CASE RECORDS AND DISSERTATIONS

IN

OBSTETRICS AND GYNAECOLOGY

SUBMITTED BY

DR JULIUS ONDIGO

FOR THE DEGREE OF MASTER OF MEDICINE

IN

OBSTETRICS AND GYNAECOLOGY

OF

THE UNIVERSITY OF NAIROBI

SUPERVISOR'S DECLARATION

We certify that these dissertations and case commentaries have been submitted for examination to the University of Nairobi by **Dr Julius Ochieng Ondigo** with our approval as university supervisors.

SUPERVISORS:

Dr. James Machoki M'Imunya

Deputy Director UNITID/ Consultant KNH

University of Nairobi, Kenya.

Signature.....

Dr. John Kinuthia MBChB, MMed (Obs/Gyn), MPH

Honorary Lecturer

University of Nairobi, Kenya.

Signature.....

SUBMITTED

TO THE UNIVERSITY OF NAIROBI IN PART FULFILMENT FOR THE AWARD OF MASTER OF MEDICINE IN OBSTERICS AND GYNAECOLOGY

PURPOSE: DISSERTATION FOR PART FULFILLMENT FOR THE DEGREE OF MASTER OF MEDICINE IN OBSTETRICS AND GYNAECOLOGY, UNIVERSITY OF NAIROBI.

INVESTIGATOR:

DR DR JULIUS OCHIENG ONDIGO MB.ChB.

Postgraduate student, Department of Obstetrics and Gynaecology,

University of Nairobi.

SUPERVISORS:

1. Dr. James Machoki M'Imunya

Deputy Director UNITID/ Consultant KNH

University of Nairobi, Kenya.

2. Dr. John Kinuthia MBChB, MMed (Obs/Gyn), MPH

Honorary Lecturer

University of Nairobi, Kenya.

These dissertations and case commentaries have been submitted by me for examination to the University of Nairobi in part fulfillment for the degree of Master of Medicine in Obstetrics and Gynecology.

Dr Julius Ochieng Ondigo Mb ChB

Postgraduate student, Department of Obstetrics and Gynaecology,

University of Nairobi.

Signature.....Ochieng 5/1/2012.....

Supervisors' declaration

We certify that these dissertations and case commentaries have been submitted for examination to the University of Nairobi by Dr Julius Ondigo with our approval as university supervisors

Supervisors

1. **Dr James Machoki M'Imunya**

   Deputy Director UNITID/Consultant KNH

   University of Nairobi, Kenya

   Signature.....

2. **Dr John Kinuthia MBChB MMed (Obs/Gyn), MPH**

   Honorary lecturer University of Nairobi, Kenya

   Signature.....

CERTIFICATE OF AUTHENTICITY

This is to certify that this dissertation is the original work of Dr Julius Ochieng Ondigo, MMED student registration number H58/7410/2002 in Obstetrics and Gynaecology department, College of Health Sciences, University of Nairobi, under the guidance of Dr James Machoki M'Imunya and Dr John Kinuthia. It has not been presented in any university for the award of a degree

Signed.....

Date.....26/01/2012.....

Prof Koigi Kamau

Associate Professor of Obstetrics and Gynaecology

Consultant Obstetrics and Gynaecology

Chairman department of Obstetrics and Gynaecology

University of Nairobi.

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Reasons for failed induction of labour with PGE_2 and $PGF_{2\alpha}$ from 28 weeks gestation among subjects who are induced at Kenyatta National Hospital maternity unit.

Appendix

- KNH/UON-ERC approval letters

BARTHOLIN'S ABSCESS – MARSUPIALIZATION

NAME: CN. **AGE:** 28 YRS

IP.NO. 1003589

DOA: 21/01/05. **DOD:** 22/01/05 28 years

Left vulval swelling for 5 days

HISTORY OF PRESENTING ILLNESS

CN was admitted and gave history of having had an incomplete abortion one week earlier at a gestation by dates of 8 weeks. Five days prior to admission the patient developed a progressive and painful left sided vulval swelling with excessive discomfort on walking. There was occasional fever but no urinary symptoms. There was minimal lochia loss, which did not have an offensive smell. She denied having any diarrhoea, nausea or vomiting.

PAST MEDICAL HISTORY Was not significant

OBS/GYN HISTORY

She had attained menarche at an age of 16 years, her menstrual cycle used to last for 28 days with a flow of 3 days and was regular. There was no history of contraceptive use.

