.

At full cervical dilatation, the patient is placed in lithotomy position (unless she has cardiac disease) and vulvovaginal toilet done with antiseptic solution. Draping and aseptic catheterisation is done then vaginal examination repeated to confirm cervical dilatation, position, and station of the vertex. Cephalopelvic disproportion is also ruled out. Under local anaesthesia, mediolateral episiotomy is performed. The largest vacuum cap that can fit into the vagina and onto the fetal scalp is then applied. A vacuum is built up increasing by 0.2 kg/cm^2 at one minute intervals to 0.8kg/cm^2 with the cup held onto the scalp. A finger is used to ascertain that no maternal soft tissues have been held. With the subsequent uterine contraction, traction is applied using the right hand while the left one continues to hold the cap firmly onto the scalp. Traction is applied at right angles to the vacuum cap and follows the curve of the birth canal. Once crowning occurs, the vacuum is released and delivery completed as described for spontaneous delivery.

Caesarean section

Preoperative preparation: Caesarean sections are either elective or emergency. Elective Caesarean sections are done at term. The haemoglobin level must be at least 10 g/dl and the renal functions should be normal. There must be at least two units of compatible blood available for necessary transfusion. The patient is normally fasted for at least six hours prior to the operation. Minimal preparations are possible for emergency Caesarean operations; blood is always taken for cross matching though transfusion may not always be necessary.

An informed consent for the operation(s) is obtained from the patient, or her spouse or other accompanying relatives when the patient is a minor or is moribund. The abdomen, vulva and perineum are shaved. Premedication is administered using intramuscular atropine sulphate 0.6 mg about half hour before operation. In a serious emergency, the dose is given in theatre intravenously by the anaesthetist.

The patient is placed on the operating table in supine position, her legs are drawn up, and aseptic catheterisation is done, the catheter being left in situ to provide continuous bladder drainage. The abdomen is cleaned using chlorhexidine, or hibitane then povidone-iodine is applied followed by sterile draping.

Anaesthesia: One hundred percent (100%) pre-oxygenation is done for five minutes. General anaesthesia is induced with intravenous 200–500 mg thiopental depending on patients' body weight. Anaesthesia is maintained with nitrous oxide and halothane via a cuffed endotracheal tube. Muscle relaxation is obtained using succinylcholine 50–100 mg intravenously before intubation, then a long acting muscle relaxant, usually pancuronium, is given.

Sometimes spinal block anaesthesia is used instead of general anaesthesia. This has the advantage of providing muscle relaxation and analgesia without inducing the patient to sleep. However, the anaesthetist is always ready for general anaesthesia if the spinal anaesthesia should fail.

Lower Uterine Segment Caesarean Section

The abdominal incision is either the sub-umbilical midline, the Pfannenstiel incision or the Joel-Cohen's incision. The first knife cuts the skin and with the second knife, the incision is extended deeper through subcutaneous tissues to the rectus sheath. This

is then opened using curved scissors after separating it from the underlying rectus muscles. These muscles are deflected laterally by blunt dissection to expose the parietal peritoneum. Using two long straight artery forceps, one on either side placed at the upper end, the peritoneum is opened, ensuring that there is no adherent viscera beneath. This incision is extended inferiorly proximal to the urinary bladder.

Moist abdominal packs may be placed on either side of the uterus in the paracolic gutters to keep the gut away from the operating field and to minimise soiling with blood and liquor.

A bladder retractor (Doyen's retractor) is used to fully expose the lower uterine segment and keep the bladder away. Using dissecting forceps, the loose vescico-uterine peritoneum is held and incised transversely in an elliptical fashion. A mounted swab is used to push this peritoneum off the lower segment thus taking the bladder down with it. Using the second knife, a shallow elliptical incision is made on the myometrium; in the midline, this is deepened to reach the fetal membranes. Guided by the index and middle fingers, the incision is extended laterally on either side using curved dissecting scissors.

The membranes are then ruptured and the right hand inserted into the uterus below the presenting part which is now gently lifted out of the uterus and abdomen. With the assistant applying fundal pressure, the baby is delivered. The cord is double clamped then cut and the baby handed over to the midwife or paediatrician for resuscitation, weighing and Apgar scoring. The anaesthetist administers intravenous syntocinon 20 IU. The placenta is delivered by controlled cord traction and the membranes extracted by gently pulling using artery forceps. The uterine cavity is cleaned using a mounted swab to remove pieces of membranes, placental tissue and clots.

Haemorrhage along the incision is controlled by applying Green-Armitage uterine clamps. The incision is then repaired in layers using continuous chromic catgut No. 2 suture mounted on a round body needle; usually one layer or using the second layer to bury the first if hemostasis is not achieved using the first layer. The vesico-uterine peritoneum may be repaired using No. 2/0 chromic catgut continuous suture. The surgeon then checks the incision site to ensure adequate hemostasis. The abdominal packs are removed and any clots cleared out. The scrub-nurse and her runner-nurse then count the swabs and instruments to ensure a correct count and informs the surgeon.

The parietal peritoneum may be closed using chromic catgut No. 2/0. The rectus sheath is closed using vicryl No. 1 mounted on a cutting needle in a continuous fashion. The subcutaneous adipose tissue layer is then closed with plain catgut No. 2/0 to obliterate potential spaces for haematoma collection. The skin around the incision is then cleaned with antiseptic solution or normal saline then repaired using interrupted mattress sutures of non-absorbable material usually nylon or using subcuteneous vicryl No. 2/0 or 3/0 on a cutting needle. The incision is then cleaned and dressed.

The drapes are removed and the patients legs drawn up; the catheter is removed (unless otherwise indicated) and the colour of urine ascertained. Gentle pressure is applied on the fundal region to expel any clots from the uterus into the vagina. A vulvo-vaginal toilet is done using mounted swabs soaked in antiseptic solutions.

General anaesthesia is reversed using intravenous neostigmine 2.5 mg and atropine sulphate 1.2 mg; the patient is then transferred to the recovery ward for continuous monitoring and thereafter to labour ward for half hourly observations until she is fully awake when she is taken to the post-natal ward.

Digital pelvic examination

The patient is informed of the procedure and the reasons for performing it. She is then asked to empty the bladder or catheterisation is done. The operator puts on sterile gloves; with the patient in semi-lithotomy position, vulval toilet is done. The external genitalia is inspected. The labia are parted using the left hand thumb and index finger, and the index and middle fingers of the right hand are inserted into the vaginal cavity.

Cervical dilation, length, consistency, position as well as uterine position is determined. The adnexa is palpated for tenderness and masses. The fornices are felt for masses. Pelvic assessment is then done; the diagonal conjugate is estimated by attempting to reach the sacral promontory using the middle finger. The prominence of ischial spines and the width of the sacrospinous ligament is assessed. The sacral curve is palpated, mobility of coccyx tested and then the width of the subpubic angle is determined using the two examining fingers. The intertuberosity distance is assessed with four knuckles (Fist). On completion of internal examination, the gloved fingers are inspected for blood or abnormal vaginal discharge.

Speculum examination

The bivalve (Cusco's) speculum and the Sim's speculum are sometimes used. The Cusco's speculum is indicated in cases of suspected preterm premature rupture of membranes and in antepartum haemorrhage (APH). It is also done to assess vaginal discharge and for removal of McDonald stitch. The procedure is explained to the patient. She is then asked to empty the bladder and lie in semilithotomy position on an examination couch. Vulvo-perineal toilet is done. The speculum is gently introduced with the right hand while parting the labia minora with the index finger and thumb of the left hand. The blades of the speculum are (in closed position) are introduced in transverse position. Once inside, the blades are opened and vaginal walls inspected. The cervix is observed for dilatation, bleeding, drainage of liquor or any other abnormality. When the procedure is complete, the speculum is withdrawn in the same way it was introduced.

The Sim's speculum is used for cevical and vaginal examination, for example when the is suspected cervical tears, vaginal tears or for genital fistulas. Two speculums are usually used. A complementary Auvard's (self-retainining) speculum may be used on the posteriour wall of the vagina. The insertion procedure is as above.

Amniocentesis

The procedure is carried out for bilirubin spectrophotometry in a Rhesus iso-immunised patient (when the indirect Coomb's test is positive). It may also be done for proposed elective delivery at 38 weeks gestation, for determination of fetal lung maturity. Amniocentesis is done in the clinic or in the ward. The procedure is explained to the patient and after having emptied her bladder, she lies in the supine position. The fetal heart is recorded. The lower abdomen is cleaned with hibitane then swabbed with spirit. The surgeon displaces the fetal presenting part cranially; using a gauge 20 hypodermic needle attached to a 10 ml syringe, the needle is gently advanced into the amniotic cavity and about 5–10 ml of fluid drawn. One may also use ultrasound guided amniocentesis.

If the test is for surfactant, 4 ml of amniotic fluid is withdrawn and for bilirubin spectrophotometry, 6 ml is withdrawn and put in a dark bottle to avoid sunlight breakdown of the bilirubin.

The colour of the amniotic fluid is noted as well as presence of lanugo hair and fetal squames. The fetal heart rate is observed again using the fetoscope. After the procedure,

the patient is advised to rest in the left lateral position and is monitored for 2 hours. During this time, the fetal heart rate is recorded 1/4 hourly and the presence or absence of vaginal bleeding or drainage of liquor is noted.

Surfactant (Bubble Shake) test

Two clean and dry test tubes are needed. One (1) ml of amniotic fluid and 1 ml of 95% ethanol (1:1 dilution) are put into the first test tube. Half (0.5) ml of amniotic fluid, 1 ml of 95% ethanol and 0.5 ml of normal saline is put into the second one (1:2 dilution). All tubes are then vigorously shaken for 15 minutes and placed in a rack for a further 15 minutes. The presence of a persistent ring of bubbles at the air fluid interface is considered a positive test.

Gynaecological services

Acute gynaecological services

These services are based in ward 1D; this ward has 40 beds that usually accommodate twice or thrice the number of patients (i.e. up to 120). Patients are mainly admitted though the gynaecologic casualty by the registrar. Those seen but do not meet admission criteria are treated and sent home while some are referred to the gynaecology clinic for follow up.

Once admitted, patients are clerked by the intern, then reviewed by the registrar. Majority of patients admitted are those with incomplete septic abortions; others are due to ectopic pregnancy, acute pelvic infections, tubo-ovarian masses, Bartholin's abscess and carcinoma of the cervix. Some are admitted through the Family Planning clinic for removal of translocated intrauterine devices. An emergency theatre is allocated on a 24 hour basis for emergency gynaecological operations.

Cases of carcinoma of the cervix are either new or previously diagnosed, admitted for resuscitation, diagnosis and management. New patients undergo examination under anaesthesia (EUA) every Friday. After confirming the diagnosis, those with stage I disease are transferred to ward 1B for Wertheim's hysterectomy while the majority with more advanced disease are referred to the Radiotherapy Department for pelvic irradiation. Those whose disease is beyond treatment with surgery or radiotherapy are referred to the Nairobi Hospice for terminal care.

Manual vacuum aspiration

There is a busy procedure room in the ward where uterine evacuation is done using Karman's cannular and syringe. Here, patients with incomplete abortions have uterine evacuation done on demand on outpatient basis; majority are discharged home immediately while those with sepsis or anaemia are retained for further management.

The cold gynaecology ward

This is ward 1B; it has 32 beds shared out among the three firms. Patients admitted here are mainly those followed and worked up for surgery in the gynaecology clinic; a few are from ward 1D. The bulk of admissions are either those requiring hysterectomy for uterine fibroids or tubal surgery for infertility. Patients with gynaecological malignancies undergoing chemotherapy are also found here. Each firm has a full day theatre once a week when elective operations are performed.

Total abdominal hysterectomy

This is one of the most common cold gynaecological operation done. The most common incision used is the subumbilical midline incision although other incisions such as Pfannenstiel incision is used.

The abdomen is opened as described Caesarean section. The abdominal organs (the liver, spleen, stomach, omentum and intestines) and pelvic cavity (the uterus, Fallopian tubes and ovaries) are inspected for any gross pathology.

Warm, moist abdominal packs are introduced into the abdominal cavity to keep the intestines and omentum off the operating site. The uterus is held with a myomectomy screw or a strong stitch (usually No. 2 chromic catgut) and if the uterine mobility allows, it is delivered through the abdominal incision. A self retaining retractor is fixed to the anterior abdominal wall to provide better exposure of the operation site.

The round ligament of one side is held with two Kocher's clamps near the uterine cornu, divided between the clamp, and distal portion transfixed with vicryl 1. From this incision site the anterior leaf of the broad ligament is opened and cut in an elliptical fashion towards the utero-vescical peritoneum reflection.

Two fingers (the index and middle) of one hand are used to push the posterior leaf of the broad ligament forward and with the other hand an incision is made between the tips

of the fingers with a pair of scissors to develop a window in the broad ligament. If the tube and ovary are to be preserved, curved Kocher's clamps are placed across the tube and ovarian ligament as close to the uterus as possible. A scalpel divides these structures between the clamps and the distal pedical is secured with a transfixing suture. If the ovary and tube are to be removed, the infundibulopelvic ligament is clamped, divided and the distal portion transfixed with vicryl No 1. The same procedure is repeated on the contralateral side.

Using a swab on a holder or swab around the finger or a mounted swab on stick, blunt dissection is done gently to separate the bladder from the uterus, cervix uteri and the upper part of the vagina. The ureters are reflected inferiorly by wide mobilization and displacement of the bladder base from the cervix, aided by traction on the uterine corpus.

The uterine vessels are skeletonized and exposed by trimming off the loose areolar tissue over the lateral aspect of the uterus and are then divided between two strong Kocher clamps and transfixed distally with vicryl No. 1. The procedure is repeated on the contralateral side.

The cardinal ligaments are then divided between clamps and transfixed. The peritoneal flap over the cervix posteriorly is dissected away to isolate the uterosacral ligaments, which are clamped, cut and transfixed adjacent to the cervix.

The lowest part of the cervix uteri is palpated and the upper end of the vagina just beneath the cervix is held with two Littlewood clamps. A stab incision is made with a scalpel between the two clamps opening into the vaginal canal. The incision is then extended circumferentially with a pair of curved scissors and the entire cervix is removed with the uterus. The specimen is taken for histopathology.

The vaginal vault is held with strong artery forceps and starting from the corners, closure is done with vicryl No. 1 ensuring hemostasis is achieved. The lateral stitches are left long enough to be incorporated in the peritonization stitch.

Using No. 1 chromic catgut, reperitonization over the vault, to cover the raw areas, is done by stitching together the reflected anterior and posterior visceral peritoneum, and incorporating the round, infundibulopelvic and uterosacral ligaments.

The abdominal packs are removed, instruments and swabs counted and the abdominal closed in layers as described previously. Post operative care is instituted as described above.

The gynaecology clinics

These are held on Tuesday, Wednesdays and Thursday afternoons, by firms I, III and II respectively. All the doctors in the particular firm are usually involved. Patients are usually referred from the casualty, other clinics in the hospital or from other peripheral health facilities.

All new patients are seen by senior registrars and consultants; a history taking and physical examination is done and investigations requested as appropriate to individual cases. Majority of patients present with infertility or uterine fibroids. Routine cervical smears are normally done. Other patients on follow-up include those who have undergone surgery or chemotherapy.

Colposcopy clinic

This clinic is run every Friday morning by a consultant and registrar. All patients with abnormal cervical smear are evaluated. Diagnostic procedures such as punch directed biopsies are done after application of either acetic acid or iodine to the cervix, vagina or vulva. Theraputic procedures such as large loop electrosugical excision of the trasformation zone (LLETZ) are carried out under local anaesthesia.

VVF clinic

This clinic is held on Tuesday afternoon where all cases of VVF are reviewed and staged for possible interventional surgery or follow-up after surgery.

Family planning services

These are offered in the Family welfare Centre, located in clinic 18. It caters for many clients from Nairobi and its environs. A wide range of contraceptives are available here. The clinic is run by specially trained nursing staff; a senior house officer is posted there each week to manage any clinical conditions that the clients may have. Cervical smears are also routinely done on all clients. Clients seeking surgical sterilisation are referred to other institutions as these are not being done on a mini-lap basis.

Laparoscopy and V.S.C. theatre

Previously located in the Family welfare Clinic No. 66, it is now located in the cold gynaecologic ward 1B. Patients for diagnostic laparoscopy are referred from the gynaecology clinics mostly due to tubal factor infertility. Clients for tubal ligation may be from within the hospital or outside centres.

All clients are reviewed by the consultants and senior registrars and those found suitable for the procedures are given an appointment. They are admitted, counselled and advised on preoperative preparations. Operations are done under general anaesthesia in main theatre on Thursdays. Those who had laparoscopy are sent back to the gynaecology clinic for follow-up to await open laparotomy tubal surgery.

The adolescent counselling clinic

This is an extension of the main Family Planning clinic. All patients in the adolescent age group presenting to the maternity or acute gynaecological ward as a result of pregnancy are followed up here. They are counselled and provided with appropriate contraceptive methods.

Common gynaecological procedures

Pap smear

This is usually done in the gynaecology and family welfare clinics. The procedure is explained to the patient who is then asked to lie in the semi-lithotomy position after emptying the bladder.

The surgeon puts on sterile gloves and without vulval swabbing inserts a Cusco's speculum into the vagina. The vaginal walls and cervix are inspected. A wooden Aryl's spatula or a cytology brush is introduced into the endocervix and rotated through 360°. The mucus and scrapings obtained are spread onto a clean dry glass slide. This is covered with a fixative and sent for cytological examinations. The speculum is then removed gently.

Supportive services

Radiotherapy services

The Radiotherapy Department serves the entire country and also neighbouring countries. Patients sent here are mainly those with inoperable carcinoma of the cervix and vulva. For cancer of the cervix external radiation is done using cobalt-60, administered daily except weekends and public holidays. Once a full course of this has been given, patients used to be given intracavitary radiation using caesium-137 twice a week. However, intracavitary radiotherapy is not currently being administered as the machines have broken down. They are followed up in the Radiotherapy clinic.

Laboratory services

The obstetric and Gynaecology unit benefits from all the laboratory services offered by the hospital. In addition, the unit runs its own laboratory which deals with semen analysis, radioimmunoassay of hormones, surfactant (or bubble-shake) test, urinary and blood ßhCG test, post coital test, urine and blood sugar determination, et cetera.

The Nairobi Hospice

Workers here offer counselling services in addition to management of terminal carcinomas. They also offer narcotic analgesia and encourage home-based care for such patients instead of hospital care. Patients are referred there from the wards or clinics whenever there is an indication for terminal care. For patients who are hospitalised and moribund, the hospice workers come to the ward and administer the treatment from there.

Case 1: Deep Venous Thrombosis in pregnancy

Name	**A.W.**	**Date of admission**	15 Feb. 2005
Age	26 years	**Date of discharge**	16 April 2005
I.P. No.:	1003270		
Parity	0 + 0 Gravida 1	**L.M.P.**	1 Jul. 2004
		Expected date of delivery	8 Apr. 2005
		Gestation by Dates	33 weeks

Presenting complaints

She had presented with a 3 week history of left lower limb swelling and associated difficulty in walking.

History Presenting Illness

She started feeling pain and slight swelling on left calf for 3 weeks prior to admission, which then progressed to the left inner thigh, the pain was worse on walking. There was no history of trauma nor any insect or animal bite. There was no history of chills or swelling of the right leg. She had never had such a problem before. She had not been on any medications.

There was no cough or chest pain. There was a history of non-dependant oedema of the lower limbs and fingers. There was no history suggestive of heart disease.

There was normal fetal activity.

Antenatal care

She had not started.

Obstetric and gynaecology history

She had menarche at 16 years; her cycle was regular coming every 28 days and lasting 4-5 days. She had never used any contraceptive.

Past medical history was non significant.

Family and social history

She was a single lady, who worked in a bureau, a teetotaller and living with her parents. Neither the patient nor her parents smoked cigarettes. She was a third born in a family of 3 siblings. There was history of chronic illness in her family; her grandmother was diabetic. There was a history of twining in the in the maternal side.

Physical examination

She was a young lady in fair general condition, not pale, not jaundiced, no oedema, no lymphadenopathy.

The vital signs were all within normal range: Blood pressure 120/70, pulse of 82/min of normal volume and regular, Respiratory rate of 20/min, and was afebrile with a temperature of 36.7°C.

Respiratory system, cardiovascular, and central nervous system were normal

Abdominal examination

The fundal height was 34 weeks, longitudinal lie cephalic presentation. The fetal heart was heard and regular at 142 beats per minute. There were no contractions palpated.

Vaginal examination: was not indicated therefore was not done.

Examination of the left thigh

The left thigh and calf swollen, warm, tender with non-pitting oedema. Comparatively the right thigh and calf were normal.

Circumferential measurements	Left limb	Right limb
Thigh (20 cm above tibial tuberosity)	58 cm	55 cm
Calf (15 cm below tibial tuberosity)	38 cm	33 cm

Impression

An impression of Deep venous thrombosis in pregnancy at 33 weeks gestation

Plan of management

She was admitted into antenatal ward and various investigations planned including: a Coagulation screen; Full Blood Count; Doppler scan of left lower limb. Meanwhile she was empirically started on intravenous heparin 1000 units/hr in infusion. A consultation was send for an urgent haematologist review who recommended that the heparin infusion should run at a rate of 1000 units per hour in normal saline and paracetamol 500 mg thrice daily. She was also started on aspirin 300 mg q8h and anti acids.

Results

1. Coagulation screen 16/2/2005

   PTI: Test 14 seconds

   Control 13 seconds therefore calculated index – 93%

   INR 1.08

   KCCT: Test 36 seconds; Control 34 seconds (ratio 1.1)
2. Blood group was O Rhesus positive, VDRL negative, Rapid test for HIV antibodies was Negative
3. Full blood count done showed; Hb 10.5g/dl, WBC 9.6 x 10^9/l, and differential: N=78%, L=20%, M=1% and E=1%. The MCV 68.3fl and Platelets 212x10^9/l.
4. Doppler scan (not done immediately) done on 11/3/05 reported: The deep veins were dilated and incompressible. Thrombosis was noted in the common femoral vein, with sluggish flow with poor response to augmentation. There were collaterals demonstrated. Conclusion: Deep Venous Thrombosis of the left popliteal and femoral veins.

The patient continued heparin infusion therapy. Her condition was improving as indicated by the reduction in daily serial limb measurements of the affected limb and a reduction in pain. After 2 days of heparin infusion a repeat KCCT on 26/2/2005 was 45 seconds (test) and 35 seconds (control) and a ratio of 1.3. However in view of the improving clinical condition despite the ratio the dose was maintained.

On 4/4/2005 (at 39 weeks +) at the patient went into spontaneous labour at noon while in the antenatal ward. She was transferred to labour ward for active management of labour. Heparin was withheld. Protamine sulphate was kept ready for her in case any noticeable excessive bleeding in the intrapartum or immediate postpartum period. The Haematologist was alerted to be on stand-by incase of any complications. Two units of fresh whole blood were also grouped cross-matched and kept ready. Labour was monitored by partogragh. She progressed uneventfully in labour and delivered SVD to live female infant who weighed 3kg, APGAR score 9/1, and 10/5 at 2.30 am on 5/4/2005. Estimated blood loss was 300 mls. The vital signs were normal; BP 120/80 mmHg, pulse 80/min, RR 22 /min. On 5/4/2005 at 10.00 am, patient was in stable condition and had started breast feeding. The baby was also okay she was restarted on heparin 5,000 units q8h.

On 7/4/2005 KCCT test was 42 sec and 32 sec for the control. Her condition was stable baby was okay and breast feeding well. She was started on Warfarin 5 mg OD on 9/4/2005 to overlap with heparin 5,000 units q8h, and on 12/4/2005 heparin was stopped, patient continued on warfarin alone. Her condition remained stable.

Post delivery tests

Full hemogram on 8/4/2005, showed; WBC 9.7 x 10^9/L; Hb 9.7 g/dl; MCV 70.1 fl Platelet 173 x 10^9/L, the peripheral film showed slight hypochromasia.

PTI on 12/4/2005: Test 31 seconds; Control 13 seconds.

On the 11th post delivery day, patient discharged home on warfarin and hematinics. She was instructed to come to postnatal clinic in 2 weeks and also to attend haematology clinic. She honored the follow up in the postnatal clinic and review showed no complications. She however did not honor the hematology clinic appointment.

Discussion

The patinet A.W. was a primigravida diagnosed to have deep venous thrombosis at 33 weeks gestation. She went into spontaneous labor and delivered vaginally with a good outcome. Post delivery she was followed with no complications.

Thrombosis is the process by which liquid blood flowing through the vascular system turns into a solid mass of platelets, cells and fibrin (1). Virchow's triad of underlying factors in venous thrombosis: hypercoagulability, venous stasis, and vascular damage – all occur in pregnancy. During pregnancy there are increases in procoagulant factors, such as von Willebrand factor, factor VIII, factor V, and fibrinogen, that occur together with an acquired resistance to the endogenous anticogulant, activated protein C, and a reduction in protein S, the co-factor for protein C. These changes are accompanied by impaired fibrinolysis through increases in plasminogen activator inhibitors 1 and 2, the latter being produced by the placenta. These changes represent the physiological preparation for the haemostatic challenge of delivery. Venous stasis occurs in pregnancy by the end of the first trimester and reaches a nadir at 36 weeks. Endothelial damage to pelvic vessels can occur during vaginal or abdominal delivery. Thus, the scene is set for the development of thrombosis in pregnancy (2). In addition to changes in the coagulation system, physiological alterations during pregnancy cause venous stasis that predisposes to venous thrombosis. There is increased lower extremity venous diameter and decreased flow, likely as a result of hormonal influences on vascular tone and the compression effects on the veins by the enlarging uterus. The measured flow velocity, particularly in the left leg, is decreased when the pregnant woman is placed in the supine position. This could explain the striking predominance of left leg DVT observed in pregnancy.(3) Vascular damage may occur due to hypertensive disease, surgery of the pelvis and pelvic or vascular infections.