FAMILY SOCIAL HISTORY

She is a housewife while her husband is a businessman. There was no history of chronic illness in the family. On examination she was a young lady in fair general condition, not pale, jaundiced or cyanosed.

Vital signs: BP 110/60 mmhg, PR 92 /min, PR 18/min, Temp.37.4 °C which were normal.

On vaginal examination she had a large left labia majora swelling involving the posterior third of the vulva. It was tender, non fluctuant, about 4cm in diameter. On digital exam the cervical os was central, closed, both adnexa and POD were free and the uterus was well involuted. There was no discharge on the examining finger.

An impression of Left Bartholins abscess was made.

Laboratory investigations done were PCV which was 32%

Management

The patient gave a written consent for marsupialization and was prepared for theatre.

Marsupialization

In theatre she was placed in operating table and put under general anaesthesia. She was placed in lithotomy position, vulvo-vaginal toilet done and draped. The bladder was aseptically catheterized and drained of 150 mls of clear urine.

Examination under anaesthesia confirmed the earlier findings. The vagina was packed, a 4cm vertical incision was made over the mucocutaneous region outside the hymeneal ring. The abscess bed was explored using curved forceps. About 10mls of yellowish whitish foul smelling pus was expressed and some submitted for culture and sensitivity. The cavity was cleaned using hibitane solution, its edges everted and sutured to the vulval skin with interrupted chromic vicryl 2/0. A betadine soaked pack was placed over the wound and anaesthesia reversed.

Post-operative care: She was observed half hourly until fully awake then 4 hourly. She was then started on oral Augmentin 625mg 12 hourly for five days, Metronidazole 400mg and Brufen 400mg 8 hourly for five days. On the first post-operative day she was stable, the wound dressing was removed and the wound was clean. She was started on saline sitz baths. On the second day she was in good condition, she was discharged home on treatment to come for review in two weeks.

Follow up: She was reviewed two weeks later and had no complains. The wound was well healed and her pus culture showed E. coli sensitive to Augmentin among other antibiotics. She was discharged from the clinic.

DISCUSSION.

The patient presented was a 28 year patient admitted with Bartholin's abscess. She underwent marsupialization under general anaesthesia and recovered uneventfully.

Bartholin's glands are a pair of racemose glands situated deep to the bulbocavernous and transverse perineal muscle on either side of the introitus. They are also referred to as the greater vestibular glands and are homologous to the Cowper's glands in the male. Bartholin's glands are drained by a duct 1-1.5cm long that runs downwards and inwards to open at 5 or 7 o'clock position external to the hymen. They are lined by cuboidal or columnar epithelium and secrete mucoid like alkaline fluids during sexual arousal. These glands are about 0.5-1cm in diameter, although it is usually impalpable unless hardened or enlarged by disease. They secrete a lubricant during sexual intercourse. The incidence of Bartholin's abscess at Kenyatta National Hospital is 1.7-1.9 %. [2]

Bartholin's abscesses usually cause chronic inflammation of the gland and duct leading to obstruction of the duct or mucous insipation. Bartholin's abscess may follow primary infection of the gland or follow duct obstruction as a result of mediolateral episiotomy, posterior colporrhaphy, insipated mucus or congenital narrowing [4] It is usually associated with Neisseria, Gonorrhoea. and Chlamydia trachomatis. Other organisms implicated include E.coli, Staph aureas, Proteus mirabilis, Candida albicans and Trichomonas vaginalis [5]. The patient presented here had E. coli isolated from pus swab culture.

Like all abscesses, the management of Bartholin's abscess is mainly surgical drainage. Treatment of infection by antibiotics alone relieves the infection but there is a great tendency for recurrence. Marsupialization is the treatment of choice in contrast to incision and drainage or excision of the gland even in cases of a Bartholin's cyst. Marsupialization is advantageous in that:-

It is a simple procedure requiring only few minutes of operation while excision is tedious and difficult. Haemorrhage is minimal and is easily controlled while in excision haematoma formation is always a possibility due to the heavy vascularity of the glands bed. Post-operative hospital stay is minimized to 24 hours maximum in favourable cases. Some glandular functional activity may be regenerated thereby offering lubrication to the vagina. Gland excision leaves the patient with a dry vagina leading to functional dyspareunia

REFERENCES

1. Krants K.E. Anatomy of the female reproductive system. Current Obstetric and Gynaecologic diagnosis and treatment. 9h Edition, Ch 2 pg21 Appleton and Lange; 2

2. Mumia J.A. Bartholin's abscess. MMed Thesis University of Nairobi 1981

3. .Ndede T. A six month survey of Bartholin's abscess at Kenyatta National Hospital. MMed Thesis University of Nairobi 1990

4. Markusen T.E, Barclay D.L. Benign disorders of the vulva and vagina. Current obstetric and Gynaecologic diagnosis and treatment. 9th Edition, Ch 34 pg 672Appleton and Lange; 2003

5. Anderson P.G., Christen S. et al. Treatment of Bartholin's abscess. Marsupialization versus incision, drainage and suture under antibiotics cover. A randomized study with 9 months follow up. Acta. Obset. Gynaecol. Scand: 79:59; 1992

6. Tindall V.R. Bartholin cyst . Jeffcoates Principles of Gynaecology. 5th Edition, Ch.2 pg 18 Butterworth 1987

GYNAECOLOGY CASE 2

RUPTURED TUBAL ECTOPIC PREGNANCY- TOTAL SALPINGECTOMY

Name: SW Age 30yerars DOA 23/10/06 DOD 26/10/06 IP 1123373

PRESENTING COMPLAINTS

R.N. was well till the day of admission when she developed severe acute lower abdominal pain and per vaginal bleeding.

HISTORY OF PRESENTING ILLNESS

She was well till the night before the day of admission when she developed colicky lower abdominal pain which progressively became severe and localized to the left iliac fossa. There was associated slight per vaginal bleeding since the morning of admission. Her last menstrual period was on 22/05/04. The period of amenorrhoea was therefore about 8 weeks. There was no associated fever, nausea, vomiting or diarrhoea. She did not give a history of dysuria or frequency. Subsequently she had developed dizziness and was unable to walk. She was seen in a private clinic where she was told that she had an ectopic pregnancy.

OBSTETRICS AND GYNAECOLOGY HISTORY

She was a Para 4+0. Her last delivery was 4 years earlier, by spontaneous vertex delivery to a live female infant who was alive and well. Her other deliveries were all SVD, and all the children were alive and well. She attained her menarche at 15 years and the cycle length was on average 26 days. The flow was usually 3-4 days.

FAMILY AND SOCIAL HISTORY

She was a casual worker in the EPZ at Athi River. She lived together with her husband, a mechanic in Kitengela. She did not smoke cigarettes or drink alcohol. There was no history of chronic ailments in the family.

PAST MEDICAL AND SURGICAL HISTORY

She had never been admitted to hospital for non-obstetric reasons. She had never been operated before.

PHYSICAL EXAMINATION

On examination she was in fair general condition. She was pale, but did not have jaundice, edema or lymphadenopathy. Her vital signs were normal.

RESPIRATORY SYSTEM: Lung fields were clear.

CARDIOVASCULA SYSTEM AND CENTRAL NERVOUS SYSTEMS

Where essentially normal.

ABDOMINAL EXAMINATION

The abdomen was not distended. However on light palpation there was tenderness in the lower abdomen, especially in the left iliac fossa. There was rebound tenderness and muscle guarding making deep palpation difficult. Parecentesis was positive, with non-clotting blood. Bowel sounds were normal.

PELVIC EXAMINATION

The cervix was long, closed, and posterior with blood on the examining finger. Cervical excitation test was positive to the left, and the pouch of Douglas was full.

TENTATIVE DIAGNOSIS

An impression of ruptured left tubal pregnancy was made. Blood was drawn for grouping and cross-match, hemoglobin level, urea and electrolytes.

MANAGEMENT

The patient was informed of the diagnosis and mode of treatment was emergency laparatomy. Informed consent was obtained. She was premedicated with I.M Atropine 0.6mg.

PROCEDURE IN THEATRE

The patient was placed on the theatre table and put under general anesthesia. She was re-positioned to the semi-lithotomy position, vulvovaginal toilet done and bladder catheterized aseptically. Pelvic examination confirmed the pre-operative findings. The patient was re-positioned to the supine position, the anterior abdominal wall cleaned and draped. The abdomen was opened through a pfannenstiel incision. On entering the abdominal cavity, massive hemoperitoneum was found. The intestines were packed away using moist abdominal packs, and a self-retaining retractor introduced to expose the pelvic organs. The left tube was

found to be bleeding, hence was brought out of the abdominal wound. A left ampullary ectopic gestation was identified. The tube was clamped to stop bleeding. The hemoperitoneum was sucked out of the abdominal cavity and blood clots removed. The right tube and ovary were inspected and found to be normal. The left ovary was also normal. The left tube was reclamped near the cornu, with further clamps paced across the broad ligament and ligated. The abdominal cavity was irrigated with warm saline and dried using suction. The specimen was taken for histology. The appendix was found to be normal.