Pregnancy is associated with an average 5–10 fold increase in the risk of venous thromboembolism compared with non-pregnant women of similar age. The risk is only moderately increased during the first and second trimesters, but increases sharply during the third trimester, and peaks during and shortly after delivery (4,5). A.W. was diagnosed with DVT in pregnancy. In a systematic review, the incidence of first deep vein thrombosis in the general population was 0·5 per 1000 person-years. The disorder is rare in children younger than 15 years, but its frequency increases with age, with incidence per 1000 person-years of 1·8 at age 65–69 years and 3·1 at age 85–89 years.

In a retrospective hospital discharge data, the prevalence of deep vein thrombosis was comparable in black (0·69%) and white adults (0·84%). In a British study, 25% of white and 22% of black people with suspected thrombosis were confirmed to have the disorder. The prevalence of deep vein thrombosis in Asian population is low. Risk for first deep vein thrombosis seems to be slightly higher in men than in women. In a population-based cohort study, the age-adjusted incidence of first venous thromboembolism was 1·3 per 1000 person-years in men and 1·1 per 1000 person-years in women. It is noteworthy that the risk for recurrence of this disorder is higher in men than in women (6,7). The incidence of antenatal DVT is about 0.615 per 1000 pregnancies in women aged younger than 35 years and 1.216 per 1000 in women older than 35 years. The rate of postpartum DVT is about 0.304 per 1000 in women younger than 35 years and 0.72 per 1000 in women older than 35 years (1,5). Antenatal DVT is more common than postpartum DVT (1,2). At Kenyatta National Hospital the incidence of DVT was reported as 0.16% of all pregnancy admission (4). A.W. presented with DVT in the antenatal period. She did not have history of DVT outside pregnancy.

The risk factors include: prior history of DVT, leg oedema or venous stasis changes, venous varicosities, degree of preoperative ambulation, pelvic or bone surgery, malignancy, prior pelvic radiation therapy, age 35 years, excessive body weight, prolonged duration of anaesthesia (8). Other predisposing factors include heavy cigarette smoking, obesity, anaemia, haemorrhage, heart disease, hypertensive disorders, prolonged labour, renal disease, operative delivery, postpartum endometritis and HIV infection (9). Blood group "A" has been related to increased risk of DVT (10).

Attention is currently being directed to a number of isolated deficiencies of proteins involved either in coagulation inhibitors in the fibrinolytic system. The main congenital thrombophilia are deficiencies of antithrombin protein C and protein S and the presence of factors V Leiden, the prothrombin gene variant and homozygosity for the thermolabile variant of methylenetetrahydrofolate reductase – MT HFR (1,2,8,9). The only apparent risk factors in the patient presented was pregnancy. Investigations were not done to rule out other aetiological factors such as screening for protein deficiencies.

A strong association between lupus anticoagulant and DVT has been established (11). Mwanda (12) recommended screening for lupus anticoagulation in those patients with thromboembolic events, positive VDRL and recurrent fetal loss. Our patient had a negative VDRL result, had no history of pregnancy loss and hence there was no

necessity for screening lupus anticoagulation. Endothelial damage to pelvic vessels can occur during vaginal or abdominal delivery. Preeclampsia is also associated with vascular injury (6,7). A.W. developed antenatal DVT and her blood pressure remained normal throughout the pregnancy.

Almost 90% of DVT affect the left side among pregnant women compared with 55% among women who are not pregnant (3). Waweru (4) found that at Kenyatta National Hospital, DVT is three times commoner in the left lower limb than the right side. This difference may reflect compression of the left iliac vein by the right iliac and the ovarian arteries which cross the vein on the left side only (2). DVT in pregnancy can present or be associated with, lower abdominal pain due to periovarian collateral circulation or thrombosis. When coupled with mild pyrexia and leucocytosis of thromboembolism, this pain can be mistaken for other intraabdominal disorders such as urinary tract infection or appendicitis (2). A.W. had DVT on the left lower limb. In pregnancy most cases of DVT are iliofemoral rather than calf vein thrombosis (72% vs 9%) (2). A.W. had DVT of the left femoral vein but did not present with fever.

Most DVT patients are completely asymptomatic. Symptoms may be subtle or classic depending upon the site and extent of the thrombosis and the status of the collateral venous circulation. Classic features include swelling of the affected site, pain, tenderness, local cyanosis and fever (6). Clinical findings were used to make a working diagnosis in our patient.

Clinical evidence of DVT of the legs precedes pulmonary embolization in only about half the cases. Importantly nearly 40% of asymptomatic patients with DVT were found to have a concomitant pulmonary embolism (13). Chest discomfort, shortness of breath air hunger, tachypnoea or obvious apprehension are signs and symptoms that should alert the physician to a strong likelihood of pulmonary embolism. The most reliable symptom is breathlessness (13).

The presently available techniques for the objective diagnosis of DVT include contrast venography, non-invasive methods, and biochemical assays. Venous ultrasonographic imaging is most widely used. Proximal veins are compressed under gentle pressure with the ultrasound transducer (compression ultrasonography) the inability to compress a vein indicates presence of DVT (11,13). Impedance plethysmography, real-time B-mode

ultrasonography, magnetic resonance imaging and computed tomography are some of the other tests that can be used. Venography remains the standard for confirmation but it has the risk of inducing thrombosis itself (13).

Treatment options in venous thrombosis include anticoagulation, caval filters, fibrinolytic therapy, and surgical thrombectomy (6,14). The last three therapeutic approaches have been assessed less extensively and are not routinely used. Anticoagulation therapy is the treatment of choice for most patients with established venous thromboembolism.

Heparin is the safest drug to use during pregnancy because it does not cross the placenta. Heparin may be given by continuous intravenous (IV) infusion, intermittent IV or subcutaneously intermittently. The major side effect is bleeding; other complications include thrombocytopenia, osteoporosis and fat necrosis. The partial thromboplastin time should be 1.5–2 times during heparin therapy (7,13,14,). A.W. was treated with intravenous heparin infusion and later subcutaneous heparin during the antenatal period. Warfarin should be avoided throughout pregnancy. Pathological effects on the fetus such as central nervous systems and ophthalmologic abnormalities have been associated with exposure to warfarin in any trimester. Warfarin readily crosses the placenta and can result in fetal and neonatal haemorrhage and placental abruptio. The usual dose of warfarin is 10–15mg daily in divided doses until the therapeutic level of prothrombin time (1.5–2.5 times the control value) is achieved.

There is no significant transfer of warfarin across the breast hence rendering it safe to use during lactation. Warfarin should be continued for 6 weeks postpartum (6,7,14). A.W. was managed in puerperium initially with heparin and later with warfarin for 6 weeks. The antidote for heparin is protamine sulphate 1 mg for 100 units of the former. The reversal of heparin by protamine sulphate is fast. Warfarin antidote is Vitamin K in a dose of 5mg intravenously but the process of antidote is slow (6). In U.K. pulmonary thromboembolism is the leading cause of maternal death. DVT underlies this disorder (5).

Thromboembolism deterrent stockings may be useful in pregnancy. These stockings stop over distension of veins and hence prevent endothelial damage. In women with multiple thrombotic events during previous pregnancies, antenatal prophylaxis should start at least 4–6 weeks in advance of the gestation at which the previous events occurred. If previous thrombosis was not associated with pregnancy, then prophylaxis should start at 20 weeks of pregnancy or earlier if additional risk factors are present (6,13).

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Case 2: Retained placenta – manual removal

Name	: E.W.	**Date of admission**	: 10 Jan. 2004
Age	: 25 years	**Date of discharge**	: 11 Jan. 2004
I.P. No.:	0988670		
Parity	: 1 + 0 Gravida 2	**L.M.P.**	: 14 Apr. 2003
		Expected date of delivery	: 21 Jan. 2004
		Gestation by Dates	: 38+ weeks

Presenting complaints

This patient was admitted from outpatient with a history of drainage of liquor of 6 hours prior to admission and labour pains for 1 hour prior to admission.

Fetal activity was present and normal.

Obstetric and gynaecology history

She was a Para 1 + 0 gravida 2. Her last delivery in 2002 was via elective Caesarean section due to breech presentation The operation was done in St. Mary's Hospital.

She had her menarche at 14 years and her cycle interval was 21 days used to bleed for 2 days. The menses were regular and without dysmenorrhoea. She had never used any contraceptives.

Antenatal care

She attended Kenyatta National Hospital where she had made 3 visits prior to admission. The period was uneventful. Her pelvis was assessed clinically at 36 weeks gestation and was found to be adequate. She was planned for trial of scar.

Profiles done: Blood group "O" Rhesus positive, both VDRL and HIV antibodies negative, Hb 12.5 g/dl. Urinalysis done on each visit was normal.

Past medical history was not contributory.

Family and social history

She was a married business woman who lived in Riruta with her husband, she did not take alcohol, nor smoke cigarettes. There was no family history of chronic illness.

Systemic inquiry was not revealing.

Physical examination

She was a young woman in good condition. She was pale, not jaundiced, and with no lymphadenopathy. There was bilateral mild pedal oedema.

The vital signs were all within normal range: Blood pressure 120/75 mmHg, pulse of 90/min of normal volume and regular. Respiratory rate of 20/min, and was afebrile with a temperature of 36.4°C.

Abdominal examination

The abdomen was evenly distended. The fundal height was term, lie longitudinal, cephalic presenting. Descent was 3/5. There was a normal fetal heart at 146 beats/min. There were 2 moderate contractions lasting 20-40 sec per 10 minutes.

Vaginal examination

Normal external genitalia. Draining clear liquor. Digital examination revealed a cervix 5 cm dilated fully effaced and no cord was palpated. There was no caput or moulding.

Pelvic examination was adequate pelvis.

Central Nervous system, Cardiovascular system, Respiratory system, Musculocutaneous system were all essentially normal.

Diagnosis

A diagnosis of para 1+ 0 G 2 with a previous scar at term in active labour was made.

Management

A decision to try the scar was made, and this had been explained to the patient during her antenatal period. An intravenous line of 5% dextrose was established and blood drawn for grouping and cross-matching and a partogram started. Vital signs were monitored quarter hourly and vaginal examination repeated every four hours and the scar tenderness checked at each vaginal examination. The liquor was also monitored closely for any sign of bleeding.

Labour progressed well during the first stage and second stage and the outcome was a live female infant who scored 8/1 and 10/5 and weighted 3.2 kg. A syntocinon drip of 20 units was started after delivery of the fetus and spontaneous expulsion of the placenta awaited.

By 30 minutes the placenta had not been expelled and attempt to deliver it by controlled

cord traction was not successful. There was no bleeding noted. The patient was explained to about the condition that it was necessary to have the placenta evacuated manually in theatre under anaesthesia. An informed consent was obtained and the patient pre-medicated with an intramuscular injection of atropine sulphate 0.6 mg.

In theatre the patient was anaesthetised. She was placed in lithotomy position. The perineum was cleaned and a vulvo-vaginal toilet done. She was draped and catheterised. Clear urine was obtained. EUA done revealed a fundal height of 20 weeks. The vagina and cervix were intact and the os was 5 cm dilated. The right hand was lumbricated with KY-jelly and inserted into the uterus identifying the placental site on the fundoposterior region. The left hand was used to support the uterus while the ulna aspect of the right hand was used to shear off the placental attachment. The hand was withdrawn and placenta delivered by controlled cord traction. The placenta was inspected and looked fibrotic and complete weighing 650 gm. The uterine cavity was explored and found to be empty and intact on both the upper and lower segments. The cervix and vagina were also found to be intact. Uterine massage was done and the drip of syntocinon commenced. The uterus contracted well and hemostasis was achieved. Estimated blood loss was 500 ml from second stage to the time of placental delivery. General anaesthesia was reversed and the patient wheeled out of theatre for observation.

Postoperative management

Intravenous infusion of syntocinon 40 units in 500 mls of 5% dextrose was continued running at 40 drops per minute. Intravenous ampicillin 1g was given as a stat dose and continued with amoxicillin 500 mg orally three times daily for five days. The patients vital signs were observed half hourly until she was fully awake and thereafter 4 hourly. These remained normal. Lochia loss remained normal and the uterus well contracted. She was discharged after 48 hours to attend the nearest MCH clinic at home.

Discussion

The patient presented had a successful vaginal birth after a Caesarean section. However, she the had retained placenta postpartum. The median time of plancetal delivery is 6 minutes and 95% of spontaneous placental deliveries occur within 30 minutes (1). In uncomplicated case, if the placenta has not delivered by 30 minutes the placenta is considered retained. The precise reason for delay in detachment beyond this time is not always obvious, but quite often it seems to be inadequate uterine contraction and retraction. Very infrequently the placenta is usually adherent to its site because of scanty or absent decidua so that the physiological line of cleavage through the spongy layer is lacking. As a consequence, one or more cotyledons of the placenta are firmly bound to be defective decidua basalis or even the myometrium when the placenta is thus densely anchored the condition is called placenta accreta (2).

Failure of the placenta to deliver spontaneously is an important cause of postpartum haemorrhage. This accounts for 5-10% of postpartum haemorrhage (3). However it has been shown that there is no risk until 30 minutes have elapsed and suggestion that conservative management is appropriate during this interval (4). In the case described conservative management using controlled cord traction and oxytocin were done though unsuccessfully. There is increased incidence of retained placenta among patients who have preterm labour (6,7). There is also an increased incidence of placenta accreta in patients with prior Caesarean section scars (7). Physically the uterus should contract soon after the placenta separates from the uterine wall and is spontaneously expelled. Spontaneous placental separation is indicated when the umbilical cord lengthens, there is a gush of blood, uterus becomes more globulous and the fundus moves upwards (2,3). These had not become apparent in E.W.

Active management of the third stage of labour by delivering the placenta manually by controlled cord traction (8) is encouraged in order to prevent postpartum hemorrhage. The principal in our unit is to wait for spontaneous expulsion, manual removal being indicated only if this expectant management fails. Use of oxytocin drugs in 3rd stage is advocated and is always used after delivery of the baby unless there is a contraindication. Oxytocin drip is occasionally used to enhance uterine contractions and aid delivery of placenta as occasionally the cause of retained placenta is uterine atony or inadequate uterine contractions. In addition to use of oxytocics in active management of

third stage, upward pressure is applied to the body of the uterus at the suprapubic region by the left hand, the umbilical cord is kept slightly taut and an upward and downward movement is performed with the right hand. This is repeated until the placenta reaches the introitus after while it is lifted with the right hand, mild traction on the cord is emphasized so as to avoid uterine inversion. This is referred to as controlled cord traction. These were done to the patient described but unsuccessfully.

Patient who have retained placenta may often present in shock due to postpartum haemorrhage. Adequate resuscitation is therefore mandatory before attempting manual removal. This should include intravenous fluids and/or transfusion if the patient is bleeding and the administration of second dose of oxytocin. The patient presented did not have postpartum haemorrhage and only got a re-administration of oxytocin by infusion.

Manual removal is performed under anaesthesia or sedation, aseptic surgical techniques is employed. After grasping the abdominal wall with one hand, the other hand is introduced into the vagina and passed into the uterus along the umbilical cord. As soon as the placenta is reached, its margin is located and the ulna border of the hand insinuated between it and the uterine wall. Then with the back of the hand in contact with the uterus, the placenta is peeled off its uterine attachment by a slicing motion. After its complete separation the placenta should be grasped with the entire hand which is then gradually withdrawn. Membranes are removed at the same time by carefully teasing them from the decidua, using ring forceps to grasp them as necessary. The uterus should always be explored before removing the hand for remnants of placenta or membranes or tears within the uterine cavity or cervix. The genitalia should also be explored to rule out tears.

Rarely placenta accreta may be encountered and the cleavage line will not be found. If bleeding is slight and conservation of fertility is desired then conservative management is advised. Necrosis and piecemeal expulsion will occur in 2–3 weeks. In such a case, the risk of infection especially with anaerobic bacteria is high and therefore prophylactic antibiotics are a must. Haemorrhage of varying degrees is common and if uncontrollable hysterectomy may be the end result (2). In the case of placenta accreta vera piecemeal removal of the placenta and use of uterotonics may be successful (2,3,9). In E.W. the

cleavage line was easily found and the placenta removed in one complete piece. There was no haemorrhage after the procedure.

Risks of manual removal of placenta included postpartum haemorrhage due to incomplete removal, uterine inversion, uterine rapture, infection and rarely inversion (2,3). The patient presented did not develop any of the complications. She was put on amoxycillin for prophylaxis.

Cytotoxic drugs such as methotrexate have been used in persistent retained placenta with a low pulsatility index of the uterine arteries and increased serum hCG. The literature states that even in cases of an undetectable hCG level, methotrexate can successfully cure persistent retained placental tissue. It is suggested that methotrexate acts on the dividing trophoblast cells and reduces neo-vascularisation and growth factors as well. Methotrexate has been shown to decrease trophoblast activity and to reduce placental vascularity. After treatment with methotrexate, the placental size, is assessed weekly by ultrasonography until there are no detectable trophoblastic tissue and the pulsatility index of the uterine arteries increases to non-pregnant values (10).

The direct delivery of an oxytocic to the retro-placental myometrium through the umbilical vein has been tried and was first tried in the 1960s. This is based on the importance of the retro-placental contractile failure in retained placenta and therefore its implication for treatment. According to the publication of a Cochrane review it is being recommended as first line treatment for retained placentas by the World Health Organisation (8,11).

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Case 3: Vaginal delivery after previous Caesarean section – Live baby

Name	: C.W.	**Date of admission**	: 14 Dec. 2003
Age	: 22 years	**Date of discharge**	: 15 Dec. 2003
I.P. No.:	0929937		
Parity	: 1 + 0 Gravida 2	**L.M.P.**	: 14 Mar. 2003
		Expected date of delivery	: 21 Dec. 2003
		Gestation by Dates	: 38 weeks

Presenting complaints

The patient was admitted at 38 weeks gestation with a 1 day history of low abdominal pains.

History of presenting complaints

She started experiencing mild low abdominal pains that were radiating to the back on the morning of admission day. The pains were intermittent and were more or less the same in intensity. She had no pains on passing urine, no drainage of liquor and no per vaginal bleeding. Fetal activity was present.

Obstetric and gynaecologic history

She was a para 1+0. She had delivered in 1997 by Caesarean section to a live male infant weighing 2950 gms due to breech presentation who was alive and well. Her last menstrual period was on 14/3/2003. Her expected date of delivery was on 21/4/2003 which made her maturity by dates as 38 weeks plus. She attended antenatal care at Kenyatta National Hospital from 8/7/2003. She had a pelvic examination done on the first visit and this was found to be adequate. An erect lateral pelvimetry was requested for but was never done.

Antenatal profile

Blood group – "A" Rhesus positive	VDRL – Negative
Haemoglobin – 13.0 g/dl	HIV serology – negative

Clinical pelvimetry: done at 36 weeks in the clinic revealed an adequate pelvis and was eligible for trial of scar.

She attained her menarche at the age of 15 years. Her periods were regular occurring every 28 days and lasting for 3 days.

Contraceptives: She had used oral contraceptives prior to conceiving current pregnancy.

Past medical and surgical history was not significant.

Family and social history

She was a married housewife. Her husband was a driver. Both neither smoked cigarettes nor consumed alcohol. There was no history of chronic illness in the family. She resided in Nairobi with her husband and child.

General examination

She was in good general condition was not pale, not jaundiced, had no oedema and no lymphadenopathy. Her blood pressure was 120/80 mmHg, pulse rate was 88 beats per minute, the respiratory rate was 22 per minute and was afebrile at 36.6°C

Central nervous, cardiovascular and respiratory systems were essentially normal.

Abdominal examination

A sub-umbilical median scar was evident. The fundal height corresponded to a term gestation. The fetus was in cephalic presentation, longitudinal lie and fetal heart sound was heard and regular at 146 beats per minute. There was no tenderness. No contractions were palpated. The fetal weight was estimated at 3000gms.

Pelvic examination

The external genitalia was normal. The cervix was not effaced, os was parous and the pelvis clinically felt adequate.

Impression

A patient with one previous Caesarean section and clinically adequate pelvis and possible urinary tract infection.

Management

She was injected with intramuscular pethidine 100 mg and was for review after 4 hours. The pain subsided and she was taken to the antenatal wards. She started experiencing low abdominal pains the following day and examination revealed that the cervix was 50% effaced and the os was 2cm dilated. She was to be monitored for onset of labour. The following day at 2 pm she was noted to be contracting and was taken to the labour ward. At 3 pm she was examined and found to have 3 contractions every 10 minutes with each lasting 30 seconds. The cervix was then 6 cm dilated. Artificial

rupture of membranes was performed and clear liquor was obtained. There was no caput or moulding of the fetal head. There was no cord palpated. Sacral promontory was not tipped, subpubic angle felt obtuse and the intertuberous diameter was able to accommodate 5 knuckles.

She was for trial of scar and the decision was relayed to her and she accepted. A partogram was to be commenced. Blood for grouping and cross-matching was taken just in case a Caesarean section turned out to be the eventuality. Intravenous fluids were commenced. She was reviewed 3 hours later and found to be fully dilated.

Second and third stages

The patient delivered vaginally at 6 pm to a female infant weighing 3200 gms. The infants Apgar score was 9 at 1 minute and 10 at 5 minutes. The placenta was delivered by controlled cord traction. The estimated blood loss was 200mls. The cervix, vagina and perineum were inspected and found intact.

Her post-delivery observations were found to be normal and she was transferred to the postnatal ward. After 24 hours in the postnatal ward she was discharged home to come for review after 6 weeks in the Family welfare clinic. However, she was lost to follow-up.

Discussion

The patient presented here was a para 1+0, one previous scar due to breech presentation in primigravidae. She underwent a successful vaginal birth after Caesarean section (VBAC) in the second pregnancy with good outcome.

For many years, the scarred uterus was believed to be contraindicated to labor out of fear of uterine rupture. However, there have been remarkable changes in obstetric practice over the last decade where women with previous Caesarean delivery are allowed to deliver vaginally (1).The safety of vaginal birth after Caesarean (VBAC) has been documented (2,3,4,5). Experience shows that more than two thirds of patients with previous Caesarean section can deliver vaginally (2,3). A study undertaken at Kenyatta National Hospital revealed that 73.9% of those who underwent trial of scar delivered vaginally (6). Previous Caesarean section is the commonest indication for performing the most frequent operation in the USA (3,6). In a review of Caesarean section deliveries at Kenyatta National Hospital, Karanja (6) got a Caesarean section rate of 17.8% with repeat Caesarean section deliveries accounting for 51.2% of all Caesarean sections. If a substantial portion of previous Caesarean patients can deliver vaginally, the number of Caesarean and the attendant increase in medical risks and costs can be reduced (4,7,8).

One of the requirements for trial of scar is for that institution to have the capacity of professional and institutional resources to respond to acute intrapartum obstetric emergencies such as performing a Caesarean delivery within 30 minutes from the time the decision is made until the surgical procedure is begun (2,5,7,9,10). The institution where the above patient underwent trial of scar has the professional and resources of performing Caesarean section in case it was an eventuality.

The American College of Obstetricains and Gynecologists (1999) recommend the candidates for vaginal birth after Caesarean delivery as:

(i) those who have one or two prior lower transverse Caesarean deliveries (in our setup we recommend only one scar),
(ii) those with clinically adequate pelvis,
(iii) those with no other uterine scars or previous rupture,
(iv) where physicians are immediately available throughout the active labor capable of monitoring labor and performing an emercency Caesarean delivery, and

(v) there is an available theatre fully functional for emercency Caesarean delivery. (11)

The patient discussed had all these conditions met.

Provided there are no contraindications, a woman with 1 previous transverse low-segment Caesarean section should be offered a trial of labour (TOL) with appropriate discussion of maternal and perinatal risks and benefits. The process of informed consent with appropriate documentation should be an important part of the birth plan in a woman with a previous Caesarean section (7). Contraindications for VBAC are:

1. Previous classical or inverted T uterine scar,
2. Previous hysterotomy or myomectomy entering the uterine cavity
3. Previous uterine rupture
4. Presence of a contraindication to labour, such as placenta previa or malpresentation
5. The woman declines a trial of labour after Caesarean and requests Elective operative abdominal delivery.
6. Should have a radiological pelvimetry with a true conjugate of the pelvis measuring less than 10.5cm.
7. Should not have an adverse medical or present obstetrical condition like hypertension, diabetes mellitus, breech or bad obstetric history. Investigators reporting result of large numbers of women labouring with previous Caesarean section (PCS) included those with two PCS and were unable to show an increase in risk (5). C.W. had one previous Caesarean section delivery and in the previous pregnancy did not have uterine rupture.

C.W. had undergone a low transverse Caesarean section due to breech at term in a primigravidae.

X-ray pelvimetry is not essential for the management of a woman with a previous Caesarean delivery. The only women who need x-ray pelvimetry are secondgravidae whose first pregnancy ended in a Caesarean section for CPD (2,10). Ogutu, and Githiru found that radiological pelvimetry does not have much value in the management of mothers with one previous scars (11,12). Pelvimetry is maternally focused and does not assess fetal size and presentation. Furthermore it is a static examination which does not take account of changes in pelvic and fetal dimensions during labour and delivery (13). Available data show that even in patient whose indication for previous Caesarean

delivery was CPD, almost two thirds of those who underwent trial of scar were able to deliver vaginally (3,4,10,13).

Some studies have examined women attempting vaginal birth after Caesarean in multiple pregnancy. All support a trial of VBAC in multiple pregnancy as being safe and effective, with success rates of 69–84% and without increased maternal or fetal morbidity or mortality (2,7). In Kenyatta those patients with multiple gestation and previous scar are delivered by elective Caesarean section. C.W. was followed up antenatally and had been found to have a singleton pregnancy. Labour after a previous Caesarean section in women with suggested fetal macrosomia (more than 4000gms) should be considered and individualized based on the actual estimate of fetal weight and maternal pelvimetry (5). C.W. was assessed clinically and the fetal weight estimated as 3000gms. She went ahead to deliver a 3200gms infant.