Post operatively she did well, and a check Hb done on the 3rd post-operative day revealed Hb level of 8.1gldl. She was not symptomatic. She was allowed home on hematinics.

DISCUSSION

Presented is a 41-year-old peri-menopausal lady who had a left ampullary ectopic pregnancy while using a copper intra-uterine contraceptive device (IUCD). She presented with acute tubal rapture and was managed by total left salpingectomy.

Ectopic pregnancy is defined as implantation of the fertilized ovum or blastocyst anywhere other than in a normal uterine cavity. This includes tubal pregnancies in the ampullary, infundibular, isthmic and interstitial portions of the oviduct as well as nontubal pregnancies involving the ovary; In the abdominal cavity, including peritoneal, omental, hepatic and splenic pregnancies; in the rudimentary horn of the uterus; in the cervix; and in foci of adenomyosis or uterine surgical defects. 95-97% of ectopic pregnancies are tubal. The ampulla is the most common site of implantation, followed by the isthmus the fimbria and the interstitial area (1).

In thiscase the pregnancy was in the left fallopian tube, ampullary portion. This is the most common site of ectopic pregnancy.

A comprehensive review of the incidence of ectopic pregnancy collected by the centers for Disease Control and Prevention, show a significant increase in the number of ectopic pregnancies in the United States during the past 20 years. In 1989, the latest year for which statistics were published, there were an estimated 88,400 ectopic pregnancies, at a rate of 6 ectopic pregnancies per 1000 reported pregnancies. This number represented a five-fold increase compared with the 1970 rates [2]

At the Kenyatta National Hospital, Mwathe found an incidence of 2-5 ectopic pregnancies per week.[3] Ectopic pregnancy remains a potentially lethal condition, and although mortality has diminished, there are still several deaths every year from ectopic pregnancies. In the United States for instance, it remains the second leading cause of maternal mortality and the leading cause of maternal mortality in the first trimester. [4] The risk of death from an extrauterine pregnancy is 10 times greater than that of vaginal delivery and 50 times greater than for induced abortion. However, with early diagnosis, both maternal survival and conservation of reproductive capacity is enhanced.

The causes of ectopic implantation are unknown, but several risk factors lead to tubal damage and dysfunction. Independent factors consistently shown to increase the risk of tubal pregnancy include; previous laparoscopically proven pelvic infection, previous tubal pregnancy, current intrauterine device use (IUCD) and previous tubal surgery for infertility (2,4). Many other factors including previous pelvic/abdominal surgery, smoking, Douching, previous genital infection and infertility have also been identified. Assisted reproduction also confers increased risk. R.N was a Para 4+0, fertile lady who was using an IUCD for contraception. She had no prior history of genital infection or pelvic surgery.

Inert and copper-containing IUCD's prevent both intrauterine and extra uterine pregnancies. Women who conceive with an IUCD in place, like our patient, are 0.4-0.8 times more likely to have a tubal pregnancy than those not using contraceptives. However, because IUCD's prevent implantation more effectively in the uterus than in the tube. A woman conceiving with an IUCD is 6-10 times more likely to have a tubal pregnancy than if she conceives without using contraception.[2]

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The diagnosis of ectopic pregnancy is complicated by the wide spectrum of clinical presentations, from asymptomatic cases to acute abdomen and haemodynamic shock. Patients

who have an ectopic pregnancy generally have an abnormal menstrual pattern or the perception of a spontaneous pregnancy loss. The classic symptom triad of ectopic pregnancy is pain, amenorrhea and vaginal bleeding. This was the case with our patient. However, the symptom group is present in only about 50% of patients, and is most typical in patients in whom an ectopic has ruptured like our patient. Shoulder and back pain, thought to result from hemoperitoneal irritation of the diaphragm may indicate intraabdominal hemorrhage. The physical findings before rupture are nonspecific, and vital signs normal. The abdomen may be non-tender or mildly tender, with or without rebound tenderness. The uterus may be slightly enlarged, with findings similar to a normal pregnancy.[5]Cervical motion tenderness may or may not be present. An adnexal mass may be palpable in up-to 50% of cases but the mass varies markedly in size, consistency, and tenderness. With rapture the patient develops hypotension and peritoneal irritation. Bowel sounds are decreased or absent. The abdomen is distended, with marked tenderness and rebound tenderness. Cervical motion tenderness is present. Frequently, the pelvic examination is inadequate because of pain and guarding.