A breech presentation with a uterine scar present should have an elective Caesarean section. Common sense suggests that the combination of risks in such a situation would result in a very high perinatal mortality (10). C.W. was assessed antenatally and found to have cephalic presentation. She delivered by spontaneous vertex. The success and safety of labour specifically after the patients particular previous Caesarean section should be discussed and documented in the prenatal record. It is also wise to inform the patient of the rare risk of uterine rupture and the rarer possibility of hysterectomy and poor fetal outcome (5). C.W. was counselled in the antenatal clinic and readily consented for trial of scar.

Because women with one low-transverse PCS and an otherwise uncomplicated pregnancy are at very low risk of intrapartum complications, they can be treated for the most part as any patient in labour with the expectation of a vaginal delivery. This might include (2,5,7): early labour at home, obtaining blood count, type and screen on admission, continuous electronic fetal heart rate monitoring or frequently intermittent fetal heart rate monitoring, optional intravenous access. C.W. had blood taken for grouping and cross-matching. Intravenous fluid were administered during labour. The fetal heart was monitored intermittently with a fetoscope. The use of oxytocin for either induction or augmentation of labour is not contraindicated in women with PCS undergoing trial of labour. Oxytocin should be administered judiciously to avoid

hyperstimulation (2,5). In 2001, Goetz, et al. performed studies in women who received oxytocin to augment their spontaneous labour in a planned TOL after Caesarean. No increase in the risk of uterine rupture, maternal morbidity, or perinatal morbidity or mortality was detected (14,16). The labour of the patient presented progressed well and oxytocin use was not necessary.

The issue that has most often prevented physicians from allowing women to undergo a vaginal delivery after a previous Caesarean section has been the fears of uterine rupture or dehiscence. Frank uterine rupture or dehiscence has been shown to occur in 1.2% of women with vaginal births after Caesarean (2). The most common presenting sign of uterine rupture is fetal distress, in particular an abnormal fetal heart rate pattern with variable deceleration that evolve into late deceleration or fetal bradycardia. The next most common finding is uterine pain that continues between contractions, located usually in the area of the previous incision. Haemorrhage is as common as uterine pain and may be abdominal, vaginal or urinary. Onset of gross haematuria suggests a uterine rupture with associated bladder damage. Other signs and symptoms of rupture not as frequently seen are loss of uterine contractions, recession of the presenting part and fetal death. A small percentage of patients will have no signs or symptoms (5). The patient being discussed did not develop any of the above ominous signs.

The necessity for routine examination of the uterine scar after a successful vaginal delivery is controversial. Scar dehiscence is usually asymptomatic and rarely complicated by excessive bleeding because the separation is in a fibrous relatively avascular area of the myometrium. If there is excessive vaginal bleeding or any sign of hypovolaemia, examination of the previous scar and the entire genital tract is mandatory. If the defect is contiguous with the peritoneal cavity and the woman has haemodynamic instability, laparotomy and repair should be undertaken (2,5). Routine digital exploration of the Caesarean scar postpartum is not necessary, except when signs or symptoms suggest uterine rupture (2,15). In Kenyatta National Hospital routine exploration is not done in women with vaginal birth after Caesarean. C.W. was explored though she did not have vaginal bleeding and the lower uterine segment found intact. She remained well and was discharged after 24 hours.

Avasia, et al. reviewed the risk of uterine rupture in women undergoing an induction TOL SOGC Clinical Practice 324 Guidelines after Caesarean. In 575 women with a

previous Caesarean section, labour was induced with prostaglandin E_2 gel (n=172), intracervical Foley catheter (n=129), or amniotomy and (or) oxytocin (n=274). Outcomes were compared with women undergoing a TOL with spontaneous labour. The risk of uterine rupture was not increased in women who underwent either amniotomy/oxytocin or Foley catheter induction but was significantly increased in those who underwent a prostaglandin E_2 induction (P=0.004) (16).

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Case 4: Cardiac Disease in Pregnancy

Name	: I.N.	**Date of admission**	: 8 Dec. 2004
Age	: 32 years	**Date of discharge**	: 11 April 2005
I.P. no.	: 0998904		
Parity	: 1 + 0 Gravida 2	**L.M.P.**	: 2 Jul. 2004
		Expected date of delivery	: 9 Apr. 2005
		Gestation by Dates	: 22 weeks

Presenting complaints

This is a patient followed up with cardiac disease New York Heart Association (NYHA) grade III and was referred from Kangundo District Hospital with congestive cardiac failure and anaemia.

Her presenting complaints were a chronic cough and generalized body weakness both for one month. The cough was productive of white sputum, non-blood stained. It was associated with minimal chest pains. There was orthopnea and paroxysmal nocturnal dyspnea. There was dyspnea on mild activity. There was lower limb swelling bilaterally. There was no chills or hotness of the body. There was a history of palpitations throughout her pregnancy. Micturation habits and bowel habits were normal.

Fetal activity was present and normal.

Antenatal care

This was attended in Kangundo Hospital were she had been diagnosed to have cardiac disease and admitted. She was referred when her condition was noted to be deteriorating. Profiles had been done but no results were available at the time of admission.

Obstetric and gynaecology history

She was a Para 1 + 0 gravida 2 with 1 living child.

No.	*Year*	*Gestation*	*Duration of labour*		*Delivery*	*Outcome*	*Comments*
1	2002	term	home delivery	SVD	? kg, M	alive and well	

She had her menarche at 14 years and her cycle interval was 21 days used to bleed for 2 days. The menses were regular and without dysmenorrhoea. She had never used any contraceptives.

Past medical history

She was a known cardiac disease patient since 1996 when she had been diagnosed with rheumatic heart disease. She had absconded follow up since 1997. She had noted that her condition was getting worse with time.

Family and social history

She was a married housewife who lived in Kangundo with her husband, she did not take alcohol, nor smoke cigarettes. There was no family history of chronic illness.

Systemic inquiry was non-revealing

Physical examination

She was a young woman propped-up who was sick looking. She was pale, not cyanosed, dyspnic. There was no finger clubbing, no jaundice, no lymphadenopathy and no splinter haemorrhages. There was bilateral pedal oedema.

The vital signs were all within normal range: Blood pressure 98/75 mmHg, pulse of 105/min of normal volume and regular, Respiratory rate of 40/min, and was afebrile with a temperature of 36.7°C.

Cardiovascular system

Pulse was 105/min, regular and good volume. Bp was 98/75 mmHg. The jugular venous pressure was raised, pericardium was hyperactive with a palpable thrill. The apex beat that was displaced at the 5th intercostal space, lateral to mid clavicular line. Both heart sounds were heard but with a mid diastolic murmur grade 4–5 loudest at apex beat.

Respiratory system

The patient was dyspnic with use of accessory muscles of respiration. Chest expansion was bilaterally equal. There were audible crepitations bilaterally and especially at the lung bases.

Abdominal examination

There was a tender hepatosplenomegally. The liver was palpable 7 cm and spleen 4 below the subcoastal margin mid clavicular line on the respective side. The fundal height was 20 weeks. The fetal heart was not easily picked.

Vaginal examination was not indicated and therefore was not done.

Diagnosis

A diagnosis of para 1+ 0 G2 with a known cardiac lesion at 22 weeks gestation by dates in congestive cardiac failure was made.

Management

She was admitted into the acute room in labour ward for stabilizing and for investigations. She was kept in the labour ward, in a propped up position, and observed for any new complications.

The investigations asked for were: Hemogram, Liver function tests, Renal function tests, HIV antibody testing, Urinalysis, Urine culture and sensitivity, Shielded chest x-ray, Obstetric scan, Echo cardiograph, Electrocardiograph.

The cardiologists were also requested to review the patient.

Results:

1 Blood group: "B" Rhesus positive

2 Liver function tests: (28/12/2005) was within normal with Total proteins 59 g/l, Alb 21 g/l, Bilirubin Direct 9.5 mmol/l, Indirect 4.5 mmol/l, AST 23 mmol/l, ALT 109 mmol/l

3 Hemogram:

Date	*WBC x10⁹/l*	*N (%)*	*L (%)*	*M (%)*	*E (%)*	*B (%)*	*Hb (g/dl)*	*Plat (%)*
8/12	6.5	58.4	29.9	4.75	5.3	1.61	9.23	424
20/12	7.88	65.2	22.9	5.24	4.55	1.77	9.24	497
14/1	5.8						9.5	268
16/2	4.9	62.5	21.1	5.0	9.26	2.16	9.26	505

The patient had a normocytic normochromic type of anaemia.

4 Urine: The cultures done the first day grew no bacteria. The cultures on 30/12/2005 grew Klebsiella species and *Staphylococus epidydymis* that were sensitive to co-amoxycluv.

5 Ultrasound done (8/12/04) showed a single intrauterine pregnancy in cephalic presentation with a normal fetal heart rate of 141 beats per minute. All limbs were outlined with no abnormality. Gestation by bipareatal diameter and femoral length averaged 23 weeks and 6 days. Placenta was fundal posterior and not low lying. Amniotic fluid was adequate. Conclusion: Viable fetus at 23+ weeks.

6 Echo done on 8/12/04: Mild to moderate eccentric mitral regurgitation, severe tricuspid regurgitation, mild pulmonary vein regugitation. The estimated pulmonary pressures were 81 mmHg.

7 Electrocardiograph showed sinus rhythm with right ventricular hypertrophy.

Medications and other management

The patient was put on strict bed rest and propped up. She was to receive oxygen by mask when needed. There was to be fluid restriction with a maximum of 75 ml/hr. She was also started on sub cutaneous Heparin 5000 IU BD, Digoxin 0.25 mg OD, Atenolol 25 mg OD, frusemide 40 mg BD and combined hematinic syrup 10 ml BD.

The clinical condition of the patient improved by the 3rd day and was gradually getting out of failure. She was transferred to the antenatal wards. While still in the ward she complained of labour pains on 22/3/2005 and she was transferred to labour ward for delivery.

In labour ward, she was admitted in acute room and placed in a propped up position, and started on oxygen by mask. Physical Examination at 12.45 pm: She was in fair general condition, not febrile, not pale, not dyspneic, not cyanosed. Cardiovascular systems: Pulse was 90/min regular, good volume, Bp was 120/70 mmHg. The murmurs had not changed much only that they were now grade 2–3. Respiratory system; Chest was clear. Abdominal examination: The fundal height was term, cephalic presentation she was having 2 contractions every 10 minutes lasting 20–30 seconds. Fetal heart was regular at 140 BPM. The presenting part was 3/5 up. On vaginal examination, there was a normal external genitalia, the cervix was 5cm dilated fully effaced, membranes were bulging, and pelvis was adequate; Artificial rupture of membranes was done, clear liquor was obtained, and there was no cord, moulding or caput. The pelvis was clinically adequate. An impression of a para 1 gravida 2 in a cardiac grade III patient in active labour was made.

She was planned for vaginal delivery. She was propped up in bed put on oxygen by mask, the partograph started. She was started on IV dextrose 10% and she was for review every 2 hours or whenever necessary. Broad-spectrum antibiotics were commenced, crystalline penicillin and gentamicin intravenously.

Review at 3.30 pm; Her general condition was stable. She was having 3 contractions every 10 minutes, each lasting over 40 seconds; the fetal heart was regular at 138 BPM. The cervix was 6 cm dilated; soft; presenting part was well applied. She was draining clear liquor. A diagnosis of active labour, with good progress was made. She was given IM pethidine 100mg for analgesia and Buscopan 20 mg stat. And she was for review after 3 hours.

A review at 6.40 PM; the patient was in stable condition. She was having 3 contractions each lasting over 40 seconds every 10 minutes. Fetal heart was regular at 138 BPM. Cervix was 8 – 9 cm diluted, fully effaced. She was draining clear liquor. The plan of management was; to monitor closely for imminent second stage and prepare for assisted vacuum delivery, and IV frusemide and syntocinon for control of bleeding.

A review at 7.10 PM; The patient reported, a feeling of bearing down, her general condition was stable. Examination revealed she was in second stage, and was taken to 2nd stage room, where she was confirmed to be in 2nd stage and an episiotomy given, and assisted vacuum delivery done. A live male infant was delivered and APGAR score of 8_1, 10_5, and 10_{10}, the infant weighed 2900gms, grossly the fetus appeared normal. IV fruseminde 120 mg was given immediately and 20 IU of syntocinon put up in a drip after delivery of the placenta. The episiotomy was repaired as described earlier in the introduction. The baby was taken to New born unit to allow the mother time for observation of the fourth stage.

Post delivery observations: Her pulse was 90 BPM, Bp 120/80 mmHg, RR 20/min. She was returned to acute room, for observations for 24 hours in 4th stage. She contained oxygen by mask, she was continued on parental antibiotics.

On 23/3/2005 at 7.45 AM; She was reviewed in labour ward acute room, she was stable, had no palpitations or dyspnea. Temperature was 37.0°C; pulse was 92/min regular; the chest was clear, breasts were active, and uterus was 20 weeks' size and well contracted.

The lochia was rubra and normal flow and not foul smelling. An impression of cardiac grade III postnatal stable patient was made. She was discharged to the postnatal ward for observations. She continued parenteral antibiotics. Rooming-in was done on the 4th postnatal day. On 6/4/2005 she was reviewed in the ward and was found to be stable and her baby was also doing well. She was discharged home to attend postnatal clinic in 6 weeks time and cardiac clinic the same time. She did not honour the appointment for six weeks postnatal clinic.

Discussion

The patient presented was a known cardiac patient since 23 years, with Rheumatic heart disease with multiple valvular lesions. She had been followed up for one year and had absconded thereafter. She was admitted as a referral from Kangundo where she had presented with anaemia and congestive cardiac failure. She went into spontaneous labour and proceeded to have an assisted vacuum delivery to a live baby. Her postnatal period was unremarkable.

There is wide regional variation (0.3–3.5%) in incidence of maternal heart disease during pregnancy with a global figure of around 1% (1). Heart disease in pregnant women is most commonly due to rheumatic heart disease, congenital abnormalities and less commonly due to ischaemic heart disease or cardiomyopathy (2). In developing countries, rheumatic heart disease is still predominant and continues to be a major cause of maternal mortality (3). Sequeira and Ojiambo in 1969 at Kenyatta National Hospital found an incidence of 0.5% with 95% of cases being of Rheumatic heart disease (RHD) origin, 35% of the RHD cases had mitral stenosis (3). In a later study Ngotho reported an incidence of 0.66% (4) again with 86.4% due to Rheumatic heart disease and 12.9% congenital heart disease (4). These results are similar to other studies from the African region where Rheumatic heart disease is predominant (5).

Rheumatic heart disease is the commonest heart disease in pregnancy in our set up in contrast to the developed world where congenital heart disease predominates. However with improving medical services and advancement on cardiac surgery some women with congenital heart abnormalities will not only survive to reach the age of child bearing, but also carry a pregnancy to term successfully (5).

Pregnancy is associated with major haemodynamic changes in the cardiovascular system that can contribute to greater morbidity and mortality in women with underlying heart disease. Therefore, the management of these disorders in the pregnant patient requires understanding of cardiovascular physiology during pregnancy, labour, delivery and the puerperium (6).

Contrary to the West, rheumatic heart disease (RHD) still predominates in developing countries such as India, where it constitutes 40–50% of all cardiac diseases during pregnancy. Among patients with RHD and pregnancy, mitral stenosis (MS) is the

most common lesion (1,2,7). Bhatt in 1978 found that the majority of his patients had combined mitral stenosis and regurgitation.

The management of heart disease in pregnancy is dictated by functional capacity of the heart and special emphasis should be placed on prevention and early detection of heart failure. The severity of heart disease is usually graded according to the New York Heart Association (NYHA) classification. The grading is clinical and depends on cardiac response to physical activity with no relationship to the extent of the heart lesion (7,8). The management calls for team approach involving obstetrician, cardiologist and anaesthetist.

Grade one and two patients are managed as outpatient after clinical evaluation. They are seen frequently by both the cardiologist and obstetrician as their grades may change to higher grades, and present with complications. At 36 weeks they are admitted into the ward to await delivery (8). Grade three and four patients are usually confined in the wards until delivery.

Restriction of maternal physical activity tends to avoid cardiovascular compromise and improves uteroplacental perfusion (7,9). The supine position should be avoided as pressure on the inferior vena cava reduces venous return. Haematinics are recommended for the prophylaxis of anaemia or its vigorous treatment when it occurs. Respiratory infections must be treated with antibiotics and oxygen liberally given if respiratory difficulties develop. It is imperative to await the spontaneous onset of labour since induction is associated with significant haemodynamic changes that could precipitate cardiac failure and incase of failed induction Caesarean section carries an added risk of pneumonia, ineffective endocarditis and pulmonary embolism (3,7,8). However Caesarean section should still be performed if there is an obstetric indication(s).

Relief from pain and apprehension without undue depression of the infant or mother is especially important during labour and delivery. The mother should be kept in a semi recumbent position in bed and oxygen given by mask if need be. The patient should be started on parenteral antibiotics and for grade three or four patients, digoxin and frusemide administered. Monitoring of vital signs, auscultation of lung bases are important to detect early signs of congestive cardiac failure. A tray containing aminophylline, digoxin, morphine, sodium bicarbonate and frusemide is kept ready for

use if need arises. Vaginal delivery should be aimed at with shortening of 2nd stage by use of elective vacuum extraction. Ergometrine should be avoided during the third stage of labour, and syntocinon used early if bleeding is excessive.

A bolus of frusemide 40–120mg is given immediately after the delivery of the anterior shoulder to offset the anticipated cardiac output increase from the placental bed. Close obstetric and medical surveillance must continue particularly during the first 24-48 hrs and infection guarded against especially infective endocarditis. Early ambulation is encouraged for patients with medical disorders complicating pregnancy.

Surgical or catheter-based angiographic correction of cardiac lesions improves pregnancy outcome. The cross-sectional area of the valve can be increased by approximately two-fold by balloon interventions, thus reducing the complications associated with cardiac overload during pregnancy. This explains the better maternal and fetal outcome seen in this study in those pregnant women who underwent corrective valve interventions (10). Pavankumar et al. (11) demonstrated downgrading of functional class NYHA III and IV to NYHA I following closed mitral valvotomy during pregnancy.

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Case 5: Cervical incompetence – McDonald stitch insertion

Name	: D.A.	**Date of admission**	: 15 Aug. 2003
Age	: 20 years	**Date of discharge**	: 18 Aug. 2003
I.P. No.:	0812277		
Parity	: 1 + 4 Gravida 6	**L.M.P.**	: 12 May. 2003
		Expected date of delivery	: 15 Feb. 2004
		Gestation by Dates	: 13+ weeks

Presenting complaints

The patient was admitted through antenatal clinic 18 with history of early pregnancy losses attributed to cervical incompetence. She was amenorrheic for 13+ weeks was scheduled for McDonald stitch insertion

History of presenting complaint

She had been followed up in antenatal clinic and found to have an incompetent cervix and had been advised to have a McDonald stitch inserted and she was admitted for the same. She had no complaints. There was no par vaginal discharge or bleeding. Micturation and bowel habits were normal.

Obstetrics and gynaecology history

Her menarche was at 12 years, her cycle was regular coming every 21 days and lasting 4 days. She had used oral contraceptives between 1991 and 1993, and experienced no complications.

She was a para 1+4; Gravida 6, pregnancies were as follows:

1st pregnancy: 1995, she delivered by SVD to a 3.2kg female who was alive and well.

2nd pregnancy: 1997 March had a spontaneous abortion at 3 months evacuation was done.

3rd pregnancy: 1997 September had spontaneous abortion at 2 months, evacuation was not done.

4th pregnancy: 1999 had a spontaneous abortion at 5 months with a McDonald stitch in situ, she went to hospital and the stitch was found to have given way, evacuation was not done.

5th pregnancy: 2000 had a spontaneous abortion evacuation was not done.

For the current pregnancy, she had done and obstetric scan on 11 Aug which showed a single intrauterine fetus at 14 weeks with a funnel shaped cervix.

Antenatal care

She had started attending at KNH. Her antenatal profile was as follows: Blood group O positive; Serology for VDRL and HIV was Negative; Brucellosis test was Negative; Haemoglobin 13.0g/dl.

Past medical history

No previous hospital admissions from other ailments apart from mentioned obstetric problems.

Family and social history

She was a married primary school teacher who never used alcohol nor smoked cigarettes. There was no history of twinning or chronic illness in the family.

Systemic inquiry was non-revealing.

Physical examination

Young lady in fair general condition, not pale, had no jaundice, no lymphadenopathy and no cyanosis. Her vital signs were a blood pressure of 110/60 mmHg, pulse 84/beats per minute, respiratory rate 20/minute, temperature 36.7°C.

Respiratory, cardiovascular and central nervous systems were essentially normal.

Abdominal examination: The fundal height was 14 weeks

Vaginal examination

Speculum examination showed normal external genitalia, the vaginal portion of the cervix was about 1 cm long, it was otherwise healthy and no obvious defect was noted. The os was closed. A digital examination was not done.

Diagnosis

A diagnosis of bad obstetric history secondary to cervical incompetence at 13+ weeks gestation.

Management

She was planned for a McDonald stitch insertion.

Investigations done

1. Check Haemoglobin 13.0g/dl
6. Biochemistry: K+ 3.5mmol/l, Na+ 140 mmol/l, Urea 2.5mmol/l

Informed consent was obtained and patient prepared for theatre, premedication IM atropine 0.6mg half an hour before theatre was given.

In theatre

The patient was anaesthetised and placed in lithotomy position. A McDonald stitch was inserted using silk 2 braided suture, bites were taken at the level of internal os at 2, 10, 7, and 5 o'clock, the knot was made at 1 o'clock position. There was minimal bleeding. Postoperatively, the patient was put on bed rest, amoxicillin, salbutamol, and Phenobarbital each for 5 days. She was discharged after 1 day.

She was followed up in the antenatal clinic. On one of her visits she complained of reduced fetal movements. A Biophysical profile score done, showed a single intrauterine fetus at 35 weeks in breech presentation, with a score of 6/10 due to the reduced amniotic fluid and movements, but umbilical blood flow was still normal. Estimated fetal weight was 2.4 kg. In view of above, a diagnosis of severe fetal distress was made, and the patient was planned for an emergency Caesarean section. An emergency Caesarean section was performed and the outcome was live male infant, who scored 7/1, 8/5, weighed 2.2kg. Intraoperatively there was no obvious cause for the fetal distress, the placenta and cord were grossly normal. The stitch was removed at the same sitting. The placenta was 400g. Baby was admitted to Newborn Unit (NBU) in view of prematurity but was discharged on the second day. Postoperatively the mother recovered uneventfully. Both mother and baby were well and were discharged home on 5th postoperative day and asked to come to the clinic for postnatal checkup after 6 weeks.

Six weeks postanatal visit, the wound had healed well, and the baby was breast-feeding well. The mother opted use progesterone only oral contraceptives as a form of contraception.

Discussion

The patient presented was a para 1+4 gravida 6 who had 2 second trimester spontaneous abortions and 2 first trimester spontaneous abortions. One of the second trimester abortions occurred with a McDonald stitch in situ, but was thought to have been due to poor technique since the stitch was found to have given way. Clinically she had a short cervix (<2 cm on the portio vaginalis). She was inserted McDonald stitch at 14 weeks and she carried the pregnancy till 35 weeks, then she had fetal distress and an emergency Caesarean section was performed. Infectious causes of preterm delivery had been ruled out earlier, the reason for the preterm delivery was never clear. The outcome was a live baby scored well and the mother and baby were both discharged home in good condition.

The term cervical incompetence is applied to a discrete obstetrical entity. Lash and Lash (1950) first used the term cervical incompetence while describing repetitive abortion presenting as "...sudden rupture of the bag of waters followed by a rapid and relatively painless extrusion of the products of conception" (1). Currently it is thought to be a nebulous and objectable term (2), the term preferred being 'premature cervical dilatation without labour'. It is characterized by painless cervical dilatation in the second trimester or perhaps early in the third trimester, with prolapse and ballooning of membranes into the vagina, followed by rupture of membranes and expulsion of an immature fetus. This term tries to explain the cause spontaneous abortions thought to be owing to cervical factors. Unless treated, this sequence tends to repeat in each pregnancy (3). This was the case in the patient presented.

Cervical incompetence is a condition in which the cervix of a pregnant woman begins to open (dilate) and thin (efface) before the pregnancy has reached term. "Incompetence" refers to the weakness of the muscle of the cervix, which cannot be voluntarily controlled. Cervical incompetence has been recognized as a small but important contributor to preterm delivery. Lesser degrees of cervical dysfunction may be more common factors in premature labour than previously realized (4).

Cervical incompetence occurs in 1-2% (4) of all pregnancies, however, it is the cause of 20-25% of miscarriages in the second trimester as well as 10% of preterm deliveries (3,4,5).

There are controversies about the case definition and diagnostic criteria. The diagnosis being made from eliciting a "classic history" (6) described as repetitive, acute, painless, second trimester evacuation of the uterus without associated bleeding or contractions. In practice, most patients do not present with this history. More commonly they are seen with histories of pregnancy loss that do not fit this classic picture. Symptoms and signs that may accompany cervical incompetence include urinary frequency, lower abdominal pressure, a bearing-down sensation, bloody show, or a watery discharge (7). Additionally menstrual like cramps and a pattern of uterine irritability or frequent small-amplitude contractions may occur as the membranes protrude, distend the cervix, and activate the Ferguson reflex. Consequently, during pregnancy the diagnosis of cervical incompetence is not always straightforward. Another presentation of cervical incompetence is the incidental discovery of an abnormally short or dilated cervix during prenatal ultrasound (2,7). Some diagnosis criteria used such as passage of an 8-mm Hegars dilator and traction on an intrauterine Foley catheter have poor validity (2). There is a lot of discussion on the correlation between anatomic measurements and function of the cervix in pregnancy (2). D.A. had the diagnosis made on history.

The cause of cervical incompetence is unknown but may be multifactorial (2). It is thought to be due to either an inherent (congenital) weakness of the cervix tissues or a result of forced dilatation or trauma. Congenital weakness can occur unexpectedly or as a result of exposure to diethylstilbestrol before birth (2,6,8). Studies have found that if surgical dilatation of the cervix is performed, the risk of cervical incompetence depends upon the number and degree of dilatation used. It is unlikely to occur because of a diagnostic dilation and curettage (e.g. for irregular periods) or after a miscarriage, when the cervix is already starting to open. No increase in risk with up to two first trimester surgical terminations of pregnancy has been found (8). Three or more does carry an increased risk, of about 12%. D.A. could have had cervical trauma during the first delivery and subsequent evacuations exarcebating the condition.

There is no clear-cut way of determining if a particular surgical event has caused it, either. Taking of biopsies as part of investigating an abnormal Pap smear does not increase the risk. Cold Knife cone Biopsy of the cervix to treat cervical pre-cancer is associated with 18% risk of cervical incompetence (9). If a biopsy of the cervix is required, this should be as small as necessary to meet the therapeutic requirements (10).

Loop diathermy or Large Loop excision of the Transformation Zone (LLETZ) has not been found to be associated with an increased risk (10).

The other risk factors for cervical incompetence are malformation of the cervix, multiple pregnancy and damage of the cervix during a prior difficult delivery (2,7,8). The occurrence of this condition in primigravida suggests uterine anomalies, prenatal exposure to DES, abnormal histology of the cervix as well as inheritance (2). The most acceptable theory of the cause is an asynchrony between the uterine corpus and cervix giving a more biochemical than an anatomical cause (2).

The goal of intervention is successful prevention of premature delivery. This allows the foetus time to develop as fully as possible, thus preventing the multitude of complications associated with prematurity (2,4,7,8). Treatment consists mainly of a procedure called cerclage, along with bed rest and possibly medications to prevent contractions and premature delivery. The majority of procedures in use today are modifications of one of the three different surgical procedures: Shirodkar, McDonald, or transabdominal cervico-isthmic cerclage (2,3,11). Other techniques reported as treatments of incompetent cervix include such diverse therapies such as electrocautery of the internal os, scarification with benzoin and talc, pessaries, intravaginal balloon devices, and various hormones (2,3,6). D.A. had a cerclage suture inserted transvaginally.

Transabdominal cerclage placed at the level of the uterine isthmus has been recommended in some instances, especially in cases of anatomical defects of the cervix or failed transvaginal cerclage (2,3). The procedure requires laparotomy for placement of suture and another laparotomy for its removal, for delivery of the fetus or both. The potential for trauma and other complications initially and subsequently is much greater with this procedure than with the vaginal procedures. There is also an increased risk of hemorrhage as the surgery is performed in a highly vascular area adjacent to the ureters (3). There are descriptions of cervical assessment before pregnancy to try and detect those who may benefit from a stitch. These include checking cervical resistance or compliance, with a dilator, or specialized instrument. Whilst promising, no studies have yet found a predictor of poor outcome as good as a previous pregnancy loss (8).

Even with cerclage and additional therapies the risk of preterm birth is high (about 25%). Preconception cerclage is associated with infertility rates as high as 50% (6,8).

Consequently most authors advocate placement after conception and usually after the first trimester in hopes of avoiding unnecessary procedures in patients who are destined to spontaneously abort. Finally if there are indications for prenatal genetic diagnosis, one may consider early chromosome diagnosis via chorionic villus sampling or early amniocentesis. D.A. had the suture inseted at the begining of the second trimester.

Contraindications to cerclage include active bleeding, preterm labour, ruptured membranes, chorioamnionitis, hydramnios or the confident diagnosis of a lethal fetal anomaly. Additionally, most authors agree that, beyond 26 weeks' gestation cervical cerclage should not be offered as neonatal survival at this gestational age now exceeds 50%. Patients presenting with cervical incompetence beyond 25 weeks' gestation have been managed with prolonged bed rest without cervical cerclage with remarkable success (7). Like that of preterm labour, the diagnosis of cervical incompetence can be difficult to make, and the problem may be overdiagnosed. Risk factors have poor predictive value, treatment options are limited, and treatment must often be initiated after changes in the cervix have occurred. There is no completely accurate test to diagnose cervical incompetence, and the diagnosis is often one of exclusion made on the basis of a history of pregnancy loss or the finding of advanced cervical dilatation well before term (2,3). Cervical incompetence is an important, but undoubtedly over-diagnosed, condition.

The treatment of cervical incompetence is surgical, consisting of reinforcement of the weak cervix by some type of purse-string suture (2,6,7, 8). Prior to insertion of the stitch it is advisable to confirm fetal viability and exclude major fetal anomalies by ultrasound and to insert the stitch at 14 weeks by which time any first trimester fetal wastage could have already occurred. Obvious cervical infection should be treated and cultures for gonorrhoea, chlamydia, and group B streptococci are recommended. For at least a week before and after surgery, there should be no sexual intercourse (2). D.A. was adviced against coitus after insertion of the suture.

If there is a question as to whether cerclage should be performed, the woman is placed at decreased physical activity. Proscription of intercourse is essential, and frequent cervical examinations should be conducted to assess cervical effacement and dilatation. Some recommend weekly ultrasonic surveillance of the lower uterine segment between 14 and 27 weeks (3). Unfortunately, rapid effacement and dilation develop even with such

precautions (3). The more advanced the pregnancy, the more likely surgical intervention will stimulate preterm labour or membrane rupture. For this reason, some prefer bed rest rather than cerclage some time after mid-pregnancy. Most advanced obstetric centres usually do not perform cerclage after 24 to 26 weeks (7).

A recent study provided a review of late second trimester cerclage, commonly known as an emergency cerclage. These authors concluded that emergency cerclage could be of benefit in some women but that the incidence of complications, especially infection, is high (12).

The stitch is usually removed after 36 weeks or where there is premature labour with inevitable preterm delivery. The abdominal cerclage requires an elective Caesarean section and the stitch is usually left in-situ for future pregnancies (2,6,8,10). D.A. had the suture removed during the Caeserean section at 35 weeks.

Complications of the stitch include rupture of membranes at the time of placement, and increased risk of infection (13). Complications, especially infection, were found to be much less frequent when cerclage was performed by 18 weeks (3). When performed much after 20 weeks, there was a high incidence of membrane rupture, chorioamnionitis and intrauterine infection. With clinical infection, the suture should be cut, and labour induced. There is no evidence that prophylactic antibiotics prevent infection, or that progestational agent or ß agonists are of any adjunctive value (2). Furthermore there is a risk of cervical damage from tears if the stitch is not removed before uterine activity becomes well established during labour. Rupture of the uterus may be a consequence of vigorous uterine contractions with the ligature in place. Membrane rupture during suture placement or within the first 48 hours of surgery is considered by some to be an indication to remove cerclage. Still, the range of management options spans from observation to removal of the cerclage with observation to removal of the cerclage and labour induction (3). There are insufficient data upon which to base any firm recommendation.

The results of cervical cerclage are difficult to assess because the diagnosis of cervical incompetence cannot be made with certainty in every case. Success rates approaching 85 to 90% are achieved with McDonald and modified Shirodkar techniques (3). Thus, there appears to be little reason for performing the more complicated original Shirodkar

procedure. The modified Shirodkar procedure is often reserved for previous McDonald cerclage failures and structural cervical abnormalities. Success rates are higher when cervical dilatation was minimal and membrane prolapse was absent (3).

There is an overall successful pregnancy rates usually in excess of 70%, in patients who had poor results without its use (14). Such reports are however, unsatisfactory because the patient acts as her own control and there is a great danger of selection bias. A much larger study (15) found more favourable results in the group submitted to cerclage but they were only marginally statistically significant; this trial, however, only included women in whom the clinician was uncertain of the benefits and the results might have been more conclusive in those women with more clearly defined evidence of cervical incompetence. The results, suggest that at present, widespread use of cervical cerclage is not justified and the procedure should be limited to those women with the clearest evidence of cervical incompetence (7).

Cervical incompetence has been thought of as an all-or-nothing phenomenon, but a continuum from cervical incompetence to premature labour has been described previously. In 1964, Bishop described cervical changes associated with the successful induction of labour at term. He noted that women with similar changes before term may be likely to deliver prematurely than those without such changes. Bishop suggested that anatomical changes in the cervix may alert physicians that patients are at risk of preterm delivery among women with cervical effacement detected on pelvic examination. Subsequent studies have used transvaginal ultrasound measurements to measure the length of the cervix in women at risk of preterm birth (3,16). Most of these studies support the concept of a continuum of cervical competence and the role of this factor in preterm delivery. The clinical importance of the association between the length of the cervix and preterm delivery is unknown. Even women with markedly shortened cervix are more likely to deliver at term than before 35 weeks, so practitioners cannot use such information to change management patterns (16).

Transvaginal ultrasound (TVS) during pregnancy has shown some promise. The usual length of the cervix is about 4cm as measured on TVS. Women with a cervical length of less than 2.5cm have been found to have a 50% risk of preterm delivery in one study. Other studies have looked at opening of the internal os ("funnelling" or "breaking")

in response to pressure on top of the uterus. It does seem that this finding in early pregnancy is suggestive of cervical incompetence and that the findings are progressive throughout pregnancy (17). The only scan done in D.A. was an abdominal scan to confirm viability and to rule out congenital malformations.

These studies are at an early stage and it takes a great leap to presume that on the basis of these findings alone a stitch will improve things. A recent study whose objective was to compare different management strategies for women at risk of cervical incompetence concluded that transvaginal ultrasonographic serial follow-up examinations of the cervix in women at risk for cervical incompetence, with secondary interventions as indicated, appears to be a safe alternative to the traditional prophylactic cerclage (18). Transvaginal ultrasonographic follow-up examination of the cervix can save the majority of women from unnecessary intervention. Placement of a therapeutic cerclage may reduce the incidence of preterm delivery at < 34 weeks' gestation among high-risk patients.

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Case 6: Severe preeclampsia with complication – blindness

Name	: S.A.	**Date of admission**	: 21 Mar. 2005
Age	: 30 years	**Date of discharge**	: 10 April 2005
I.P. no.	: 1017130		
Parity	: 4 + 1 Gravida 6	**L.M.P.**	: 3 Aug. 2004
		Expected date of delivery	: 10 May 2005
		Gestation by Dates	: 30+ weeks

Presenting complaints

This patient was referred from a private clinic in Riruta with severe P.E.T. She had presented there with a 1 week history of blurring of vision, dyspnea on exhaustion and reduced fetal activity.

History Presenting Illness

The blurred vision was associated with a persistent global headache. She had diminished visual acquity with history of seeing yellow spots over the period of sickness. She did not report loss of consciousness or fits.

The dyspnea on exhaustion was gradually worsening. There was no cough, orthopnea or paroxysmal nocturnal dyspnea. There was a history of non-dependant oedema of the lower limbs and fingers. There was no history suggestive of heart disease. She reported normal micturition habits. There was no abdominal pains and no history of jaundice. There was no history of swelling of the legs or upper limbs. She had not been put on any treatment from her referral clinic.

There was reduction in fetal activity with occasional weak fetal kicks.

Ante natal period

She started attending her antenatal care in a private clinic in Riruta, Nairobi. Antenatal profiles were not done. She had received 2 tetanus toxoid injections and was scheduled to have a BTL after delivery due to having the desired family size.

Obstetric and Gynaecologic History

She was a Para 4 + 1 gravida 6 with 4 living child.

Contraceptives used: She had used Depo-provera her last injection being in Jan. 2004

No.	Year	Gestation	Duration of labour	Delivery	Outcome	Comments
1	1992	term	? (home delivery)	SVD	3kg, M	alive and well
2	1994	term	12 hrs	SVD	4.5kg, M	alive and well
3	1996	term	8 hrs	SVD	3.5kg, F	alive and well
4	1999	term	? (home delivery)	SVD	3.5kg, M	alive and well
5	2004	3 months	abortion (not evacuated)			

Past medical history. This was non significant.

Family and social history

She was a married lady, unemployed, a teetotaller and living with her spouse who was the father of all her other children. The spouse was a casual labourer. They did not smoke cigarettes. She was a third born in a family of 3 siblings. There was no history of chronic illness in her family.

General condition

She was in a fair condition. She was not pale, not jaundiced and did not have oedema. The vital signs were: Blood pressure 152/95 (referral BP 180/120), pulse of 102/min of normal volume and regular, Respiratory rate of 24/min, and was afebrile with a temperature of 36.5°C.

Central nervous system

The patient was conscious, not restless, oriented in time, space and person. The neck was soft and the Kerning's sign was negative. The Reflexes were brisk.

Abdominal examination

The abdomen was uniformly distended. There was no reported area of tenderness. The liver span was normal with no tenderness over the hypochondrial and epigastric regions.

The fundal height was 26 weeks (and not correspinding to dates), the presentation was cephalic. Fetal heart was heard and regular at 136 beats/ minute. There were no contractions palpated.

Vaginal examination

She had normal external genitalia. The cervix was in mid position, soft, about two

centimetres long and with a parous os. There was no per vaginal discharge or bleeding. The Bishop score was 5/13

Impression

An impression of Severe P.E.T. at 30 weeks gestation with a poor Bishops score was made.

Management

The patient was admitted. Management plan was to do the following:

1. Investigations: Urinalysis, Liver function tests, Urea, creatinine, Uric acid and Electrolytes, Full blood count, and Obstetric scan with resistive index
2. Intravenous canullars were inserted. She was started on:
   a) Intravascular Hydralazine 10 mg as a stat dose over 15 minutes.
   b) Hydralazine 40 mg in a drip of normal saline in a titrated manner to maintain the blood pressure at a diastolic blood pressure of approximately 90 mmHg.
   c) An intravenous bolus of Magnesium Sulphate 4 g stat over 10 minutes
   d) An intravenous drip of Magnesium sulphate to run a rate of 1 g per hour for the next 24 hours.
   e) Oral antihypertensives were given: Methyldopa 500mg Q.I.D.

Results of investigations

a) Urinalysis: This showed a proteinuria of 2+ on dip stick.
b) Liver function tests: Total protein 95g/dl, Albumin 53 g/dl, Bilirubin 2.9 µmol/l, AST 5µmol/L.
c) Biochemistry: Urea 3.6 mmol/l, creatinine 96 µmol/l, Uric acid 415µmol/l, Sodium 141 mmol/l Potassium 3.8 mmol/l.
d) Full hemogram showed; Hb 12g/dl, WBC 10 x 10^9/l, Polymorphs 78%, Lymphocytes 20%, Monocytes 1% and eosinophils 1%, Platelates 218 x 10^9/l.
e) Obstetric scan (121/3/05): This showed a single intrauterine pregnancy in cephalic presentation with normal activity. The fetal heart rate was 150/min. The placenta was anterior and not low lying. There was reduced liquor. Gestational age by BPD and AC was approximately 30 weeks. Gross fetal parts were normal. The Restrictive Index was elevated for gestation at 0.68.

A decision to deliver her the same day by Caesarean section was made due to the elevated resistive index and poor Bishops score. She was given intramascular

dexamethasone 12 mg stat and operated after about 7 hours. Intra-operatively a live female infant weighing 1.3kg, APGAR score of 2_1, 4_5, 4_{10} was delivered and was taken to the new born unit due to prematurity and the poor score. A bilateral tubal ligation using the Pomeroy's method was done on the mother at the same seating.

Post operative period

1st post op day: The patient started complaining of diminishing visual acquity. Examination revealed a patient in fair condition, who was not pale, not jaundiced and had no oedema. The vital signs: blood pressure was 142/95 mmHg (range 120-175 mmHg systolic and 79-109 mmHg diastolic). The other vital signs remained normal. The chest was clear and breast were not active.

Abdominal examination revealed a soft abdomen with the uterine fundal height corresponding to 18 weeks well contracted. Bowel sounds were present. Lochia flow was normal. The input was 1500 ml against an output of 1000 ml for 24 hrs. The baby died in New Born Unit after 22 hrs. due to respiratory distress.

2nd post op day: The patient developed blindness with no perception of light. All other parameters remained within normal range.

By the 4th day the patients vision started improving. Ophthalmologic review showed a visual acuity of counting fingers of 6 m and 1 m in the right and left eye respectively. Reflexes to light were normal. Direct ophthalmoscopy showed cotton wool spots on the retina. Slit lamp microscope examination showed Dot haemorrhages and Leopard spots on the retinal surface. There were cotton wool spots at the macula. An impression of Hypertensive retinopathy with preeclampsia with no retinal detachment noted was made. There was a possible central retinal vein occlusion. The plan was to have a strict blood pressure control with antihypertensives. The patient was also started on junior asprin 150 mg O.D. for one month.

20th post op day: The vision had improved markedly with an acquity of 6/18 and 6/9 on the right and left eye respectively. Retinoscopy revealed a reduction in the Dot haemorrhages and Leopard spots.

The blood pressure ranges over 100–140 mmHg systolic and 70–80 mmHg diastolic. The blood parameters also remained within normal. The patient continued with anti-hypertensives until her discharge on the 25th post op day. She was to be reviewed in the postnatal clinic in two weeks. She was, however, lost to follow-up.

Discussion

The patient presented had severe PET. A decision was made to deliver her by emergency Caesarean section. The outcome was a live female infant whose birth weight was 1300 grams (very low birth weight) and scored 2_1 4_5 4_{10} and succumbed in new born unit due to prematurity.

Preeclampsia, a multisystem syndrome that affects between 0.4% and 2.8% of all pregnancies in the developed countries (1), is associated with substantial maternal and neonatal morbidity and mortality (2). Preeclampsia is a syndrome characterized by: hypertension and proteinuria with or without non-dependant edema among pregnant women.

Pregnancy can induce hypertension in normotensive women or aggravate already existing hypertension. Preeclampsia is a condition peculiar to pregnancy. It is a multisystem disorder of pregnancy that is seen more frequently in women with prior preeclampsia, chronic hypertension, renal disease, insulin- dependent diabetes mellitus, and multiple gestation, and in women with abnormal uterine artery Doppler scans. It is a multisystem disease of the vascular endothelium with vasoconstriction, and vascular damage and abnormal coagulation in the mother, and varying degree of intrauterine growth retardation in the foetus. The unifying organ is the placenta, removal of which is curative with reversal of the disease in most women within 48 hours of delivery (3,4). The disease has a characteristic morphological change in the glomeruli and the severity of the glomerular lesion tends to roughly parallel the degree of proteinuria found clinically (4).

Preeclampsia is typified by a sustained rise of blood pressure to 140/90 mmHg or more at least two occasions, 24 hours apart after the 20th week gestation in the absence of evidence of an underlying cause for hypertension. Preeclampsia is uncommon before 20 weeks unless in the presence of hydatidiform mole or multiple gestation (4). Our patient presented at 30 weeks. Other predisposing factors are preexisting hypertension, renal disease, diabetes mellitus, foetal hydrops, black race, low socio-economic factor and extremes of reproductive age. A rise of blood pressure of 20 mmHg in the first trimester accompanied by proteinuria of at least 0.5 grams/litre in 24 hours also typifies preeclampsia (3,4,5). Our patient was noted to have a high blood pressure of 152/95 mmHg at 30 weeks. She had a proteinuria of 2+ at admission by dipstick. She also

manifested with non-dependant edema. The predisposing factors were black race and low economic status.

The incidence of preeclampsia varies with geographical location. It is however the most common complication of pregnancy and the largest single cause of maternal mortality and principal cause of perinatal mortality in developed countries (6,7). It occurs in about 8% of the general population in the United States (7).

Mati (8) found an incidence of 1.5–9% of all pregnancies in different parts of Kenya. At KNH, he found an incidence of 45.4/1000 deliveries while Kibaru (9), found an incidence of 5.6 per 1000 deliveries.

Preeclampsia can be classified into two categories, namely, mild and severe. Severe preeclampsia is differentiated by the following criteria:

(a) Blood pressure greater than 160 mmHg systolic or 110mmHg diastolic recorded on two occasions at least 6 hours apart with the patient at rest.
(b) Proteinuria exceeding 5g/24 hours or 3-4+ on dipstix testing
(c) Oliguria (<400 ml of urine output in 24 hours).
(d) Thrombocytopenia (platelet count <100,000).
(e) Pulmonary edema.
(f) Cerebral or visual disturbance.
(g) Epigastric pain.

S.A. had severe PET as evidenced by a diastolic blood pressure of 120 mmHg and visual disturbances. Other indicators or pointers to severe preeclampsia include the HELLP Syndrome which is characterised by presence of abnormal hepatic function, low platelet count (as above) and haemolysis (4,5,10). The other is intrauterine growth restriction. Our patient did not have clinical or ultrasonographic evidence of intrauterine growth retardation. However, the fundal height was less than dates due to the reduced liquor.

Preeclampsia is a "disease of theory" because the cause is unknown. In some mysterious way, the presence of chorionic villi in certain women incites vasospasm and hypertension (4). Pregnancy induced or aggravated hypertension is likely to develop in women who (a) is exposed to chorionic villi for the first time (b) is exposed to a super abundance of chorionic villi, as with twins or hydatidiform mole, (c) as preexisting

vascular disease or (d) is genetically predisposed to hypertension during pregnancy (10,11). There is the possibility that immunological as well as endocrine and genetic mechanisms are involved in the genesis of preeclampsia. The immunological concept is supported by the observation that preeclampsia develops in multiparous women by a new consort or in those pregnant as a result of donor insemination. Limited data suggest that excess circulating soluble fms-like tyrosine kinase 1 (sFlt-1), which binds placental growth factor (PlGF) and vascular endothelial growth factor (VEGF), may have a pathogenic role. Increased levels of sFlt-1 and reduced levels of PlGF predict the subsequent development of preeclampsia. (1,2,11,13).

Calcium, zinc and magnesium deficiency has been implicated as predisposing factors to preeclampsia (5,10,11). Preeclampsia has also been associated with increased production of prostaglandin E2 and thromboxane production and decreased prostacycline synthesis (5,11).

Mati (8), also found that preeclampsia was commoner in patients living in area endemic of malaria and was also associated with concomitant glomerular nephritis.

Proper management of preeclampsia is dependent upon early diagnosis, close medical supervision and timely delivery, the latter being the ultimate cure. As to whether expectant management or delivery is to be adopted is guided by the following factors; the severity of the disease process, the status of the mother and the foetus and the gestational age. The ultimate goal however, is the safety of the mother first and delivery of a viable infant in stable state (5,9,14). Management of mild preeclampsia is mainly conservative depending on the gestation. In severe preeclampsia the lives of both, the mother and foetus are in jeopardy and prompt action must be taken regardless of the gestation in the interest of the mother if viability is not yet reached or both if the baby is already viable. Our patient presented with severe PET with a poor Bishop's score and she was delivered by emergency Caesarean section.

In mild preeclampsia the maternal risk is small but perinatal mortality is high between 10-20% mainly from placental insufficiency. Which may be acute or chronic, intrauterine retardation, prematurity and intrauterine retardation, prematurity and intrapartum distress or stillbirth can occur (5,12). The risks to the mother include development of eclampsia

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Case 7: Gestational diabetes mellitus – live birth

Name	J.W.	**Date of admission**	3 Aug. 2005
Age	35 years	**Date of discharge**	12 Aug 2005
I.P. No.	0718112		
Parity	1 + 1 Gravida 3	**L.M.P.**	9 Nov. 2004
		Expected date of delivery	17 Aug. 2005
		Gestation by Dates	38 weeks

Presenting complaints

J.W. did not have any complaints. She was admitted via the antenatal clinic where she was found to have elevated fasting blood sugar at 38 weeks gestation.

History of presenting complaints

J.W. had been seen at the ANC clinic and found to have a random blood sugar of 8.8 mmol/l. A fasting blood sugar done the following day was 8.0 mmol/l. She was thereafter admitted for serial blood sugar monitoring and control. On admission, she gave no history of symptoms of polyuria, polydypsia or polyphagia. She had no urinary symptoms or per vaginal discharge. She had not been known to have had impaired glucose tolerance. Foetal movements were perceived and normal.

Obstetric and gynaecologic history

She was a para 1+1, gravida 3 with 1 living children. The 1st pregnancy ended in an spontaneous complete abortion at 12 weeks. She delivered in 2001 in KNH, by Caesarean section at term due to reduced fetal movements in a patient with gestational diabetes. The outcome was a live male infant who weighed 4200gms.

She attained menarche at 14 years of age. Prior to the pregnancy, her menses were regular occurring after 30 days and lasting about 5 days. The flow was occasionally heavy with associated dysmenorrhoea. She had used and intrauterine contraceptive device that was removed in may 2004 for contraception.

Antenatal period

She had been followed up at the KNH antenatal clinic from 20 weeks gestation. She had no complaints and was found to be in good general condition. Vital signs were normal. She weighed 65kg initially and had gained 14kg by term. Antenatal profile done revealed, blood group A-positive, Hb of 11.4g%, serology for VDRL and HIV were

negative. Her random blood sugars had ranged from 5.2mmol/l to 9.5 mmol/l. She had been on insulin both long (18 IU per day) acting in combination with short (8 IU per day) acting. She had opted to have an elective Caesarean section.

An ultrasound done at 34 weeks gestation showed a single intrauterine foetus at 34 weeks 4 days maturity and in cephalic presentation. The foetal body movements and cardiac activity were observed. No foetal abnormalities were seen. The placenta was fundal and liquor volume was adequate. The estimated foetal weight was 2831gm. The expected date of delivery by ultrasound was 10.8.05.

Past medical history

J.W. was not known to have diabetes outside pregnancy. There was no history of any other chronic illness. She had no known allergy to any drugs or foods.

Family and social history

She was a teacher who lived with her husband who was also a teacher and child. She did not take alcohol or smoke cigarettes. She denied any family history of diabetes or other chronic illness.

Physical examination

She was in good general condition, was not pale and had no oedema, jaundice, oral thrush or lymphadenopathy. Her blood pressure was 110/70mmHg, pulse rate 84/min., respiratory rate 20/min and temperature 36.4°C. Body weight 80kg.

Cardiovascular, respiratory and central nervous systems were essentially normal.

Abdominal examination

The abdomen was uniformly distended and moved with respiration. A healed Pfannenstiel incision was noted. Fundal height corresponded to 36 weeks gestation with the foetus in cephalic presentation and longitudinal lie. Foetal heart rate was regular at 142/min. The liver, spleen and kidneys were not palpable. There were no areas of tenderness.