Diagnosis is made from history, physical examination and laboratory data such as serial Beta HCG measurements. In ectopic pregnancy, HCG levels rise slowly and the doubling time is prolonged.[6]Abdominal ultrasound is invaluable in terms of diagnosis, but transvaginal ultrasound may be more accurate in detecting unraptured ectopic pregnancy. In case of suspected rapture, paracentesis yielding, non-clotting blood is usually confirmatory. This was the case with our patient; hence no laboratory Beta HCG levels were done. In case of unraptured ectopic pregnancy, laparoscopy remains the gold standard of diagnosis. [5]Diagnosis in our patient was based on history and physical examination alone. The management of ectopic pregnancy can either be medical or surgical. Both methods are effective, and the choice depends on the clinical circumstances, the site of the ectopic, and the available resources. Operative management is the most widely used treatment for ectopic pregnancy. It is the only method used in our unit. It is especially urgent if rapture has occurred with associated haemodynamic compromise. Linear salpingostomy is currently the procedure of choice when the patient has an unraptured ectopic pregnancy and wishes to retain her potential for future fertility. [2]The products of conception are removed through an incision made into the tube on its antimesenteric border. Operative laparoscopic techniques or laparotomy

can be used. Milking of fimbrial ectopic gestation may also be effective. Salpingectomy was preferred in our patient because the tube had raptured, and our patient wan at the eve of her reproductive career.

The drug most frequently used for medical management of ectopic pregnancy is methotrexate, although other agents like potassium chloride (KCL), hyperosmolar glucose, prostaglandins and RU-486 have been studied. These agents may be given systemically (Intravenously, Intramuscularly or orally), or locally (Laparoscopic direct injection, transvaginal ultrasound-directed injection or retrograde salpingography). A single dose methotrexate regimen has been found cheaper, patient acceptable, associated with less side effects and having better future fertility prospects and is currently preferred. It's not currently available as a method of treatment of ectopic pregnancy in our unit, because early diagnosis is uncommon and follow up likely to be unreliable.

After an ectopic pregnancy, there is a 7-13 fold increase in the risk of a subsequent ectopic pregnancy. Post-operative counseling was done for R.N, to change her contraceptive method (IUCD), which was removed at operation to one with a lesser recurrent risk of ectopic pregnancy.

REFERENCE

1. Guy I, Benrubi. J.B; Obstetric and Gynecologic Emergencies. Chapter 5 pg. 41-57. Lippincott Company, Philadelphia 1994.
2. Thomas G. Stovall. Early Pregnancy loss and Ectopic pregnancy. In Novak's Gynecology 13"' edition, Lippincott Williams & Wilkins, Philadelphia, PA 19106 USA, 2002.
3. Mwathe E. G. Pattern of ectopic pregnancy at KNH. M. Med. thesis U.O.N 1984.
4. Cunningham F.G, McDonald, Gant, et al. In: Williams Obstetrics 21st edition, 2001. McGraw-Hill Company: chapter 34; Pg 883-905.
5. Sivin I. Dose and age-dependent ectopic pregnancy risks with intrauterine contraception. Obstetric Gynecology 1991, 78:291-298.
6. Dorfman S.F, Grimes DA Cates W.J. et al. Ectopic pregnancy mortality, United States: 1979 to 1980: Clinical aspects. Obstetric, Gynecology 64: 386. 1984.

GYNAECOLOGY CASE 3

VESICO – VAGINALFISTULA – REPAIR.

NAME:	E.W	PARITY:	1+0
AGE:	32Years	HEIGHT:	164cm
IP NO:	0972375		
DOA	19/09/06		
DOD:	27/09/06		
WARD:	1B		

PRESENTING COMPLAINTS

E W presented with a 4-month history of leakage of urine.