Pelvic examination

This was not indicated then and was therefore not carried out.

Impression

An impression of gestational diabetes in a 35 year old para 1+1 at 38 weeks gestation was made.

Management

She was admitted for serial blood sugar monitoring and control of sugar levels.

Investigations

1. **Serial blood sugar:** Fasting blood sugar (6.00 a.m.) ranged from 5.0–9.8mmol/l
   Preprandial (11.00 a.m.) ranged from 6.2–10.6mmol/l
   Postprandial (3.00 p.m.) ranged from 6.5–9.7mmol/l
2. **Serum biochemistry:** Sodium 133mmol/l, Potassium 4.0mmol/l, BUN 4.2mmol/l, Creatinine 60μmol/l
3. **Hemogram:** WBC 9 x 10^9/l, Neutrophils 68%, Lymphocytes 24%, Monocytes 8%, Hb 11.5g/dl, Platelets 277 x 10^9/l
4. **Urinalysis:** PH 5.0, Glucose glycosuria 1+, Protein Nil, Ketones Nil, RBC Nil, WBC 0-I/HPF, Epithelial cells Few, Deposits: no cysts, no yeast, no trichomonas

She was commenced on subcutaneous soluble insulin 10 units given three times in a day given thirty minutes before meals. She was put on a diabetic diet. Blood sugar control was monitored daily. Foetal well being was monitored by foetal kick chart and daily examination. On the third day after admission she reported lack of foetal movements. She was examined and foetal heart tones were heard. A decision to deliver her by urgent Caesarean section was made. She was informed about this decision and informed consent was obtained. Blood was drawn for pre-op investigations. Hb was 11g/dl, renal function tests were normal, fasting blood sugar (FBS) was 4.6mmol/l. The pubic hair was shaved and she was wheeled to theatre.

In theatre aseptic catheterization was done and then placed in the supine position. The abdomen was cleaned and draped. General anaesthesia was induced and the abdomen was opened through the old scar (pfannenstiel incision). A live male infant was extracted with an Apgar score of 8_1, 9_5 minutes with a weight of 4000g, the baby was taken to the new born unit. Membranes and placenta were delivered whole and complete with no obvious gross abnormality noted. The uterus was sutured in layers. The abdomen was closed in layers. General anaesthesia was reversed uneventfully. Post-operatively, she

was put on broad spectrum antibiotics, crystalline penicillin, gentamicin, metronidazole and intravenous fluids.

On the 1st post-operative day she was commenced on oral sips and graduated to light diet and later continued on her diabetic diet. Serial blood sugars done thereafter were within the acceptable ranges. She was subsequently weaned off insulin.

The baby was taken to the mother on the 5th post-operative day. The wound was inspected and was found to be clean and dry. Lactation was established by the 4th post-operative day. Both the baby and mother were discharged on the 6th post-operative day. She was to be seen at the post-natal clinic after 1 week for review with blood sugars. These was found to be normal at 5.0 mmol/l. She was seen at the post-natal clinic as scheduled. The wound was healed and random blood sugar was 5.8 mmol/l. She opted to use progesterone only implants as a form of contraception.

Discussion

Presented is J.W, a 35 year old who was para 1+1 gravida 3 with 1 previous scar. She was diagnosed to have gestational diabetes and had been on insulin. She delivered via Caesarean section at 38 weeks. The outcome was a live male infant who scored well and with a birth weight of 4000gm. Both the mother and the baby did well post delivery and were discharged in good condition.

Diabetes mellitus is a clinical syndrome characterised by hyperglycemia due to an absolute or relative deficiency of insulin or resistance to the metabolic action of insulin (1). The resulting metabolic disturbance affects the metabolism of carbohydrates, proteins, fats, water and electrolytes (2). Gestational diabetes mellitus is defined as any degree of glucose intolerance which was first recognised during pregnancy (3). J.W. was discovered to have high blood sugar levels during the second trimester. This was similar to the previous pregnancy. She therefore had gestational diabetes.

The prevalence of overt diabetes mellitus in women of child bearing age in the general population is about 1%, while 1-5% of all pregnancies in the western countries are complicated by gestational diabetes mellitus (GDM) (4,5,6). In the USA, 2.6% of all live births in 1998 were complicated by diabetes in pregnancy, 90% of which was gestational diabetes (6,7). In KNH, the incidence of diabetes in pregnancy was found to be 0.15% (8).

Deterioration of glucose tolerance occurs normally with advancing pregnancy. Human placental lactogen is the hormone mainly responsible for insulin resistance and lipolysis. Prolactin levels increase 5 to 10 fold during pregnancy and may have an impact on carbohydrate metabolism. The net effect is higher fasting and postprandial glucose which facilitate placental transfer of glucose (2,3,5,6). J.W. was noted to have high blood glucose levels during the second trimester.

There are various ways of classification of diabetes mellitus. Pregnancies complicated by diabetes may be separated into two groups: i) Gestational diabetes – women with carbohydrate intolerance of variable severity with onset or first recognition during present pregnancy as in the case of our patient or, ii) Pre-gestational diabetes – women known to have diabetes outside pregnancy (5). The Diabetes Data Group or the National Institute of Health proposed a classification system based on aetiological factors and

insulin dependence (9): a) Type I; Insulin dependent diabetes mellitus (IDDM), b) Type II; Non insulin dependent diabetes mellitus (NIDDM), c) Type III; Gestational or carbohydrate intolerance, and d) Type IV; Secondary diabetes. Based on this classification, J.W. had type III diabetes.

The American College of Obstetrician and Gynecologists in 1986 and 1994 modified a classification proposed by Priscilla White about 40 years ago. It relates the onset of diabetes, its duration and the degree of vasculopathy to the outcome of pregnancy (10). The modified white's classification is shown below:

ClassAl Gestational diabetes FBS<l05mg/dl (5.8mmol/l)
2 hours postprandial glucose <120mg/dl (6.7mmol/l)
managed by diet alone.

Class A2 Gestational diabetes FBS>105mg/dl (5.8mmol/L)
2 hours postprandial glucose >120mg/dl (6.7mmol/L)
managed by insulin.

Class	Age of onset (yr)	Duration (yr)	Vascular disease	Therapy
B	>20	<10 years	None	insulin.
C	10-19	10-19	None	insulin
D	before 10	>20	chronic hypertension or benign retinopathy	insulin
F	Any	Any	nephropathy	insulin
R	Any	Any	Proliferative retinopathy	insulin
H	Any	Any	coronary heart disease	insulin
T	Any	Any	Renal transplant	insulin

As a rule, the severity and the degree of vasculopathy in pregnancy is directly proportional to poor foetal prognosis. J.W. had gestational diabetes managed by insulin. She therefore was in modified White's class A2.

The diagnosis of diabetes may be suggested by symptoms such as polyuria, polydipsia polyphagia with or without weight loss. Polydipsia and polyuria occur when blood glucose levels significantly exceeds the renal reabsorption constant for glucose. An associated osmotic diuresis with dehydration and electrolyte loss may occur (3).

Some authors have outlined history or conditions used as criteria for the suspicion of diabetes mellitus. Accordingly, screening for gestational diabetes should include an assessment of the clinical characteristics of all pregnant women to determine the risk of gestational diabetes. These include (2):

Clinical screening for gestational diabetes mellitus

Risk category and clinical characteristics	Recommendation for serum or plasma glucose screening
High risk (one or more of the following) Marked obesity Diabetes in first-degree relative History of glucose intolerance Previous infant with macrosomia Current glycosuria	At initial antepartum visit or as soon as possible thereafter; repeat at 24–28 weeks if no diagnosis of gestational diabetes mellitus by that time
Average risk The patient fits neither the low- nor the high-risk profile	Between 24 and 28 weeks of gestation
Low risk (all of the following) Age <25 yr Belongs to low-risk race or ethnic group No diabetes in first-degree relatives Normal prepregnancy weight and weight gain during pregnancy No history of abnormal blood glucose concentrations No prior poor obstetrical outcomes	Not required

Using these criteria, J.W. had a previous child had a birth weight of 4350g and had a previous abortion. She was therefore screened for diabetes.

There has been lack of consensus regarding the optimal approach to screening all mothers for gestational diabetes during antenatal period. The fourth International Workshop Conference on Gestational Diabetes held in Chicago in 1997 recommended that screening for gestational diabetes should be performed between 24-28 weeks in those women not known to have glucose tolerance earlier in pregnancy (6). In our set up, routine screening of all mothers for gestational diabetes is not done. In the case of J.W. had a previous history of gestational diabetes with a history of a big baby. Screening was done using random blood sugar. Later on, a serial blood sugars confirmed the diagnosis on admission.

The diagnosis of diabetes is confirmed by elevated blood sugar levels. A fasting venous plasma glucose value ≥8mmol/l or a postprandial (2 hours) value of ≥11 mmol

is diagnostic of diabetes mellitus. A fasting level of <6mmol/l excludes diagnosis of diabetes. A fasting level of 6-8mmol/L requires oral glucose tolerance test (OGTT) for clear diagnosis (2). In an OGTT, two or more of the values as defined by standard tables must be met or exceeded for a diagnosis of gestational diabetes to be made with the use of either test. J.W. had fasting plasma sugar of 10.6mmol/l. She did not smoke or have any diuretic therapy. Later she was found to have a 2 hour post prandial venous plasma sugar of 9.7mmol/l. She was therefore diagnosed to have gestational diabetes without confirmatory OGTT.

The management of diabetes mellitus aims at normalising blood glucose to the level of a non-diabetic individual, monitoring foetal well-being and planning deliveries at term in the absence of other complications (4,6). After diagnosis of GDM, the patient receives nutritional counselling, which is the mainstay of therapy in these patients. The aim of the meal plan is to limit hyperglycaemia, minimise hypoglycaemia and provide nutrients that will meet the fuel requirements of the foetus. The patient is put on a diabetic diet to provide 30–35 kcal/kg of ideal body weight (1800–2400 kcal/day) of which 40–50% should be provided by carbohydrates, 30% by fat and 20–30% by proteins (5). High fibre diet should be encouraged and refined sugars avoided to help in satiating hunger and help reduce hyperglycaemic surges. Diet should be divided into 3 meals and 3 snacks. Our patient was put on a diabetic diet which did not control her blood sugars adequately. If fasting blood glucose levels are at least 5.8mmol/l or 2 hour postprandial levels of at least 6.7mmol/l, insulin therapy is began (2,5,10). Some workers have recommended prophylactic insulin therapy for all GDM patients even those that are seemingly well controlled on dietary therapy. It has been shown to further reduce neonatal morbidity (11,12). J.W. was commenced on insulin due to elevated venous plasma sugars (FBS 10.5mmol/l, postprandial 9.7mmol/l) despite being on a diabetic diet. In KNH, patients are commenced on low dose regular insulin 30 minutes before meals and the dose titrated against the serial blood sugars as was done in our patient. Careful monitoring of blood glucose levels is very important. Fasting and 2 hour postprandial glucose levels are done daily until stabilization then thereafter at least twice weekly. The aim is to keep blood sugar levels between 3 and 6mmol/l. Glycosylated haemoglobin HbA_1C is used to check long-term control (2). Our patient was on both preprandial and postprandial blood sugar surveillance. The role of oral hypoglycemic (OHG) agents for control of sugars is not well stutied. However, glyburine, a second generation sulfonylurea, does not

cross the placenta and has been used to treat GDM, and its use has been comparable to insulin in improving glucose control with out evidence of adverse maternal and neonatal complications (13). OHG agents are not used in our setup and, therefore, J.W. was not given any.

Appropriate exercises have been recommended. These include the use of the upper body muscles that place little mechanical stress on the trunk region. Some workers have reported such upper body exercises have resulted in lower glucose levels and permitted cardiovascular work-out without fear of foetal distress (14,15). Foetal well being is monitored using weekly non-stress tests, if need be, contraction stress test and biophysical profile. Maternal surveillance of foetal movements from 32 weeks is an inexpensive and useful method. When elective delivery is anticipated, amniocentesis for lung maturity tests should be done (3,4). J.W. was on daily foetal kick chart monitoring.

Gestational diabetes is not in itself an indication for Caesarean delivery. Nonetheless, the rates of Caesarean delivery among women with gestational diabetes are more than double those for nondiabetic women (2). Some of the increase may be due to an increase in the number of infants with macrosomia. The timing of delivery, in the absence of maternal or fetal jeopardy, should take into account fetal growth patterns as well as the risks associated with the induction of labor and premature delivery. Surfactant-deficient respiratory distress syndrome is rare in term infants of mothers with gestational diabetes (14). Accordingly, testing of fetal lung maturation is not recommended after 38 weeks of gestation in cases in which there are reliable estimates of gestational age and good maternal glycemic control (2,5). In the absence of any obstetrical complication such as hypertension or abnormalities of foetal growth, the pregnancy should be allowed to go to term and await spontaneous onset of labour. Vaginal delivery is aimed at and Caesarean section reserved for the usual obstetric indications. However it is prudent to estimate the foetal weight before vaginal delivery is attempted. If estimated weight is >4.5kg Caesarean delivery is recommended to prevent shoulder dystocia and birth trauma. If the weight is 4–4.5kg individualized management based on the size of the patient and her previous obstetric history is necessary (11). J.W. had one previous scar due to a big baby, and reported loss of foetal movements. Foetal heart tones were heard. She was therefore delivered via a Caesarean section.

American College of Obstetricians and Gynecologists (ACOG) recommends the use of regular insulin for patients in labor. Dilution of 5 IU in 250mls of normal saline is used. The level of blood glucose determines the rate of insulin infusion. Blood sugar is maintained at 3.8–5.0mmol/l. If blood glucose is less than 100mg/dl (5.5mmol/l), no insulin is infused. If blood glucose is between 100–140mg/dl (5.5–7.8mmol) infuse 1 unit/hour. If blood glucose is 141–180mg/dl (7.8-10.ommol/l) infuse 1.5units/hr, if blood glucose is 181–220mg/dl (10–12.2mmol/l) infuse 2units/hr. If above 220mg/dl (12.2mmol/l), infuse 2.5units/hr. Adequate rehydration is maintained at 125mmol/hr of fluids. If blood sugar is below 7.8mmol/l use 5% dextrose in Ringer's lactate. If blood sugar is above 7.8mmol/l use normal saline. After delivery, the regime continues until normal meals are started. When labour is to be induced, the morning dose of insulin is omitted and blood glucose is monitored closely. For elective Caesarean section a similar infusion of insulin and dextrose is given and blood glucose level determined hourly. Operations are scheduled for early morning when sugar levels are normal. Additional insulin if required is given as bolus injections intra-operatively. Our patient was delivered via Caesarean section and the normal dose of insulin was withheld. Blood sugar was done 1–2 hours and the dose of insulin titrated against the blood sugars.

Effects of diabetes to the pregnancy can be summarised as either maternal or fetal (2,5,6,10). Adverse maternal effects include: Increased rate of abortions, a four-fold increased risk of pre-eclampsia and eclampsia, increased risk of infections; 80% of IDDM develop at least one infection which include candidal vulvovaginitis, UTI, respiratory infection, pyelonephritis, peuperal pelvic infection, increased risks of pre-term delivery, increased risks of injury to the birth canal and higher rate of Caesarean section (up to 42-72%) and increased surgical risks, high incidence of polyhydramnios which at times causes cardio-respiratory symptoms and, higher risk of postpartum haemorrhage than the general population. J.W. had no adverse maternal effects were experienced. Post-operative period was also uneventful.

Risks to the foetus include: a) congenital malformation which affect 5-10% of IDDM and account for 50% of perinatal morbidity (the proposed mechanisms include hyperglycaemia induced free radical molecules that are embryotoxic); b) cardiac anomalies are most frequent followed by neural tube defects and skeletal malformation, c) intrauterine growth restriction which occurs in up to 20% of diabetic pregnancies,

unexpected foetal demise (the proposed mechanisms decreased PO_2, increased PCO_2 and lactate due to foetal hypoxia in utero from reduced blood flow in the intravenous space as a result of villous oedema, increased foetal intravillous blood volume as a result of HbA_1C having a higher affinity for oxygen and therefore releasing less readily than HbA), d) macrosomia due to hyperinsulinaemia combined with hyperglycaemia results in glucose metabolism through the hexose-monophosphate shunt pathway with resultant increased triglyceride synthesis in foetal adipose tissue, also maternal obesity and high serum concentrations of amino acids and lipids leads to macrosomia (2). In the case presented, apart from the loss of foetal movements experienced by the mother, there was no obvious congenital anomaly. Birth weight was 4100g.

Neonatal adverse effects include: a) hypoglycaemia due to hyperplasia of the foetal pancreatic islets cells induced by chronic maternal hypergylcemia, b) hypocalcaemia (<1.75mmol/L) the cause of which is unknown. (Theories propose aberration in magnesium–calcium economy unique in diabetic pregnancy), c) hyperbilirubinaemia due to prematurity and polycythaemia with haemolysis, d) cardiac hypetrophy leading to C.C.F due to hyperinsulinaemia, and e) respiratory distress syndrome which is common with GDM rather than overt diabetes and is higher by 6 fold to general population. Fetal hyperinsulinamia inhibits pulmonary surfactant production and may also interfere with glycocorticoid enhancement of lung maturity. Up to 3% of infants develop RDS despite positive 2:1 lecithin: sphingomyelin ratio in amniotic fluid (3,10). J.W. did not develop any of the maternal, foetal or neonatal adverse effects of diabetes in pregnancy. The early neonatal period in the case presented was uneventful.

Breast feeding should not be discouraged, but the mother is advised to increase her caloric intake just before nursing because insulin requirements are lower after breast feeding and may result in hypoglycaemia (10). Our patient breast fed exclusively without difficulty.

Women with a history of gestational diabetes should use effective contraception to minimize the chance that they will become pregnant with untreated hyperglycemia, which increases the risk of birth defects in their infants. Long-term treatment with low-dose combination oral contraceptives does not appear to increase the risk of diabetes after gestational diabetes. The copper-medicated intrauterine device and the progesterone emitting IUD are highly effective contraceptives that is metabolically neutral (16,17).

Progestin implant (Norplant, Norplant-II) has minimal effects on carbohydrate metabolism and may be the ideal contraception for diabetic women. Progestin only pill may also be utilized. (18) Other safe contraception methods include abstinence and rhythm method but with a high failure rate especially in women with irregular menstrual cycles. Condoms and cervical caps may be used. Sterilization is the best form of contraception after completion of desired family size. Vasectomy offers a safer method since it is not associated with a high incidence of infection seen after tubal ligation in diabetic women (16,17,18). J.W. opted for progesterone implants.

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Case 8: Burst abdomen after Caesarean section – secondary repair

Name	A.C.	**Date of admission**	10 Mar. 2005
Age	30 years	**Date of discharge**	21 Mar. 2005
I.P. No.:	1018670		
Parity	2 + 2 Gravida 5	**L.M.P.**	4 Jun. 2004
		Expected date of delivery	11 Mar. 2005
		Gestation by Dates	38+ weeks

Presenting complaint

A.C. had presented on 10 Mar. 2005 as a referral from a private nursing home with a gaping wound post Caesarean section.

History of presenting complaint

A.C. underwent an emergency Caesarean section in a private nursing home on 3 Mar. 2005 due to prolonged labour due to persistent occipital posterior position. The outcome was a live female infant weighing 3800gms with an Apgar score of 7 at 1 minute, 8 at 5 minutes and 9 at 10 minutes. She had history of prolonged rupture of membranes prior to the operation. The uterus was acessed via a low midline incision. She had been put on parenteral antibiotics post operatively, and was on oral antibiotics by the time of admission. On the seventh postoperative day after removal of stitches the wound gaped. She opted to be transferred to Kenyatta for further management due to financial constrains.

Obstetric and gynaecological history

She was a 32 year old lady, para 2 + 2 in puerperium having delivered on 5 Mar. 2005 at a gestation of 40 weeks. In 1995 she delivered her first baby by spontaneous vertex delivery (SVD) at term. Her second delivery was at home in 1999 at term. She had spontaneous abortions in 1997 and 2004 at 4 and 2 gestation respectively.

Past medical and surgical history

She was a known asthmatic on treatment PRN but had not had an attack for 1 year.

Family and social history

She was a homemaker married to a salesman with whom she lived with in Nairobi. She never smoked cigarettes nor took alcoholic drinks. There was no history of chronic illness in the family.

General examination

She was found to be obese, anxious, with absence of pallor, jaundice and peripheral lymphadenopathy. The body temperature was recorded as 36.8°C, pulse rate was 75 beat per minute and blood pressure was 120/70 mmHg .

Respiratory. Cardiovascular and central nervous systems: were essentially normal.

Abdominal examination

The mid-points of the wound had already gaped and a loop of intestines was evidently trapped. There was no pus but there was poor granulation.

Pelvic examination

External genitalia was normal, there was minimal lochia serosa which had no offensive smell.

Impression

An impression of burst abdomen was made.

Management

The remaining skin stitches were removed thus freeing the entrapped gut causing relieve to the patient. The plan of management was explained to the patient and a consent obtained. Blood for grouping and cross-matching was taken and intravenous fluids started. A full hemogram was taken. Intramuscular pethidine 100mg was given to allay anxiety and premedication with intramuscular atropine 0.6mg stat. The protruding intestines and wound were covered with sterile gauze roll that had been made wet with normal saline. The patient was then taken to theatre.

Operation

In the operating room, she was placed in supine position and general anaesthesia administered. Vulvo-vaginal toilet was done in semilithotomy position after which urethral catheter was placed and the bladder emptied. The patient was then repositioned to supine position and the abdomen was the cleaned and draped. The gauze roll covering the intestines was wet using lukewarm normal saline then removed with ease. The loops of small intestines were examined in turn throughout the entire length to confirm viability and patency.

The pelvis was inspected and found clean. Uterine wound was found healing and intact. The wound edges of the abdomen were freshened after removal of cut gut suture used to suture initially. There were no signs of sepsis, however, specimens from the wound edges were taken for culture and microscopy. The abdomen was cleaned with Rifocin and mass closure done after freshening the edges. Using nylon loop No. 1 mass closure was performed. The skin was then closed with nylon number 2/0 using interrupted vertical mattress. Using povidone iodine solution, the wound was cleaned then dressed. The patient was reversed from general anaesthesia and later wheeled back to the ward.

Post-operative care

The vital signs were observed quarter hourly until the patient was fully awake the 4 hourly thereafter. Pethidine was given in a dose of 100 mg 8 hourly for 24 hours. Intravenous cefuroxime 750 mg twice daily and intravenous metronidazole 500mg tds were given for 5 days. She remained afebrile throughout her stay in hospital. Oral sips were commenced from first post operative day and light diet from second day post operatively. The wound was exposed on the fourth operative day was found to be dry and painted with betadine. Hemogram results were normal. Specimen cultures were found to be negative.

Her recuperation was uneventful and all stitches were removed on the tenth post-operative day by which time the wound had healed. She was discharged and advised to visit the postnatal clinic after 6 weeks.

She was however lost to follow up.

Discussion

The patient presented had burst abdomen and evisceration that was a complication of Caesarean section. Burst abdomen refers to disruption of all the abdominal layers and evisceration is when the intestines protrude through the incision. Dehiscence refers to disruption of any of the layers (1); mostly the skin.

Literature from general surgery reports a dehiscence rate of between 0.5 to 5% and 1% from gynaecology. The lower gynaecology dehiscence rate is ascribed to increased elasticity after pregnancy and the fact that the surgery is done infra-umbilically (2)

Burst abdomen is a serious complication that carries a mortality of 15-30%. It usually occurs 6-10 days post operatively and is often heralded by the "pink sign" a serosanginous discharge onto the wound dressing (1). The patient presented developed dehiscence on the 7th postoperative day. The most common cause of death being cardio-respiratory insufficiency and peritoneal sepsis (3,4,5). Wound healing in an otherwise healthy subject begins with inflammation an culminates in synthesis of collagen by a population of new fibroblasts. The 5 distinct biological processes: inflammation, epithelialization, fibroplasia, wound contraction and scar maturation. These do not occur in a strict sequlae but occur simultaneously (2,3,6).

The incidence of wound disruption after Caesarean section is reported to be between 0.14% and 4.37% (2). The local incidence has not been studied. No single aetiologic factor can account for all obstetric wound disruptions. Frequently it will be a combination of factors that ultimately leads to the complication. The various aetiological factors associated with wound dehiscence may be summarized in 4 general categories. i) Type and location of the incision, ii) Specific type of suture, iii) Inherent strength of the tissues, iv) Mechanical factors (3,4). The incidence of wound disruption is usually lower in transverse incisions as compared to vertical incisions (1). In the patient presented, the wound disruption followed a vertical incision.

The aetiology of wound disruption is associated with preoperative, intra-operative and postoperative factors. The preoperative factors implicated are mainly nutritional due to deficiency of proteins, vitamins and minerals (6,7). Other preoperative factors implicated are enhanced age and obesity (6). The patient being discussed was obese and was not advanced in age.

The intra-operative factors implicated in wound interruption are technical faults. Post-operative factors implicated are mainly infection and presence of anaemia (6,7,8,9). The technical faults include use of strong suture, non secure knots and fraying of sutures (5,6).

The tensile strength of the fascial layer is greater than any other abdominal layer hence a strong suture should be used. Catgut has an unpredictable rate of absorption as it is degraded by proteolytic enzymes and hence is not a good suture for closing the rectus sheath (9,10). Disruption of the wound due to technical faults normally occur before 6 days post-operative. The peak incidence is on the sixth to eighth post-operative day with 24% occurring between second and fifth post-operative day. Fifty five percent (55%) between sixth and ninth post-operative day and 21% after the tenth post-operative day (10). Polyglycolic acid (dexon) and polyglactin 910 (vicryl) sutures are degraded by hydrolytic enzymes and adequately maintain their tensile strength, hence, good for fascial closure. In the unused state these sutures are stronger than non-absorbable sutures. At 14 days postoperatively, they have lost 50% of their tensile strength but are still as strong as silk suture (1,10).

Monofilament nonabsorbable sutures such as nylon and polypropylene (prolene) are also good choices for closing fascia. Monofilament nylon loses only 16% of its tensile strength by 70 days and only 20% by 200 days. Silk and cotton are good sutures but they may promote infection. Stainless steel also potentiates infection (10). The patient being discussed most likely developed wound dehiscence as a result of a technical fault or due to poor nutritional status (obesity). As noted above, catgut which is not a good suture material for closing fascia had been used for this particular patient during the Caesarean section.

Wound disruption begins with separation of peritoneum then proceeds outwards. In some cases the skin is spared and the patient presents later with incisional hernia (1). When wound dehiscence occurs, the abdomen should be instantly covered with sterile gauze. Closure should be done as soon as possible to reduce morbidity and mortality that are associated with delayed closure (1,5).

In the recommended Smead-Jones closure, a through and through approach is used. Using an appropriate suture like monofilament nylon, all the abdominal layers are

brought together in a continuous fashion with stitches placed 2.5 to 4cm either side of the wound margin and about 1.5cm apart. The peritoneum and fascial are closed together. It is recommended that the stitches be removed on the fourteenth post-operative day (1,2,5). This patient was repaired with through and through nylon for the mass closure. Stitches were removed on the tenth day. The result of the secondary closure were excellent.

To prevent wound dehiscence the preoperative implicated factors can be overcome with parenteral nutrition. The intraoperative factors can be minimised with good aseptic and surgical techniques. One should avoid weak sutures, avoid too fine sutures for tissue closure, avoid damaging sutures with hemostat or fraying by improper tying. When using monofilament sutures, a square knot is advocated to prevent slipping (1,10).

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Case 9: Prelabour rupture of membranes – Induction, vaginal delivery

Name	: M.M.	**Date of admission**	: 10 Apr. 2005
Age	: 26 years	**Date of discharge**	: 11 Apr. 2005
I.P. No.:	10288444		
Parity	: 2 + 0 Gravida 3	**L.M.P.**	: 16 Jun. 2003
		Expected date of delivery	: 23 Apr. 2005
		Gestation by Dates	: 36 weeks

Presenting complaint

The patients presented with a 1 day history of draining of drainage of liquor.

History of presenting complaint

M.M. presented with a sudden gash of fluid from the vagina 8 hours prior to admission. It had drained up to her legs. The liquor was clear. She did not have per vaginal bleeding and did not have labour pains. She perceived fetal activity.

Obstetric and gynaecologic history

She was a gravida 3. Her first delivery was in 1999 and her second delivery was in 2002. Both deliveries were by SVD. She had not attended any antenatal clinic. She had attained her menarche at the age of 15 years. Her menses were regular occurring every 28 days and lasting for 4 days. She had never used any contraception.

Past medical history was not significant.

Family and social history

She was a married college teacher. She was the last born in a family of 6 siblings. She lived with her parents at Ngong.

Physical examination

She patient was in good general condition. She was not pale, not jaundiced and had no oedema. Her temperature was 36.8°C, blood pressure was 120/80 mmHg, pulse rate was 84 beats per minute and had a respiratory rate of 20 per minute.

Cardiovascular, respiratory and central nervous systems were essentially normal.

Abdominal examination

There was no tenderness on palpation. The fundal height was term, lie was longitudinal

with cephalic presentation . Fetal heart was heard at 144 beats per minute and was regular. There was no contraction palpated.

Vaginal examination

A digital vaginal examination was done. There was liquor seen flowing freely at the introitus. The cervix was about 2 cm long central, not dilated. The liquor was clear and not offensive.

Diagnosis

A diagnosis of preterm prelabour rupture of membranes was made.

Investigations

Hemogram: Hb 12.0 g/dl, WBC 8.2 x10^9/l, Plat 240 x 10^9/l.

Management

She was started on amoxycillin and was planned for induction. Syntocinon 5 IU in 5% dextrose was used to induce labour. She delivered vaginally 6 hours after induction to a live male infant that weighed 2650 gms with an Apgar score of 8 and 10 at 1 minute and 5 minutes respectively. The placenta weighed 500gms. Both the mother and infant did well post-deliverly and were discharged 1 day after delivery. The mother was advised to come for review after 6 weeks.

Discussion

The patient presented was a para 2+0 gravida 3 with preterm premature rupture of membranes (PPROM) at 36 weeks. She had no signs of chorioamnionitis or fetal distress and was induced with syntocinon and had SVD to a live infant. Her previous pregnancies were uncomplicated and she had term deliveries to live babies who were alive and well. There was no obvious precipitating factor for the PPROM. Her postnatal period was otherwise unremarkable.

The membranes surrounding the amniotic cavity are composed of the amnion and the chorion, which are closely adherent layers consisting of several cell types, including epithelial cells, mesenchymal cells, and trophoblast cells, embedded in a collagenous matrix. They retain amniotic fluid, secrete substances both into the amniotic fluid and toward the uterus, and guard the fetus against infection ascending the reproductive tract. The membranes normally rupture during labor. Premature rupture of the fetal membranes is defined as rupture of the membranes before the onset of labor (1). It is also known as prelabour rupture of membranes. Prelabour rupture of the membranes (PROM) occurring before 37 weeks gestation is usually referred to as preterm prelabour rupture of the membranes. At term, 8 to 10 percent of pregnant women present with premature rupture of the membranes; these women are at increased risk for intrauterine infection when the interval between the membrane rupture and delivery is prolonged (1).

The incidence of PROM ranges from 2-18%. Approximately 60-80% of the cases with PROM occur in term patients (1,2). At Kenyatta National Hospital the incidence of PROM was reported as 8.23% by Wanjala (3) and 9.3% by Otieno (4). Although most cases of PROM occur at term, its impact arises predominantly from the 20-40% of cases that occur before 37 weeks gestational age (2). PROM occurs in approximately 1 percent of all pregnancies and is associated with 30 to 40 percent of preterm deliveries. It is thus the leading identifiable cause of preterm delivery (after less than 37 completed weeks' gestation) and its complications, including respiratory distress syndrome, neonatal infection, and intraventricular hemorrhage.

PROM which often results into preterm birth is the single most important clinical health problem encountered in the practice of Obstetrics and Gynaecology (2). It is the greatest cause (other than congenital abnormalities) of neonatal morbidity and mortality. Even for those infants who survive the sequelae of untimely birth often results in permanent

disabilities. After PROM the latency period from membrane rupture to delivery decreases inversely with advancing gestational age. For example, at 20 to 26 weeks' gestation, the mean latency period is 12 days; at 32 to 34 weeks' gestation, it is only 4 days (5). The rate of neonatal sepsis after preterm premature rupture of the membranes ranges from 2 to 20 percent, and the incidence of neonatal death caused by infection is approximately 5 percent.

The risk factors associated with PPROM include: changes in collagen content, structure and catabolism (6,7,8); connective tissue disorders such as in Ehlers-Danlos syndrome (9), nutritional deficiencies such as, vitamin C & E deficiency (10,11) and copper deficiency (12). Increased collagen degradation as a result of altered balance between Matrix Metalloproteinases (MMP) and Tissue Inhibitor of matrix metalloproteinases (TIMP) (13). Others factors include cervicovaginitis, incompetent cervix, cigarette smoking, chorionic villus sampling, amnioscentesis, prior PROM or preterm delivery, prior cervical surgical procedures, placenta abruptio, marginal cord insertion, and hydramnios (2,3,6).

Infections are associated with collagen degradation and PPROM. There has been debate on whether intrauterine infection is a cause or a consequence of premature rupture of the fetal membranes. Indirect evidence that genital tract infection precipitates rupture of the membranes in animals and humans does exist. Microbial invasion of amniotic cavity is involved in 30 to 40% of patients with preterm premature rupture of membranes, and the lower the gestational age, the higher is the association with intrauterine infection. Epidemiological data demonstrate an association between colonization of the genital tract by group B streptococci, *C. trachomatis, N. gonorrhoea*, and the microorganisms that cause bacterial vaginosis (vaginal anaerobes, *Gardnerella vaginalis*, Mobiluncus species, and genital mycoplasmas) and an increased risk of PPROM (14-16). Furthermore, in some studies treatment of infected women with antibiotics decreased the rate of preterm premature rupture of the membranes (16,17).

The mechanisms responsible for membrane rupture in the setting of intraamniotic infection are poorly understood. One view is that bacteria are a source of an enzyme that degrades the extracellular matrix of membranes. Alternatively a host response to infection may participate in the mechanisms of membrane rupture. Bacterial infection and the host inflammatory response also induce prostaglandin production by the fetal

membranes, which is thought to increase the risk of preterm premature rupture of the membranes by causing uterine irritability and collagen degradation within the membranes. Certain strains of vaginal bacteria produce phospholipase A2, which releases the prostaglandin precursor arachidonic acid from membrane phospholipids within the amnion. Furthermore, the immune response to bacterial infection includes the production of cytokines by activated monocytes that increase prostaglandin E2 production by chorionic cells (18). Cytokine stimulation of prostaglandin E2 production by the amnion and chorion appears to involve induction of cyclooxygenase II, the enzyme that converts arachidonic acid into prostaglandins (18,19). The precise regulation of prostaglandin E2 synthesis in relation to bacterial infection and the host inflammatory response is not understood, and a direct link between prostaglandin production and premature rupture of the membranes has not been established. However, prostaglandins (specifically prostaglandin E2 and prostaglandin F2) are considered to be mediators of labor in all mammals, and prostaglandin E2 diminishes collagen synthesis in fetal membranes and increases MMP-1 and MMP-3 expression in human fibroblasts.(20,21).

Glucocorticoids production is another component of the host response to infection. The anti-inflammatory action of glucocorticoids is mediated by suppression of prostaglandin production. However, in some tissues, including the amnion, glucocorticoids paradoxically stimulate prostaglandin production. Furthermore, dexamethasone reduces the synthesis of fibronectin and type III collagen in primary cultures of amniotic epithelial cells (22). These findings suggest that glucocorticoids produced in response to the stress of microbial infection facilitates rupture of the fetal membranes. However, there has been no conclusive demonstration that infection precedes premature rupture of the fetal membranes in humans. Nonetheless, microbial infection and the host inflammatory response may at the very least increase the activity of matrix metalloproteinases in the fetal membranes and be involved in the pathogenesis of some membrane ruptures (22).

Other conditions associated with premature rupture of membranes include, altered hormonal balance progesterone, estradiol (23), relaxin (24,25), programmed cell death (26,27); cervical incompetence, multiple pregnancies, hydramnios, decreased tensile strength of membranes as in membrane stretch (3,28) and familial history of premature rupture of membranes are other predisposing factors (27).

The essentials of diagnosis include a history of a gush of fluid from the vagina, continued leakage of fluid from the vagina, demonstration of leakage of amniotic fluid from the cervix and demonstration of oligohydramnios by ultrasound (29). A most important step in accurate diagnosis is examination with a sterile speculum. This is conducted only after careful abdominal examination reveals no contraindication to vaginal examination. The posterior vaginal fornix is exposed by means of a speculum, and the PH of the pool fluid is tested with Nitrazine paper – if amniotic fluid present, changes from yellow to blue (Amniotic fluid PH is 7.0-7.25); hair and vermix in the pool of fluid are some of the gross indictors that it is amniotic fluid. Other procedures useful in the diagnosis are: Cervical secretions should be collection for culture; fluid should be collected from vaginal pool for potassium hydroxide and wet mount examination, and fern test for arborization, observation of leakage of fluid from cervical os on Valsalva manoeuvre, cough or fundal pressure; fluid can also be collected for determination of L:S ratio, phosphatidyl glycerol or quick surfactant test (3,29).

During speculum examination one should determine the cervical dilatation, and effacement, check for cord prolapse and if no free fluid is found give a pad and observe for subsequent leakage. Occasionally there will be such concern about whether or not membranes are ruptured that it will be necessary to perform amniocentesis and inject a dye (indigo carmine). This is done following removal of amniotic fluid for physiologic maturity testing, analysis of white cell count, or bacteria, and possible culture and sensitivity. After 15–20 minutes, insertion of vaginal speculum should reveal a dye in the vagina if the membranes are ruptured (29).

Management of preterm premature rapture of membranes takes into consideration, two important aspects; directed towards fetal lung maturity and possible chorioammontis. Assuming that no untoward prenatal outcome occurs due to cord accidents or from placental abruption, the greatest concern with prolonged rupture is the risk of maternal or fetal infection (1,3,29). Fortunately, most infants born after 34 weeks gestation survive, due to advances in neonatal care. Therefore, in conservative management, one aims to reach 34 weeks then we deliver the baby.

Diagnosis of chorioammnionitis should be followed by prompt efforts to effect delivery, preferably vaginally. Fever is the only reliable indicator for making diagnosis; a temperature of 38°C or higher accompanying ruptured membranes implies infection. Maternal Leucocytosis by itself has been found to be unreliable by most investigators

(29). The ominous signs that are watched for are; maternal temperature ≥38°C recorded at least 4 hours apart, fetal tachycardia of more than 160/minute sustained for 10 minutes, maternal tachycardia of over 100/minute, uterine tenderness, and uterine contractions. Investigations include twice weekly complete blood count (intervene if notice a rising trend or a value of over 15 x 10^9/L), weekly ultrasound to evaluate amniotic fluid volume with intervention in the event signs of severe oligohydramnios such as maximum vertical AF pocket <2cm or amniotic fluid index <3 cm. Daily evaluation of amniotic fluid leakage using pad and strict bed rest are recommended.

Use of antimicrobial agents provides marginal benefits, broad spectrum antibiotics and metronidazole are given both to prevent infection and to treat any infection that could have caused the PPROM. Use of corticosteroids is advised as well as tocolytics where not contraindicated. The combined use of corticosteroids and antibiotics has been associated with a reduced risk of respiratory distress as compared with use of corticosteroids alone (29,30). Tocolytic drugs used independently or in conjunction with corticosteroids or antibiotics have not consistently been shown to provide further improvement in outcome (31). Overall no strategy has resulted in a reduction in preterm birth after preterm rupture of membranes (31). Postpartum antibiotic is continued since these patients with PPROM are predisposed to postpartum endometritis (31).

Markers of degradation of the extracellular matrix of fetal membranes could be used to identify women who are at risk for premature rupture of the membranes and preterm delivery. The most extensively studied candidate marker is fetal fibronectin, which is present in the extracellular matrix of fetal membranes and is structurally different from the fibronectin of adult tissues. The production of fetal fibronectin by human amniotic cells is stimulated by inflammatory mediators (including interleukin-1 and tumor necrosis factor) that are considered important in initiating preterm labor (32-34).

Prevention of PROM has become a topic of considerable interest in the development of general and specific inhibitors of matrix metalloproteinases for the treatment of periodontal disease and arthritis and for the prevention of tumor metastasis. These agents include tetracycline antibiotics, synthetic matrix-metalloproteinase inhibitors such as batimastat (which selectively chelates the zinc atom at the active site of the enzymes), and the native inhibitors TIMP-1 and TIMP-2. The ability of such substances to prevent or retard changes in the extracellular matrix of fetal membranes before preterm premature rupture occurs has yet to be evaluated (35).

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Case 10: Antepartum haemorrhage – placenta previa type IV

Name	: H.G.	**Date of admission**	: 18 Jan. 2004
Age	: 35 years	**Date of discharge**	: 25 Jan. 2004
I.P. No.:	0940628		
Parity	: 1 + 1 Gravida 3	**L.M.P.**	: 13 Apr. 2003
		Expected date of delivery	: 20 Jan. 2004
		Gestation by Dates	: 40+ weeks

Presenting complaints

The patient was admitted via casualty with a history of per vaginal bleeding for 2 hours.

History of presenting complaint

She had been spotting on and off form the 26 weeks of gestation. This had been managed conservatively. The current bleeding was sudden, painless, consisting of fresh blood. She had changed 2 socked pads since the onset of the bleeding. She had no history of trauma to the abdomen or the genitals. She perceived fetal activity.

Antenatal period

She had attended ANC in a peripheral clinic. An obstetric ultrasound done on 14/10/03 that showed a single fetus in cephalic presentation with the biparietal diameter and femur length corresponded to 28 weeks gestation. The fetal heart was demonstrated at 154 beats per minute. There were adequate fetal movements. The placenta was posterior, extended low and covered the internal os. A repeat ultrasound scan was recommended at 36 weeks. This was not done. Antenatal profile were done but the results were not available.

Obstetric and gynaecologic history

She was para 1+1. Her last delivery was in 2001 by Caesarean section due to fetal distress. She had an abortion at 12 weeks in 2002 for which she was evacuated in KNH. She had used oral contraceptive pills between 2002 and 2003

Past medical and surgical history was not significant.

Family and social history

She was a married homemaker. The husband was a teacher. They lived in Eastleigh. She neither consumed alcohol nor smoked cigarettes. There was no family history of chronic illness.

Physical examination

She was in fair general condition, was mildly pale, had no edema, no jaundice and had no lymphadenopathy. Her blood pressure was 100/60 mmHg, pulse rate was 80 beats per minute, respiratory rate was 20 per minute and temperature was 36.4°C. She was on intravenous fluid drip.

Abdominal examination

There was no tenderness on palpation. The fundal height was term. The presentation was cephalic and lie was longitudinal. On auscultation, the fetal heart was detected and the rate was 140 per minute.

Speculum examination

The external genitalia was covered with blood stains. There were clots at the introitus. The vaginal walls and the cervix were intact. There was active bleeding coming from the cervical os.

Impression

An impression of antepartum haemorrhage due to placenta previa was made.

Management

A line of intravenous fluid had already been established at casualty whereby blood had also been taken for grouping and cross-matching. Two units of cross-matched blood was requested for possible emergency Caesarean section. An informed consent was obtained from the patient for double set-up examination under anaesthesia and Caesarean section. She was pre-medicated with intravenous atropine 0.6mg stat. She was then wheeled to the operating room.

In the operating room, she was placed in semilithotomy position after which vulvo-vaginal toilet was done. Aseptic bladder catheterisation was performed. Fresh per vaginal bleeding was noted and had formed more clots. The abdomen was cleaned and draped. General anaesthesia was administered. On speculum examination there was active bleeding from the cervical os. The cervix os was 3cm dilated and was about 0.5cm long. On digital examination, the fornices felt boggy all round. An impression of placenta previa type IV was made and a decision to do an emergency Caesarean section was made.

A live female infant weighing 2550gms. The Apgar score was 8_1, 9_5 and 10_{10} was delivered. The placenta was found low lying and covered the os completely. The placenta was removed by controlled cord traction. There was no gross pathology on the placenta. The placental bed contracted well and hemostasis was achieved. The uterus was found to have multiple intramural fibroids.

Post-operative management

She was managed as described in the introductory part. A check haemoglobin on the third post-operative day was 6.5g/dl. The mother was discharged on the fifth post-operative day on haematinic which she had started on the third post-operative day. She was advised on contraceptives.

Discussion

This was 36 year old lady, para 1+1 who had spotting for 3 months and then had severe per vaginal bleeding. An ultrasound done had revealed placenta previa which intraoperatively was confirmed.

Placenta previa is the implantation of the placenta in the lower uterine segment within the zone of effacement and dilatation of the cervix, thus constituting an obstruction of descent of the presenting part (1,2,3). Four degrees of abnormalities are recognized:

Type I: the major part of the placenta is attached to the upper segment and only the lower margin encroaches onto the lower segment but not up the os. The placenta edge do not actually reach the internal os but is in close proximity. This is also known as a low-lying placenta.

Type II: The placenta covers the internal os partially. This is also known as a marginal placenta previa.

The type I and II is further designated A or B if it is anterior or posterior respectively.

Type III: the placenta completely covers the internal os when closed but does not entirely do so when fully dilated. This is also known as a incomplete or partial central placenta previa.

Type IV: the placenta completely covers the internal os even after it is fully dilated. This is also known as a central or total placenta previa. (2,3)

The patient presented here had placenta previa type IV.

The prevalence of placenta previa at term is approximately 0.3-06%. Its frequency before 20 weeks is tenfold higher and approximately 5% at 16 weeks (2,3). In Kenyatta National Hospital, Ojwang (4) reported an incidence of 1 in 4,000 in 1974, while Kirima (5) reported and incidence of 1 in 116 in 1981.

Women with a history of Caesarean delivery are 50% more likely to have a subsequent birth complicated by placenta previa than those without such a history (6). Mutiparity and advancing age especially for those women with age greater than 35 years is seen to increase the risk of placenta previa (1,2,7). The other factors that increase the risk of placenta previa include smoking either passively or actively. It is thought carbon monoxide hypoxia due to smoke causes compensatory placenta hypertrophy and a

defective decidual vascularization the possible result of inflammatory or atrophic changes has been implicated in the development of placenta previa (2). The patient presented was 35 years of age and was gravidae 3 with one previous scar and a history of uterine evacuation.

Placenta previa is also associated with placenta accreta, increta or percreta. This firm attachment may be anticipated because of poorly developed decidua in the lower uterine segment (2,3,8). The most characteristic event in placenta previa is painless haemorrhage which usually does not appear until near the end of the second trimester or after. In about 10% of cases, there is some initial pain because of co-existing placental abruption, and spontaneous labour may be expected over next few days in 25% of the patients. The uterus usually is soft, relaxed and non-tender. A high presenting part cannot be pressed into the pelvic inlet (1,2,6). The patient presented here started spotting at 26 weeks gestation. On admission, she presented with painless per vaginal bleeding. There was no abdominal tenderness and the fundal height was bigger than dates with a high presenting part.

Bleeding in placenta previa may be due to any of the following causes: mechanical separation of the placenta, either during the formation of the lower uterine segment or effacement and dilatation of the cervix in labour or as a result of intravaginal manipulation, placentitis, rupture if poorly supported venous lakes in the decidua basalis that have become engorged with venous blood (2,9). The cause of the bleeding in our patient was not apparent before or during operation.

The possibility of placenta previa should always be suspected and should not be dismissed until appropriate evaluation has proved its absence. Clinical digital examination can be used to establish the diagnosis. However such examination of the cervix is never permissible unless the woman is in an operating room with all the preparations for immediate Caesarean delivery as ever gentle examination can cause torrential hemorrhage (2). The simplest most precise and safest method of placental localization is provided by transabdominal sonography with considerable accuracy of up to 96% (1,2). During the middle of the second trimester, the placenta will be observed by ultrasound to cover the internal cervical os in about 30% of cases. With development of the lower uterine segment, most of these low implantations will be carried to a higher station (2,3). Other modalities used and which may be more accurate are transvaginal

ultrasonography (10) and magnetic resonance imagine (11). The differential diagnosis include partial abraptio or circumvallate placenta (1,2). H.G. underwent sonographic study at 26 weeks gestation which revealed placenta previa. This diagnosis was confirmed intraoperatively.

Patient must be managed as in-patients once diagnosis is confirmed. Two or more units of blood should be typed, cross matched and kept ready for transfusion (1,2,3,4). Our patient was diagnosed by ultrasound at a peripheral hospital. She however was not confirmed after the ultrasonography. Women with placenta previa may be managed using the following criteria:

- those with preterm fetuses but there is no pressing need for delivery
- those in whom the fetus is reasonably mature
- those in labour
- those in whom haemorrhage is so severe as to mandate delivery despite fetal immaturity (2).

Patients with preterm fetus, fetal survival can often be enhanced by expectant therapy. Tocolytics can be used to prevent premature labour and to prolong pregnancy to at least 36 weeks. After 36 weeks the possibility that repeat haemorrhage may be associated with small intrauterine growth retardation must be considered. About 75% of cases of placenta previa are now terminated at between 36 and 40 weeks. In selecting the optimum time for delivery, tests of fetal lung maturation, including assessment of amniotic fluid surfactant and ultrasonic growth measurements are invaluable adjuvants (1,3,9).

In a patient who is unequivocally at 37 weeks gestation with evidence of uterine activity or with persistent bleeding double setup examination should be performed in theatre under general anaesthesia. The patient should first undergo a careful speculum examination where placental tissue may be revealed in the cervical os. If the diagnosis of placenta previa cannot be made with a speculum examination, the obstetrician should next examine the vaginal fornices. Fullness in the fornices suggests the presence of the placenta extending down towards the cervix. Finally the examining fingers should be carefully introduced in the cervical os to detect the placenta. In patients with type II posterior, III and IV placenta previa Caesarean section delivery is employed. In other

types of placenta previa, the membranes are artificially ruptured and the patient allowed to deliver vaginally (2). Vaginal delivery is also employed for patients with little or no prospect of salvaging the fetus (1). Our patient presented at 40 weeks gestation with active bleeding and a live fetus. Caesarean section delivery was employed and a live baby extracted that weighed 2550gms.

Maternal haemorrhage shock and death may follow severe antepartum haemorrhage secondary to placenta previa. Death may also occur as a result of intrapartum and postpartum haemorrhage, operative trauma, infection or embolism (2,3). When encountering a patient with placenta previa, the possibility of a placenta accreta, increta or percreta should be considered. The incidence of placenta accreta in patients with placenta previa without previous Caesarean section or other uterine surgery is 4% while those with prior Caesarean section or other uterine surgery is between 16–25% (3). Our patient was managed with intravenous fluid and fortunately did not go into shock.

Patients with uterine Caesarean scars and a placenta previa should have an ultrasound scan looking for thinning and distortion of the uterine serosa, the hallmark of placenta accreta. If there is suspicion of placenta accreta, patients should be counselled well in advance about the possibility of hysterectomy. Prophylactic placement of catheters for angiographic embolization should be considered if a radiologist experienced in this technique is available (12).

Prematurity (gestational age less than 36 weeks) due to placenta previa accounts for 60% of perinatal deaths (1). The high incidence of placenta previa and placenta accreta with repeated Caesarean deliveries is yet another reason to encourage vaginal delivery after previous Caesarean wherever possible (9).

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Case 11: Sickle cell disease in pregnancy – Caesarean section delivery

Name	: P.M.	**Date of admission**	: 10 May. 2005
Age	: 28 years	**Date of discharge**	: 7 Jul. 2005
I.P. No.:	1026463		
Parity	: 0 + 0 Gravida 1	**L.M.P.**	: 25 Sept. 2004
		Expected date of delivery	: 2 Jul. 2005
		Gestation by Dates	: 32+ weeks

Presenting complaints

She had been admitted with a painful peripheral joints which had been present for the past 2 days prior to admission. She was previously well. She had no chest pains. She also reported dyspnea on exhaustion but no limb swelling or orthopnea.