HISTORY OF PRESENTING COMPLAINTS

E. W was well till her delivery 4 months earlier. After being in labor for 2 days in a private clinic in Nairobi she was referred to Pumwani maternity hospital (PMH) because of obstructed labor. At PMH, a vacuum extraction was done, the result being a fresh stillbirth weighing 3.6 kilograms. After delivery no catheter was inserted. A few hours later in the post-natal ward she noticed continuous drainage of urine. A catheter was then inserted and she was started on antibiotics. At discharge she was referred to KNH for specialized management. At the Gynecological outpatient clinic (GOPC) the catheter was changed and she was booked for repair on 27/7/04.

OBSTETRICS AND GYNAECOLOGY HISTORY

She attained her menarche at the age of 14 years. Her menses were regular, occurring every 24-26 days, and lasting 3 days. She had dysmenorrhoea on her first day of menses. She had never used any method of contraption. She had never had a pap smear done. Her last menstrual period was on 14/7/04

FAMILY AND SOCIAL HISTORY

She was married to a businessman in Nairobi. They lived together in Kariobangi. She was the 4 th born in a family of 8 siblings, and her parents lived in Mwingi. She did not take alcohol,

or smoke cigarettes. There was no family history of chronic ailments.

PAST MEDICAL HISTORY

She had never been hospitalized, apart from the one mentioned, related to her current condition.

PHYSICAL EXAMINATION

She was a young lady in general condition and well nourished. She was not pale, did not have Jaundice, edema or Lymphadenopathy. Vital signs were a temperature of 36.6°c, pulse rate of 72/min, respiratory of 16/min and blood pressure of 110/70 mmhg.

ABDOMINAL EXAMINATION

The abdomen was scaphoid, soft and non-tender. There were no masses or organomegaly.

PELVIC EXAMINATION

There was excoriation of the skin in the perineum and external genitalia. There was obvious leakage of urine from the vagina. There was some fibrosis on the anterior vaginal wall with leakage about 2cm from the external urethral meatus.

DIAGNOSIS

Vesico-Vaginal fistula type 11was made.

A full hemogram and urea and creatinine were ordered were normal.

PRE-OPERATIVE PREPARATION

On the day of admission she was given a fluid diet, an enema in the evening prior to the operation. Shaving was done and she was advised not to eat anything 12 hours before the operation. On the morning of the operation a second enema was given, and a drip of 5% dextrose started. Informed consent was obtained and intramuscular atropine 0.6mg given half hour before being taken to theatre.

EXAMINATION UNDER ANAESTHESIA AND REPAIR AT THE SAME SETTING

In theatre the patient was put under general anesthesia. She was positioned into the exaggerated lithotomy position. The vulva and perineum were cleaned with antiseptic and examination performed. A diagnosis of VVF type IIAa was made.

A medial lateral episiotomy was done to access the fistula site. Sharp dissection was done of the vaginal wall from the bladder. The bladder was then mobilized. Tension free transverse Bladder-urethral closure was done in a single layer using Vicryl No. 3/0. A number 18 foleys catheter was then inserted into the bladder and a dye test done. No leakage was observed. Adaptation of the anterior vaginal wall was done in an everted fashion using Vicryl 2/0. The episiotomy was then repaired in layers. The anesthesia was reversed and patient transferred to the ward.

POST-OPERATIVE CARE

Post-operatively the patient was advised to take plenty of fluids and no leakage of urine was observed. She was discharged on the 5^{th} post-operative day, and instructed to come back for dye test on the 14^{th} post-operative day, which she honored and reported no leakage of urine. A dye test done did not show any leakage. She was instructed to continue pelvic muscle stimulation and exercises in the physiotherapy department and to avoid coitus for 3 months. She was for subsequent follow up in the GOPC, and was advised to start antenatal care promptly in the next pregnancy and report to the attending doctor that she had a WF repair. She was advised that all future deliveries should be by cesarean section.

DISCUSSION

Presented is an 18-year-old Para 1+0, who had obstructed labor in her only delivery. A vacuum extraction was done, but unfortunately she developed a vesico-vaginal fistula soon after the delivery. Her fistula was successfully repaired. Advice was given that all future deliveries should be by cesarean section. John Fatio did the first successful repair of fistula in 1675, though the pioneering work that set the stage for modern surgical repair of fistulae has been credited to Marion Sims (1). The actual incidence of fistula is impossible to calculate but Harrison has suggested an incidence of 95 per 100,000 (2). By far the leading cause of

vesicovaginal Fistula in Africa is obstetric trauma. Mati (1982), reported that 87.8% of the cases of urinary fistula were labor related in his series (3), while Orwenyo (1984), reported a rate of 90.7% (4). The estimated prevalence of genital fistula in Africa is 1-2 per 1000 deliveries (3, 4, and 5). This is in contrast to the developed world where obstetric trauma is rarely responsible for fistula formation. The patient presented developed fistula after obstructed labor, in which the baby died in the process of labor. Other causes of fistula include cancer of the cervix, surgical procedures such as hysterectomy and pelvic repair operation.