Micturation habits were normal. She perceived normal fetal activity.

Past medical history

She was a known patient with sickle cell disease since childhood and was followed up in the medical outpatient clinic at KNH. As a child, she had been treated for a swelling in the abdomen. She was admitted once and transfused one unit of blood in childhood. She was on two paludrine tablets daily and one tablet of Folic acid daily.

Obstetric and gynaecologic history

She was a primigravida. Her menarche was at 16 years. Menses lasted 2 days and cycles were 21 days, regular. The flow was not heavy and she had no dysmenorrhoea. She had not used contraceptives.

Antenatal period

She could not remember when quickening was. She had attended antenatal clinic in a private clinic. An obstetric scan was done at 20 weeks which confirmed multiple gestation.

Family and social history

She was single and not employed and stayed in Kayole with her parents. There was no family history of sickle cell disease or any other chronic illness. She neither smoked cigarettes nor drunk alcohol.

Physical examination

She was in fair general condition and mildly wasted. She had no oedema or lymphadenopathy. She was, however, pale and had a tinge of jaundice. Her pulse was 95/minute regular and of good volume, respiratory rate 22/minute, blood pressure 110/70 mmHg and was afebrile with a temperature 36.8°C.

Cardiovascular system

There was no tachycardia. The JVP was not raised and the apex beat was not displaced. The first and second heart sounds were normal and there was a pansystolic murmur.

Respiratory and central nervous systems: were normal.

Abdominal examination

The abdomen was uniformly distended and moved with respiration. She had no surgical scars or therapeutic marks. She had a non-tender hepatomegaly of 4cm below the costal margin. The spleen was not palpable. The fundal height was 36 weeks gestation. Multiple fetal parts were palpated with presenting breech non engaged. One fetal heart rate was 140/minute and regular. No contractions were palpated. Vaginal examination was not done

Impression

An impression of sickle cell anaemia in pregnancy was made.

Investigations done

1. Hemogram: Hb 5.2 g/dl; WBC 20.3 x 10^9/1, neutrophils 52%, lymphocytes 40%, monocytes 8%; RBC 1.6 x 10^9/1; Platelets 247 x 10^9/1
   Peripheral blood film: Red blood cells: target cells, sickle cells, macrocytes, polychromasia
2. Blood group B Rhesus positive
3. VDRL negative
4. Urinalysis: albumin +, no blood or leukocytes
5. Haemoglobin electrophoresis showed Hb SF with S being 90% and F 10%
6. Obstetric scan 17.05.2000: Showed a diamniotic twin gestation. First twin in breech presentation with an estimated weight of 1500 gms, the second twin in cephalic

presentation with an estimated weight of 2100 gms. The placenta was fundo-anterior and not low-lying. The amount of liquor was adequate.

Management

She was started on Folic acid 5mg daily orally. She was managed for the painful crisis. The haematologist reviewed the patient and recommended blood transfusion of up to 6 units. She got transfused 5 units over her antenatal period. She remained stable while in the ward. She was planned for elective Caesarean section.

She went into preterm labour on 16.06.2005 and had an emergency Caesarean delivery as the presenting twin was non-vertex. The outcome was two live dichorionic diamniotic female infants who weighed 1700g and 2600 gms and both scored 7_1, 8_5, 10_{10} respectively. The first twin was admitted to newborn unit due to the low birth weight. The placentae were complete and healthy. The first twin died in NBU in the fourth postnatal day due to respiratory distress.

Postoperatively the patient was put on intravenous antibotics (crystaline penicillin, metronidazole and gentamicin). However, she got a burst abdomen (wound dehiscence with evisceration) on the fourth postoperative day. Secondary suturing was done and the wound healed well thereafter. She remained stable after delivery and was not transfused blood.

She remained stable while in the ward postnatally and was discharged home with her baby on 9.7.2005. She was advised to come for postnatal check-up after six weeks. She was advised on the contraceptive options she had. She was also referred back to the medical outpatient clinic (MOPC) for further follow-up of her medical condition.

Postnatal follow-up

She had no complaints. The baby was fine and breast feeding well. On examination she was in fair general condition but pale. The uterus was not palpable abdominally. She was counselled about family planning and opted for implants. She was also counselled on the fact that sickle cell disease is an inherited disease and her offspring to have the condition being passed on to them.

Discussion

This was a 28-year-old primigravida with sickle cell disease since childhood. She had a preterm delivery by Caesarean section to a live babies.

Sickle cell disease is a genetic disorder characterised by an abnormal haemoglobin, which causes red blood cells to become sickle shaped. Sickle cell haemoglobin results from a genetic substitution of valine for glutamic acid in the sixth position from the N-terminal end of the beta chain. The resulting haemoglobin is called haemoglobin S (Hb S) (1).

The sickle cell gene is inherited as autosomal recessive and can be passed to both sexes. Because there are two beta globin genes, the sickle cell disorders can be heterozygous or homozygous (2). Heterozygous patients have Hb AS and are said to have sickle cell trait. 25-45% of their haemoglobin is Hb S. Their clinical course is benign. Patients with sickle cell anaemia are homozygous for haemoglobin S (Hb SS) which represents 75-100% of their haemoglobin. Individuals are symptomatic and have reduced life expectancy (1). The patient had Hb SS.

Other variants of the sickle cell syndrome include sickle cell haemoglobin C disease (Hb SC) and sickle cell beta thalassaemia disease (Hb Sß$^{+}$ thal) and sickle cell ß^{0} thal. These later two variants are relatively uncommon and, therefore, most experience of sickle cell disease in pregnancy has been gained from mothers with Hb SS and Hb SC disease (2,3). Severity of complications in these patients is related to the Hb S concentration (2,4).

Sickle cell haemoglobinopathy is the most common inherited cause of haemolytic anaemia in the world. About 10% of black people in the United States of America carry the sickle cell trait and 1 in 500 has sickle cell anaemia (4). In Kenya sickle cell disease is found mainly among young children from western Kenya and coastal regions. The distribution follows closely that of *Plasmodium falciparum* malaria endemicity (5). Kibunguchy, in his study on the prevalence of anaemia in pregnancy at Coast Provincial General Hospital found that among the anaemic patients 10.6% had sickle cell trait and 0.7% had sickle cell disease (6). The patient presented was from Western Kenya and had Hb SS.

The abnormal amino acid (valine instead of glutamate) in the beta chain reduces both the stability and solubility of the haemoglobin. Low oxygen tension causes haemoglobin

S to polymerise, thus causing the globulin moieties to be arranged in parallel producing distorted red blood cells that have the characteristic sickle shape. There may also be changes in the cell membrane proteins and lipids causing it to become rigid with increased fragility and increasing sickle cell adherence. The deformed red cells in turn obstruct the microvasculature resulting in thrombosis. Usually, the thrombosis only causes pain but sometimes the thrombi damage organs like the kidneys, heart and lungs (1,4). These patients have chronic anaemia because of shortened red cell survival due to circulation trauma and intravascular haemolysis or phagocytosis by the reticuloendothelial cells in the liver and spleen. These patients are more prone to infections due to tissue damage by infarcts (especially the spleen) and lowered activity of the complement system (4). P.M. had anaemia but had no obvious focus of infection.

Infection, dehydration, excessive drop in temperature, extreme humidity, hypoxia and pregnancy precipitate vaso-occlusive episodes, also called sickle cell crises. Pain crisis involves the bones and joints. Aplastic crisis is characterised by rapidly developing anaemia due to cessation of red cell production by the bone marrow. Acute splenic sequestration crisis is associated with severe anaemia and hypovolaemic shock resulting from sudden massive red cell trapping within the splenic sinusoids (1,4). P.M. came in a painful crisis.

Diagnosis of sickle cell is made by laboratory investigations. The patients have severe anaemia. On peripheral blood film sickle cells and macrocytes are seen. Reticulocyte count is increased due to increased red cell production . The sodium metabisulphite test involves using one drop of fresh 2% reagent mixed with one drop of blood on a slide, covering with a cover slip and sealing the sides with molten wax to prevent drying up and exclude air. Sickling occurs in both sickle cell trait and sickle cell disease. Haemoglobin electrophoresis is done to confirm the major genotypes in the diagnosis of sickle cell disease (4). In the patient presented Hb electrophoresis done showed Hb SS and Hb SF: 90% of the haemoglobin was HbS and 10% was HbF.

Sickle cell disease does present with a wide range of clinical and hematological features which may be mild to severe disease. There is now increasing awareness of long term survivors as well as benign cases at younger age. The pattern of severity of disease are governed by genetic and environmental factors. Environmental factors include adequate nutrition, hygiene, malaria treatment and prevention, and improved health care.

Genetic factors include co-inheritance of ß-thalassaemia trait and glucose-6-phosphate dehydrogenase deficiency and heterocellular persistence of fetal haemoglobin. High levels of HbF is associated with mild clinical and haematological features (1,3,7). The patient presented had both HbF and HbS but, the HbF was not in high concentrations to prevent crisis occuring.

Sickle cell disease is a recognisable risk factor in pregnancy and this is principally due to anaemia (2,3). There is increased risk of infections. Folate deficiency occurs due to increased erythropoiesis. The frequency of sickle cell crises increases in pregnancy especially in the third trimester (3,8). There is also increased risk of PET, preterm delivery and Caesarean section deliver (2,3). Maternal mortality is due to septicaemia, thromboembolic episodes and cardiac failure following haemolytic crisis (2,8). Maternal mortality rate is 4-5% in the united states (2). Otieno in KNH reported a mortality rate of 36% (5). The patient presented had a painful crisis on admission but never developed another while in the ward. She was also noted to be anaemic with a Hb of 5.2g/dl. No apparent cause of the cause of the crisis was found.

There are multiple fetal complications which include high abortion rate, intrauterine growth retardation, high still birth rate and prematurity. The overall fetal wastage is about 30% (8). These are due to anaemia and the sickling process in the placenta causing placental insufficiency as a result of placental infarcts and increased blood viscosity (1,8). Perinatal mortality rate is 180/1000 births in the United Kingdom (9). Otieno in his study found a perinatal mortality rate of 450/1000 births (5). Perinatal mortality is due to low birth weight and intrapartum asphyxia secondary to chronic maternal anaemia and impaired placental function (8). This patient had a preterm delivery; one baby survived while the other died in the newborn unit due to prematurity.

Antenatal management should have a multidisplinary approach between the obstetrician, haematologist and later on a neonatologist as was the case in this patient. Folic acid should be given to aid haemopoiesis. Infections should be treated promptly and aggressively. These patients require weekly non-stress test together with serial ultrasound to monitor fetal growth and amniotic fluid from 32 weeks gestation. Vasoocclusive crises require admission to hospital. Management is by hydration, analgesia and warmth. Blood transfusion should be reserved for those patients with Hb less than 6 g/dl, severe vaso-

occlusive crisis, severe infection, acute splenic sequestration, and acute chest syndrome (pulmonary infection combined with pulmonary sickling). The aim of transfusion is to maintain the HbS concentration at <50% (4,9). P.M. was transfused severally but the HbS concentration was never done.

Prophylactic transfusion has been advocated in some centres. The aim is to reduce the level of HbS to under 50% and improve oxygen carrying capacity. In these studies prophylactic transfusion reduced the risk of sickling crises thus reducing the maternal morbidity (4). Prophylactic transfusion has, however, not improved the perinatal outcome when compared to those not transfused (4,9,10). Complications of transfusion include over-transfusion, which results in hyperviscosity leading to precipitated crisis. Other complications include allo-immunization and transmission of infections hepatitis B and transfusion reactions (4,10).

Uterine contractions during labour causes hypoxia in the placental bed. This can lead to a sickling crisis in the placental bed resulting in fetal demise. Bone marrow embolism is also common during labour. The patient should be adequately hydrated and a high oxygen tension maintained. Adequate pain relief must be given during labour. Caesarean section delivery should be done at the earliest for any obstetric indications (2). The preferred form of analgesia is spinal anaesthesia as there are less chances of iatrogenic hypoxia which can precipitate a crisis (2,4). P.M. was operated under general anaesthesia but pre-induction oxygenation was done and rapid induction was used.

Following delivery, there should be careful active management of the third stage of labour to minimise blood loss. The patient should be kept well hydrated. Prophylactic antibiotics and heparin are given (2). This patient was started on prophylactic antibiotics postoperatively and was observed in acute room for 24 hours.

Sickle cell disease is amongst the conditions which expose a woman to an increased risk of unwanted pregnancy (11). The current recommendations for contraceptive usage are;

(a) conditions for which there is no restriction for the use of the contraceptive method being Progestogen-only pill (POC), Depot medroxyprogesterone acetate, norethisterone enantate, Levonorgestrel implants (Norplant and Jadelle) and etonogestrel implants (Implanon) and, Levonorgestrel emitting intrauterine

contraceptive device. Among women with sickle cell disease, POC use did not have adverse effects on haematological parameters and, in some studies, was beneficial with respect to clinical symptoms (11).

(b) conditions where the advantages of using the method generally outweigh the theoretical or proven risks these being: Low-dose combined oral contraceptives, Combined injectable contraceptives, patch and ring, and Copper intrauterine devices (2,11). Sterilization (bilateral tubal ligation and vasectomy) should be considered in case of desired family size or if maternal complications are too life-threatening (11). This patient chose use Levonorgestrel implants.

As a preventive measure, it is important for women in stable relationships to have their partners tested for sickle cell disease or trait and appropriate genetic counselling given. Where there is risk of the fetus having sickle cell disease, prenatal diagnosis can be suggested and tests like chorionic villi sampling, amniocentesis or cordocentesis done (12).

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Case 12: Rhesus isoimmunization in pregnancy – poor outcome

Name	:	J.N.	**Date of admission**	:	15 Oct. 2003
Age	:	33 years	**Date of discharge**	:	22 Nov. 2003
I.P. no.	:	0921606			
Parity	:	4 + 0 Gravida 5	**L.M.P.**	:	25 Mar. 2003
			Expected date of delivery	:	1 Jan. 2004
			Gestation by Dates	:	30+ weeks

Presenting complaints

Loss of fetuses inutero and babies after birth

History presenting illness

This patient was admitted via antenatal clinic in Kenyatta. She had been referred from Murang'a District Hospital for follow-up and further management of her condition. She had been diagnosed to have Rhesus isoimmunization.

She was a Para 4 + 0 gravida 5 with no living child. She was therefore admitted due to a bad obstetric history and Rhesus isoimmunization. The fetal loses were:

Fetal activity was present and normal

Ante natal period

She started attending her antenatal care in Murang'a District Hospital then was referred to Kenyatta National Hospital.

Ante natal profiles done:

Blood Group : O Rhesus NEGATIVE

Indirect Coomb's Test : Positive

Antibody titres

Date	17 July	16 September	14 October	24 October
Titres	1:256	1:128	1:64	1:16

Serology for VDRL, Brucella Titres, Toxoplasmosis Titres, ELISA HIV all were negative

She was started on parenteral celestone twice weekly while in antenatal clinic. In the ward, as will be discussed later, she continued with a weekly dose.

Her blood pressures during the visits had remained consistently normal. There was no charted weight. She had until admission had an uneventful period.

Obstetric and Gynaecological History

She is a Para 4 + 0 Gravida 5

Pregnancy	Year	Gestation	Duration of labor	Delivery	Outcome	Comments
1	1998	term	8 hours	SVD 3 kg	live male infant	died in 2 days with jaundice
3	2001	term	10 hours	SVD ? wt	IUFD	
3	2001	6 months	?	SVD ? wt	preterm	died in 1 day. had jaundice
4	2003	7.5 months	?	SVD ? wt	IUFD	

Menarche was at 15 years. Her cycles were regular with the periods coming for 3-4 days and the cycle lasting 28 to 30 days. There was no dysmenorrhea and flow was normal. There was no history of having done a pap smear.

Past Medical and Surgical History

She had been transfused in Childhood in Murang'a. The reason for the transfusion was not known. She had otherwise not been admitted any other time. She was not on any medication chronically.

Family and social History

She was a divorced lady due to the pregnancy loses. Her first husband had blood O rhesus Positive. She did not know the current husbands blood group. She is a peasant farmer. She does not take alcohol or consume tobacco by smoking, chewing or sniffing. There was no known history of pregnancy loses in the family. Her spouse is a farmer. He does not smoke tobacco or take alcohol.

General examination

She was in good general condition. She was not pale, not jaundiced and did not have any edema, finger clubbing, or oral thrush. Her blood pressure was normal at 110/80 mmHg, pulse 84/min (normal), temperature of 36°C (afebrile), pulse of 92/min (normal).

Par abdomen

There was no area of tenderness. The liver and spleen were not palpable. The fundal height was corresponding to 30 weeks, lie longitudinal, Presentation Cephalic. Fetal heart rate was normal, heard and regular at 140 beats/min. The presenting part was 5/5 above the pelvic brim.

Other systemic examination was essentially normal.

Impression

An impression of Bad Obstetric History in a para 4 + O Secondary to Rh Isoimmunization at 31+ weeks gestation was made.

The patient was admitted for close monitoring and management in the ward. She was planned for:

1. Weekly Biophysical Profile and obstetric scans. Of particular interest was the organ development and to rule out Hydrops fetalis.
2. Bilirubin Spectrophotometry. This was not done as there was no laboratory in Nairobi that did this investigation
3. Fetal Kick Chart. This remained normal with adequate fetal kicks (10 in 10 hrs. or less) until the day of delivery as will be described below.
4. Paediatric review. The neonatologist advised us to try and push the gestation to as near term as possible otherwise the patient was to be delivered earliest at least 34 weeks gestation.
5. Serial indirect Coomb's were performed whose results are given above.
6. The patient was put on weekly injection celestone, hematinics

Investigations done and results

Serial Biophysical profiles and obstetric scans were performed.

1. 15 Oct. 2003 (gestation by dates 29 wks.): Singleton fetus, in breech presentation, with a regular heart rate of 167/min. The average gestation by femur length was 27 weeks 3 days. The placenta was not low lying. Resistive index was 0.692 which is normal for gestation. The average weight was 1205 grams.

The biophysical profile:

Fetal tone = 2	Fetal movements = 2
Amniotic fluid volume = 2	Breathing movements = 2
Total = 8 out of 8	

2. 26 Oct. 2003 (gestation by dates 30+ wks) Singleton fetus, in cephalic presentation, with a regular heart rate of 150/min. The average gestation by femur length, biparietal diameter and abdominal circumference was 29 weeks 4 days. The organs were normal by sonograghy. The placenta was not low lying. Resistive index was 0.647 which is normal for gestation. A conclusion of a viable fetus at 29 weeks 4 days with a normal resistive index was made. The average weight was 1370 grams.

   The biophysical profile:

Fetal tone = 2	Fetal movements = 2
Amniotic fluid volume = 2	Breathing movements = 2
(Non-stress test not done)	Total = 8 out of 8

Due to financial constrains the patient could not manage to do any other obstetric scan and biophysical profile.

17 Nov. 2003 at 33 wks 5 days gestation the patient was noted to have reduced fetal activity on the fetal kick chart; there were 4 kicks in 12 hours. On the 16th of the same month there were 8 kicks in 12 hours.

The patient was planned and taken for emergency Caesarean Section. The outcome was a live male infant, weighing 1800 grams Apgar score was 7_1, 8_5, 10_{10}. The baby was grossly normal or review. He was taken to New born unit due to prematurity and for possible exchange transfusion. Cord blood was obtained for blood group, bilirubin levels and full blood count. The placenta and membranes were delivered whole by controlled cord traction. The placenta, membranes and cord were all grossly normal on inspection. The patient was managed as described in the Caesarean section procedure.

The babies blood group was "A" Rhesus Positive. The baby died in new born unit with jaundice while awaiting exchange transfusion. The patient was lost to follow-up.

Discussion

The patient presented was a Para 4 + 0 gravida 5 with no living child. She had Rhesus isoimmunization and the outcome was poor.

The red blood cells have about 400 recognized antigenic factors of which the most common are ABO, Rhesus, Kell, Lutheran, Duffy, Kidd, P and MNs. Landsteiner and Weiner discovered the Rhesus factor in 1940 the presence of which makes an individual Rhesus positive and its absence Rhesus negative. The Rhesus antigens are inherited independently of all other blood group antigens. The Rhesus antigen status has five major antigenic loci namely C, D, E, c, e. The rhesus antigens C, E, c and e are considerably less immunogenic than the D antigen which is responsible for severe haemolytic disease of the newborn (1).

Considerable racial variations in the distribution of rhesus blood groups occurs. The Basque population have the highest incidence of Rhesus negativity (30-35%). Caucasians have an incidence of 15-16% and African Americans 7-8%. Asiatic groups and American Indians are all virtually Rhesus positive. The incidence among Mongoloid races is nil (2,3,4). The incidence in Nairobi is reported to be 5% of all mothers attending antenatal clinic (5). At Kenyatta National Hospital the incidence has been reported to be 4.1% (6).

Rhesus isoimmunization occurs when a rhesus-negative mother carries a rhesus positive pregnancy and also following a previous transfusion of rhesus positive blood (2,3). Under normal conditions, fetal cells do enter the maternal circulation but in small numbers which are destroyed by the maternal immune system before provoking an antibody reaction especially where the ABO blood group of the mother and fetus are incompatible (2).

Maternal immunization occur depending on a number of factors; First, it depends on her in-born ability to respond to the rhesus antigenic stimulus, about two-thirds of rhesus-negative women are responsive. Second, there is significant protection against immunization when there is also ABO-incompatibility between the fetal red cells and the mother – this reduces the incidence of rhesus isoimmunization to about one-tenth of that when they are ABO-compatible. Third, there is some variation in the strength of the rhesus antigenic stimulus, depending on the rhesus genotype of the fetal blood, e.g.

the Cde/cde genotype seems to be relatively 'strong'. Fourth, the volume of fetal blood entering the maternal circulation is very important, with 0.25ml representing a critical sensitizing volume and with the likelihood ad severity of sensitization increasing with greater volumes (4).

The initial (primary) response of a rhesus-negative individual to rhesus positive cells is formation of IgM antibodies which do not cross the placental barrier due to their large size; subsequently, IgG antibodies are formed in the secondary immunological response and these cross from mother to fetus and cause haemolysis of the fetal red blood cells leading to hydrops fetalis and kernicterus; depending on the extent of haemolysis (2,3). The patient presented was blood group "O" Rhesus "D" negative.

Usually the initial isoimmunization reaction is minimal but subsequent reactions tend to be stronger and more severely affected (2,3,7). It is known that fetomaternal haemorrhage goes on throughout pregnancy, but the amount of blood involved may be so small for any clinically significant complications. Risk factors that predispose to fetomaternal haemorrhage include amniocentesis, abortion, abdominal trauma, abruptio placenta, Caesarean section, antepartum haemorrhage and manual removal of placenta (2,3).

The overall risk of isoimmunization for a Rhesus positive ABO-Compatible infant with a Rhesus negative mother is 16% (1,2,3). The risk of Rhesus isoimmunization seems to be less than 2% after infusion of relatively small volume (<30mls) of Rhesus incompatible cells as would occur in multiple deliveries. With infusion of a large volume (>200ml) the risk is slightly greater than 8%. However at no volume, does there seem to be 100% immunization risk (1,2). About 2% of Rhesus negative women with ABO compatible blood with the fetus will be immunized by the time of delivery, another 7% will have anti-D antibody by 6 months postpartum, and the remaining 7% will be sensitized (1). The chances of a rhesus negative mother bearing a rhesus positive child is 75% (10) but even so, she has only 15-16% chance of being sensitized at the time of delivery of the first child (3,4). Of these, 1.5-2% of the isoimmunization will occur intrapartum, 7% within 6 months of delivery and the remaining 7% early in the next pregnancy most likely as a result of amnestic response (1,3).

The introduction and widespread use of anti-D gamma globulin has made the frequency of sensitized pregnancy to decline. This immunoglobulin prevents Rhesus isoimmunization by competitive inhibition. All the antigenic sites are covered or blocked from the lymphoid cells by the antibody. It may also interfere with the fetal red cells antigen processing by maternal macrophages, thus preventing the initiation of immune response (1,7).

The routine administration of anti-D after delivery or abortion prevents up to 95% of Rhesus sensitization. New cases continue to occur mainly due to sensitization during pregnancy, which renders prophylaxis after delivery useless. Giving anti-D antenatally will prevent these cases. Anti D given antenatally during the last trimester of the first pregnancy is highly protective not only for that pregnancy but for the next two pregnancies and possibly the third. There is 1-6% failure rate of prophylactic anti D when given after delivery compared to 0.1% when given antenatally (8,9).

The standard recommendation is to give anti D. This is a 7S immunoglobulin G usually given within 72 hours postpartum, but women at risk who have not received this regimen within 72 hours would still be treated. In fact some authors recommend treatment up to 28 days postpartum (3). This is given to the negative non-sensitized mothers to prevent the hazards of sensitization. It should be emphasized that when there is doubt about whether to give anit-D immunoglobulin then it is always safer to give it (1).

The most important part of the management of pregnancies at risk of rhesus isoimmunization is serial monitoring of maternal antibody levels throughout pregnancy (9). Indirect Coomb's test (ICT) should be done once during the first and second trimester then weekly thereafter until delivery. A titre that is less than 1:16 almost always means that the fetus will not die in utero from haemolytic disease (3). Higher titres indicate the possibility of severe haemolytic disease requiring exchange transfusion for hyperbilirubinaemia (9).

Where the mother is already sensitized, the maternal antibodies should be quantified. The mother is followed up by serial antibody titres until a critical level is reached (2,3) after which serial amniocentesis and ultrasound evaluations to rule out signs of hydrops fetalis are performed (1). Fetal haemolysis as may be shown by amniocentesis may

necessitate early delivery. Liley's charts are used to evaluate the severity of haemolysis and the intervention criteria for management is set up depending on the fetal condition.

Depending on the severity of the disease amniocentesis is repeated at 1-3 weekly intervals. Zone 1 generally indicates an unaffected fetus or one who will have a mild disease but a D negative fetus is also a possibility. In zone 2 the fetus is at moderate to severe risk of haemolytic disease. In the lower zone 2, the expected fetal Hb is 11.0–13.9 8.0–10.9 g/dl whereas in the upper zone 2 the expected Hb is 8.0–10.9 g/dl. In zone 3 the fetus is severely affected and death within 7–10 days may be expected unless intrauterine transfusion or delivery is effected. The expected Hb less than 8g/dl (1,2). Values in zone 1 and 2 require a repeat amniocentesis or fetal blood sampling to establish the trends of the two values and therefore used to estimate the severity of the hemolytic process and giving a rough guide on the actual condition of the fetus. Values zone 3 demand immediate delivery or fetal red blood cell transfusion (1). Unstable values, that is, values which tend to raise from zone 2 to 3 require the fetus to be delivered (1,2,9).

In our unit intrauterine transfusion is not done but wherever it is practised direct intravascular transfusion into the circulation (umbilical vein, hepatic vein or intracardiac) is done under ultrasound guidance. This has improved the outcome of fetuses with severe anaemia and hydrops fetalis. Due to lack of these facilities perinatal mortality and morbidity remains high among the isoimmunised women in our set up. In Kenyatta National Hospital a perinatal mortality of 600 per 1000 was reported (11).

Abnormal heart rate patterns have been described in severe rhesus isoimmunization. Sinusoidal and deceleration patterns have been associated with very low cord haemoglobin concentration at delivery and high perinatal mortality rates (9). The only direct method of assessing the severity of the disease is measuring the fetal haematocrit. This procedure is invasive and may result in bradycardia, abortion or preterm labour (9).

Other modes of treatment which have been tried unsuccessfully include plasmapheresis, immunosuppression with high dose of steroids, promethazine and D-positive erythrocyte membranes in enteric coated capsules to induce T- suppressor cell formation (3,9). For the patient who has been sensitized and has had repeated pregnancy losses artificial insemination by Rhesus D negative donor sperm can be done (9,10).

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Case 13: Frank breech in 2nd stage – assisted breech delivery

Name	B.O.	**Date of admission**	21 May 2005
Age	35 years	**Date of discharge**	22 May 2005
I.P. no.	1028133		
Parity	3 + 1 Gravida 5	**L.M.P.**	13 Aug. 2004
		Expected date of delivery	20 May 2005
		Gestation by Dates	40+ weeks

Presenting complaints

The patient presented with complaints of labour pains for 8 hours and draining of liquor for 2 hours prior to admission.

Obstetrics and gynaecology history

She was a para 3 + 1 gravida 5 with 3 living children. Her last delivery was in 2002 by SVD to a 3.7kg infant who was alive and well. She had a spontaneous complete abortion in 1999 at 2 months.

Her menarche was at 13 years, she had a regular cycle of 28 days and used to bleed for 4 days. She had used oral contraceptives until Feb. 2004.

Antenatal care

She attended at Kariobangi City Council clinic. The antenatal profile done showed blood group B Positive, serology for HIV and VDRL both negative and check Hb 13.0g/dl. She had received 2 injections of tetanus toxoid. Her baseline blood pressure was 120/80mmHg. Her antenatal period was otherwise uneventful. She had attended 4 visits.

Past medical history: This was non-significant.

Family and social history

She was a married and unemployed who never took alcohol nor smoked cigarettes. There was no history of twins or chronic illness in the family.

Systemic inquiry: This was non- revealing.

Physical examination

She was a young lady in fair general condition, not pale, not jaundiced, had no oedema and no lymphadenopathy. The vital signs were normal.

Respiratory, cardiovascular and central nervous systems: These were essentially normal.

Abdominal examination

The fundal height was term, longitudinal lie, with breech presentation. She was having 3 strong contractions in 10 minutes, each lasting over 40 seconds. Fetal heart was heard and regular at 138 beats per minute. Descent of the presenting part was 1/5. The estimated fetal weight was 2.8kg.

Vaginal examination

She had a normal external genitalia; the cervix was fully dilated. There was breech presenting, in sacral anterior position, thighs were flexed at hip joints and legs flexed at knee joints (complete breech); there was no cord felt. The presenting part was well applied to the cervix. She was draining meconium grade III liquor. The pelvis was clinically adequate.

Diagnosis

A diagnosis of à complete breech (flexed breech) in 2nd stage of labour with adequate pelvis.

Management

She was planned for assisted vaginal breech delivery. The patient was informed of the decision and course of management. She was explained to the need for cooperation during the procedure for a successful outcome. A paediatrician was called, and a team of the registrar and midwives experienced in breech deliveries was assembled.

She was then taken to the delivery room for assisted breech delivery. An intravenous line was put up with intravenous dextrose, and the patient explained to the procedure. In delivery room, a mediolateral episiotomy was made after the breech dilated the perineum ("crowning") the breech was then allowed to deliver sponteneously up to the level of umbilicus. During this time an assistant was regularly listening to the fetal heart every 5 to 10 minutes.

Once the breech was delivered beyond umbilicus, it was allowed to hang on its own weight and the mother encouraged to bear down with each contraction. Soon after the trunk upto the umbilicus is born the legs are delivered by pressure on the popliteal fossa

in a manner of abduction and flexion of the thigh. Once the axillar was visible, the corresponding shoulder and upper limb were delivered. When the nape of the neck was visualized, the Mariceu-Smellie-Viet Manoeuvre whereby the right hand of the operator was introduced and the index and middle fingers were placed on the malar eminences to aid in flextion of the fetal head while, continuous gentle traction was being exerted with the assistant keeping suprapubic pressure on the head to maintain flection. At this time the babies trunk was resting on the operators forearm and the Burns–Marshall manoeuvre was used by elevating the trunk of the fetus in a sweeping motion to allow delivery of the chin over the perineum, the head was expeditiously delivered. The outcome was a live male infant, with an Apgar score of 7/1; 9/5, and 10/10, the birth weight 2950gms. The placenta was delivered by controlled cord traction. Cervix and vagina were inspected and found to have no tears or lacerations. The episiotomy was repaired. The baby was examined and found to be grossly normal. Rooming-in was instituted and the baby started breast feeding well. The mother was observed in 4th stage for 4 hours and once her condition was satisfactory she was discharged to the postnatal ward.

Postpartum, the mother and baby were doing well and they were discharged home the following day. She was seen six weeks later in the postnatal clinic. She was well and opted to use combined oral contraceptive pills.

Discussion

The patient presented was a para 3+1 who presented with undiagnosed flexed breech presentation in 2nd stage of labour, she had a successful assisted vaginal breech delivery to a live male fetus who weighed 2950g and had an Apgar score of 7_1, 9_5 and 10_{10}. She had attended antenatal clinic in City council clinic but the breech presentation had not been diagnosed. During delivery the mother did not sustain any perineal tears or lacerations. The immediate postpartum period was otherwise unremarkable.

Breech presentation at term occurs in 3–4 % of all singleton pregnancies (1). The approach to delivery is controversial. In the management, a diligent search for any other complication, actual or anticipated, that might justify Caesarean delivery has become a feature of most philosophies for managing breech delivery. Caesarean delivery is commonly used in the following circumstances to deliver:

1. all but the extremely immature fetus whose potential for survival is negligible.
2. a large fetus (<3500 gm)
3. any degree of contraction or unfavourable shape of pelvis
4. an extended head
5. no labour, with maternal or fetal indications for delivery such as PET or PPROM
6. uterine dysfunction
7. all types of breech except frank
8. an apparently healthy but preterm fetus of 25 to 26 weeks or more, with the mother in either actual labour or in need of delivery
9. severe fetal growth restriction
10. previous perinatal death or children suffering from birth trauma, or previous bad obstetric history
11. a request for sterilization. (2)

The significance of persistent breech presentation is; an increased frequency of the following complications can be anticipated: (i) Perinatal morbidity and mortality from difficult delivery; (ii) Low birth weight from preterm delivery, growth restriction or both; (iii) prolapsed cord; (iv) placenta previa (v) fetal, neonatal, infant - anomalies; (vi) Uterine anomalies and tumours; (vii) Multiple fetuses, and (viii) Operative intervention especially Caesarean delivery (2). In considering vaginal delivery, in general, the prognosis of the fetus in breech presentation is considerably worse than when in cephalic presentation (2). Major contributing factors are; Preterm delivery (Low birth weight);

increased risk of congenital anomalies (6.3% vs 2.4%); birth trauma, for example, brain, spinal cord, liver, adrenal glands and spleen; delayed head delivery, for example, hypoxia, acidaemia, forced delivery leading to compression, traction or both (2).

The "Standard of Care" for delivery of term and preterm singleton breech presentations is use of a flexible approach that is individualized. Caesarean or vaginal delivery are both reasonable and acceptable in current Obstetric practice (2,3). B.O. presented in second stage of labour and a quick assessment revealed no immediate contraindication to vaginal breech delivery. Assessment of cervical dilatation and effacement and the station of the presenting part are essential in planning the route of delivery. If labour is too far advanced, there may not be sufficient time to obtain pelvimetry (3). This alone should not force the decision for Caesarean delivery. Satisfactory progress in labour is the best indicator of pelvic adequacy (2,3).

The fetal condition is crucial for determination of the mode of delivery and a quick thorough assessment is necessary to ensure, for example, that a Caesarean section is not done under emergency conditions for an anomalous infant with no chance of survival (2). Fetal monitoring is essential. The fetal heart rate should ideally be monitored by continuous electronic monitors with concurrent tocometer readings or at least every 5 minutes (4). Intravenous infusion and laboratory values are necessary during labour and delivery. Recruitment of nursing and medical personnel as additional help is required for managing labour and delivery of a breech. For labour, one-on-one nursing should be maintained due to risk of cord prolapse or compression, and all relevant physicians must be readily available should there be an emergency (2,3,4).

It is essential that the delivery team include: (a) an obstetrician skilled in the art of breech delivery, (b) an associate to assist with the delivery (c) an anaesthesiologist who can assure adequate anaesthesia when needed, and (d) an individual trained to resuscitate the infant, including tracheal intubation (2,3,4). The Obstetrician should consider and abide by the preferences of the informed patients (5). Delivery is easier and in turn, perinatal morbidity and mortality is probably lower when the breech of the fetus is allowed to deliver spontaneously to the umbilicus. If a non-reassuring fetal heart rate pattern develops before this time a decision to perform manual breech extraction or to default to Caesarean section (2,5). The recommended mode of delivery is assisted breech delivery. This was the mode of delivery done on B.O.

For a favourable outcome with any breech delivery, at the very minimum, the birth canal must be sufficiently large to allow passage of the fetus without trauma. The cervix must be fully dilated and if not, then a Caesarean delivery nearly is the more appropriate method of delivery when suspected fetal compromise develops (3,4).

Criteria for vaginal delivery: The criteria used was developed by Zatuchni and Andros in 1965. as shown in the table below:

	Score given		
	0	**1**	**2**
Parity	0	1	2
Gestational age (wks)	39+	37–39	<37
Estimated fetal weight (gms)	>3600	3000–3600	<3000
Previous breech delivery	0	1	2
Dilatation	2	3	4
Station	-3	-2	-1

If the score is 0–4, then Caesarean section is recommended.

Other factors considered broadly while considering vaginal breech delivery consists of (i) Frank (extended) breech presentation, (ii) gestation age of 34 weeks' or more, (iii) estimated fetal weight of 2000–3500 gms, (iv) flexed fetal head, (v) adequate maternal pelvis as determined by x-ray pelvimetry (pelvic inlet with transverse diameter of 11.5cm and A-P diameter of 10.5cm, midpelvis with transverse diameter of 10cm and A-P diameter of 11.5cm), (vi) previable fetus (gestation age <21 weeks and weight <750g), (vii) documented lethal fetal anomalies, (viii) presentation of mother in advanced labour with no fetal or maternal distress, even if Caesarean section was originally planned (a carefully performed, controlled vaginal delivery is safer in such cases than a hastily executed Caesarean section), (ix) some carefully selected cases of complete or footling breech (continuous electronic monitoring must be done to detect variable fetal heart rate deccelerations due to umbilical cord prolapse, if this occurs immediate Caesarean section is indicated (2,5). In the scoring system, the greatest indicatior to alllow for vaginal delivery is cervical dilatation. B.O. presented in advanced labour and there was no contraindication to vaginal delivery.

Management during labour: As delivery of the breech occurs, increasingly larger diameters (bitrochanteric, bisacromial, biparietal) of the body enter the pelvis, whereas in cephalic presentation the largest diameter (biparietal diameter) enters the pelvis first, this poses unique complications (5). Three general methods of breech delivery through the vagina are used. These are (i) spontaneous breech delivery, (ii) partial breech extraction and (iii) total breech extraction.

In spontaneous breech delivery the infant is expelled entirely spontaneously without any traction or manipulation other than support of the infant. This is no longer recommended as a form of delivery for viable infants. In partial breech extraction (assisted breech extraction) is employed when the operator discerns that spontaneous delivery will not occur or that expeditious delivery is indicated for fetal or maternal reasons. The body is allowed to deliver spontaneously up to the level of the umbilicus. The operator then assists in delivery of the shoulders, arms and head. An assistant supports the body while the operator rotates the spine as is necessary until it rests directly under the pubic symphysis. The operator locates the right humerus and applies gentle downward pressure until the right arm is delivered. The body is rotated until the left shoulder is beneath the symphysis, and the left arm is delivered in a like fashion. Rotating the spine again to a position below the pubic symphysis, the operator begins to deliver the head. As the body is lifted gently upward and as fundal pressure is applied from above to keep the head in a flexed position, the head may be delivered spontaneously over the perineum. The operator may elect to manually assist in delivery of the head by performing the Maricean-Smellie-Viet Manoeuvre. In this procedure, the index and middle fingers of one of the operator's hands are applied over the maxilla as the body rests on the palm and forearm of the operator. Two fingers of the operator's other hand are applied on either side of the neck with gentle downward traction. At the same time, the body is elevated towards the pubic symphysis, allowing for controlled delivery of the mouth, nose and brow over the perineum. Delivery of the head may also be conducted by the Burns–Marshall manoeuvre (6), but more safely with obstetric forceps (3,4,5).

The alternative technique for breech delivery is the Bracht Manoeuvre. The breech is allowed to deliver spontaneously to the umbilicus. The fetal body then is held, but not pressed against the maternal symphysis. This force is meant to be the equivalent of gravity. The suspension of the fetus in this position, coupled with the effects of

uterine contractors and moderate suprapubic pressure by an assistant, often results in spontaneous delivery (6).

In the event of the head entering the pelvic cavity with the occiput posterior, delivery may be affected by using the Prague grip in reverse, the direction of shoulder traction being downwards and backwards. In occiput posterior one hand of the operator supports the shoulders from below while the other hand gently elevates the body upward toward the maternal abdomen. This flexes the head within the birth canal and results in delivery of the occiput over the perineum (3,5,6).

Occasionally during partial breech extraction and more often during total breech extraction, excessive downward traction on the body to effect delivery of the scapulas results in a single or double nuchal arm. Because the body descends too rapidly through the birth canal, one or both arms are extended upwards from their normal flexed position against the chest and become lodged behind the neck. A single or bilateral nuchal arm is suspected when delivery of the shoulder is difficult or the shoulder blade is rotated away from the spine. To dislodge an impacted nuchal arm, the operator rotates the body in a half circle to bring the elbow towards the face. The humerus can then be readily identified by palpation and delivered as previously described. For bilateral nuchal arms, the fetus is rotated counter clockwise to deliver the right arm and often clockwise to dislodge and deliver the left arm. If rotation does not dislodge the arm, the operator must insert a finger into the maternal pelvis, identify the fetal humerus, and possibly extract the arm. Fractures of the humerus or clavicle may result as a complication (2,6).

In total breech extraction (which has a higher perinatal morbidity), the Obstetrician delivers the infant with no assistance from the mother. The process has been used to expedite delivery in cases of fetal distress or prolapsed cord, and occasionally when progress ceases in second stage, or for delivering the second twin. A footling presentation is easier to extract than a breech with extended legs, in which the foot must first be delivered by inserting a hand into the uterus and using Pinard's manoeuvre, to convert it into a footling breech (3,5,6). Pinard's manoeuvre is used sometimes in a case of Frank breech presentation to deliver a foot into the vagina. Two fingers are carried up against one extremity to the knee then push it away from the midline. Spontaneous flexion of the knee follows, and the foot of the fetus is felt to impinging upon the back of the hand. The fetal foot then may be grasped and brought down (6).

If the head is entrapped in a premature delivery, a hysterostomatotomy (Duhrssen's incision) must be considered to preserve fetal life. Incisions are made in the posterior cervix at 6 o'clock to loosen the entrapped head. Occasionally additional incisions are necessary at 2 and 10 o'clock. This can be dangerous if incision extends to the lower segment causing severe haemorrhage (3,5,6). As so often happens other methods of delivery are Zavanelli, Woods cockscrew manouvers.

Some of the complications associated with vaginal breech delivery include: fetal asphyxia, neurological damage, infection and trauma. Maternal complications are; infection, uterine rupture, and haemorrhage (3,4,5,6). In general the Caesarean section delivery has increased steadily from about 30% in the 1970s to 75% in the 1990s. This can be attributed to the poorer outcome with a three to fourfold significantly higher perinatal mortality rate and neonatal morbidity due to trauma in planned vaginally delivered infants compared to those undergoing elective Caesarean section delivery (7).

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Case 14: Fulminant genital warts in pregnancy – Caesarean section delivery

Name	D.A.	**Date of admission**	23 Oct. 2003
Age	20 years	**Date of discharge**	3 Nov. 2003
I.P. No.:	0928064		
Parity	0 + 0 Gravida 1	**L.M.P.**	30 Jan. 2003
		Expected date of delivery	6 Nov. 2003
		Gestation by Dates	38 weeks

Presenting complaints

The patient presented with complaints of drainage of liquor vaginally and associated labour like pains for 3 hours prior to admission.

History of presenting complaints

She was well then she suddenly noticed drainage of clear fluid from the vagina and followed by labour like pains for 3 hours before she reached hospital. The drainage of fluid was significant flowing until her legs There was accompanying lower abdominal and back pain. Bowel and micturation habits were normal

Obstetrics and gynaecology history

She had menarche at 14 years and her cycle interval was 28 days and she used to bleed for 3 days. She had never used any contraceptives. She was a Para 0+0 gravida 1.

Antenatal care

She had attended only once at Kayole nursing home at 36 weeks. Antenatal profile had not been performed. She had received one tetanus toxoid injection. The antenatal period was otherwise uneventful.

Past medical history

She had never been admitted to hospital before and had no history of genital tract infections or sexually transmitted infections.

Family and social history

She was single woman who had been working as a house girl in Nairobi, but moved to live in her rural home in Kangundo with her parents after she became pregnant. She did not take alcohol nor smoke cigarettes. There was no family history of twins or chronic illnesses such as diabetes, tuberculosis or hypertension.

Systemic inquiry

She had been having genital itchiness with bleeding on itching and had noted the genital warts 3 months before conception, but they were small so she never sought any treatment. She otherwise had been of good health.

Physical examination

She was a young lady who was in fair general condition looking, not pale, had cervical lymphadenopathy, no oral thrush, no jaundice, and no edema. Her vital signs were a blood pressure of 110/60 mmHg, pulse 106/beats per minute, respiratory rate 20/minute, Temperature 37.2°C.

Respiratory, cardiovascular and central nervous systems were essentially normal.

Abdominal examination

The fundal height was term with a longitudinal lie and cephalic presentation. The fetal heart was heard and regular at 136 beats per minute. There were no contractions palpated. The presenting part was 5/5 up.

Pelvic examination

On inspection in lithotomy position, there were massive fulminant fungating cauliflower-like genital lesions covering and distorting the external genitalia and entire perineal region and medial thigh. There lesions were not however clear fluid was seen to be flowing from the introitus region. The vaginal orifice was stenotic and it was not possible to do a speculum examination to determine the source of the fluid.

Diagnosis

A diagnosis of fulminant genital warts in pregnancy with prelabor rupture of membranes.

Management

She was planned for and emergency abdominal delivery. A rapid HIV test done was positive for HIV antibodies.

Preparations

Informed consent was obtained; a sample of blood was taken for group and cross-match. An intravenous (IV) line was fixed and IV fluids Ringer's lactate and 10% dextrose

was started. Premedication with intramuscular (IM) atropine 0.6mg half an hour before theatre and nevirapine 200 mg orally was given and she was taken to theatre.

In theatre

A thorough pelvic examination was done under general anaesthesia. The warts were found to involve most of the vaginal cavity. A decision was made to perform an emergency Caesarean section.

A lower uterine segment Caesarean section was performed and a live female infant was delivered. She weighed 2650 grams and Apgar score was 7/1, 9/5 and 10/10. The placenta was fundo-posterior.

The uterus was cleaned and repaired in layers and hemostasis achieved. Both ovaries and tubes were inspected and found to be grossly normal and healthy. The abdomen was closed in layers. Gentle vulvo-vaginal toilet was done. There was minimal bleeding from the warts. The vulval area was cleaned with betadine and sofratulle dressing applied on the lesions. Estimated blood loss was 700 mls. Post-operatively a catheter was left in-situ to assist bladder drainage for 7 days. She was started on broad-spectrum antibiotics, crystalline penicillin, gentamicin and metronidazole. Post operatively the mother and baby recovered well. The results of investigations done on 3rd post operative day were ass follows: hemogram Hb 10g/dl, WBC 11 x 10^9/l, platelets 230 x 10^9/l, VDRL was Negative, blood group was O positive. She was informed of the need for close follow up and various options for infant feeding in regards to her HIV status. She opted to breast feed.

The catheter was removed on the 7th postoperative day, she able to pass urine comfortably. A review on 10th postoperative day, she was stable, the warts were regressing, and the baby was okay. The wound had healed well and the mother and baby were discharged home. She was given an appointment to come for postnatal check-up and treatment of warts after 6 weeks. However, she did not honour her appointment.

Discussion

The patient presented was a single 20-year-old seropositive Para 0+0 who presented with massive genital warts covering the entire vulva and vagina and PROM. She had attended antenatal clinic only once and antenatal profile had not been done. Vaginal examination was impossible due to the warts, and she was taken to theatre for examination under anaesthesia. An emergency Caesarean section was performed and a live female infant who weighed 2650g and scored well was delivered. She opted to breast feed. Her recovery was otherwise unremarkable.

Condylomata accuminata (sexually transmitted genital warts) are caused by a virus of the papovavirus group — Human Papilloma virus (HPV), types 6 and 11 but may be caused by 16, 18, 30s group, 40s group, 50s group and 60s group (1). Papillary growths, small at first tend to coalesce and form large cauliflower like masses that proliferate profusely during pregnancy. The virus also can cause laryngeal papillomatosis in children, and evidence is consistent that types 6 and 11 in some cases may be transmitted by aspiration of infected material at delivery. Some studies have shown that the virus may be transmitted transplacentally (2). The rate of perinatal viral transmission is unknown and ranges between 5%–30 depending on the type of virus, but all symptomatic disease in children is rare (3).

For unknown reasons, genital warts frequently increase in number and size during pregnancy, sometimes filling the vagina or covering the perineum, making it difficult to perform vaginal delivery or episiotomy. In women without grossly visible lesions a study found an association of papillomavirus infection and episiotomy breakdown (4). Certainly the mucorrhoea throughout pregnancy offers ideal moist conditions for viral growth. Accelerated viral replication with advancing pregnancy has been hypothesized to explain the growth of perianal lesions, progression of some to neoplasm, and increased detection of viral DNA from the cervix in some studies of pregnant women (5,6).

Vulval lesions often improve rapidly or disappear postpartum, possibly due to loss of either vascularity, excessive moisture, or the alleged immunosuppression of pregnancy. Other causes of immunosuppression also lead to rapid increase in the size of warts. The patient presented was found to be HIV positive and this could have contributed to the immunosuppression leading to the rapid growth of the warts (3).

Treatment in pregnancy may be unsatisfactory, but it is usual for the lesions to clear rapidly following delivery. During pregnancy washing of external genitalia, plus cleansing of vagina by gentle douching, followed by thorough drying of external genitalia performed at least once daily, may inhibit proliferation of warts as well as minimize discomfort. Because lesions commonly regress after delivery, it is not always necessary to try to eradicate them during pregnancy. Therapy is directed towards minimizing toxicity to the mother and fetus and debulking the genital warts in the late second or third trimester so that recurrence is less likely prior to delivery.

There are several agents available for treatment outside pregnancy but their safety is a concern in pregnancy. Treatment failure is common. Trichloroacetic acid 80%–90% both in 70% ethanol applied topically 3 times weekly or once a week is an effective mode of therapy (3). Cryotherapy and laser ablation of visible lesions are preferred modes of therapy in pregnancy but combination with trichloroacetic acid gives better results (7).

Podophyllin resin, 5% 5-fluorouracil cream, imiquimod cream and interferon therapy should not be used in pregnancy due to concerns about maternal and fetal toxicity (7)

Occasionally, the warts acquire enormous size and these may necessitate Caesarean section especially if the birth canal is blocked or stenotic. In events where the warts bleed excessively or may bleed during vaginal delivery, a Caesarean section may be necessary (3). This was the case in the patient presented. Enthusiasm has been expressed for use of the carbon-dioxide laser during pregnancy to remove large lesions under anaesthesia (9). This can be done several weeks before delivery. Electrocautary, cryotherapy or laser ablation can also be used.

An indeterminate, but probably small number of infants and children born to woman with genital papillomavirus lesions will become infected and develop laryngeal papillomatosis. The question has been posed that Caesarean section might avoid infection of the fetus-infant, but this is not recommended currently (8).

After delivery the treatment method will be determined by whether the mother breast feeding or not. Before treatment is started, the entire lower genital tract should be examined with a coloscope and a cytopatology smear taken from the cervix. Other STIs must also be ruled out due to the coexistance of these diseases. During treatment, the patient should keep the area as clean as possible and abstain from sexual intercourse or use barrier methods (10).

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