The most common cause of obstetric fistula is pressure necrosis following compression of vesicovaginal septum. This leads to devitalization of tissue by ischaemia that then sloughs off between the second and fourteenth postpartum day (6). The area usually affected is the anterior vaginal wall and the underlying bladder neck; occasionally the upper vagina and anterior lip of the cervix. Although prolonged and obstructed labor is the direct leading cause of obstetric fistula, it is invariably a result of neglected or unmanaged cephalopelvic disproportion. In Nairobi, it has been shown that the average true conjugate among women sustaining fistula was less than 9.Ocm (7). It has been suggested that the small pelvic may be a result of protein malnutrition in childhood. Improvement in childhood diet has been recognized as one of the major contributions, to reduction of cephalopelvic disproportion in developed nations.

In Kenya, the peak incidence of fistula was observed in the 20-24 years of age group by Gunaratne and Mati (3). The majority of women suffer the injury while giving birth to their first child. In the majority of cases, the baby is stillborn or dies shortly after birth. The birth rate has been reported to range between 64% and 79%, and those born alive, more than 50% die in neonatal period (3). Orwenyo reported a perinatal mortality rate of 80.4% (4).

Cultural factors also contribute to the risk of complicated childbirth. Even when skilled maternity care is available, utilization may be poor, and confidence in its value is hard to win, so prenatal

clinics are poorly attended and elderly relatives or unskilled traditional birth attendants usually conduct deliveries at home, referring patients when it is too late. Obstetric fistula is associated with other complications including recto-vaginal fistula in 10% of cases (3). Obstetric palsy, severe vaginal stenosis and secondary amenorrhoea also occur.

Once a fistula is formed, continuous bladder drainage and control of infection with antibiotics have been shown to reduce the size of the fistulae and may even head to closure of the fistula (6). WaaldJik reported that insertion of an indwelling catheter immediately after obstructed labor is detected promoted spontaneous healing of small fistulae up to 2cm in size. This may occur in up to 60% of all patients (8).

Vesico-vaginal fistula has been classified into two:

- Type 1 Fistula involving the closing mechanism. Usually 5cm or more above the urethral opening
- Type 2 Fistula involving the closing mechanism. Usually within 5cm from the urethral opening.

These are further subdivided into:-

- A Without total involvement of urethra.
  - (A) Without a circumferential defect.
  - (B) With a circumferential defect.
- B With total urethral involvement.
  - (a) Without a circumferential defect.
  - (b) With a circumferential defect.
  - (c) Miscellaneous e.g. Uteral-vaginal fistula and exceptional fistula.

The above classification has prognostic value, with a worsening prognosis for the higher class. The system also predicts outcome of surgery and the likelihood of incontinence postoperatively

(5). Most authors maintain that repair of fistula should not be attempted till about 3 months after the causative event. This can reduce the size of the fistula by spontaneous healing, and allows tissue reaction to subside and revascularization to occur (6, 7). Currently, early (after 6-8 weeks) full repair is advocated as soon as the slough has disappeared (8). Subsequently, the current recommendation is that any woman who develops an obstetric fistula should have a catheter inserted and As soon as the slough has disappeared and the fistula is clean an early repair (6-8 weeks) should be done unless the fistula has already healed.Good postoperative care is crucial to fistula repair. The aim is to have a high volume of uninterrupted urine flow as this prevents ascending urinary tract infection and prevents catheter blockage which is essential to maintain the bladder empty and thus avoiding putting tension on the suture line. A minimum of 4 liters of fluids in 24 hours is recommended. Antibiotics are not thought to be absolutely essential, since pressure necrosis and not infection is the principal cause of the fistula.

Successful repair is gauged by whether the woman is continent of urine. The site and extent of the fistula may affect the operative success, but Lawson states that experienced surgeon supported by a competent nursing staff should be able to achieve 75% success at the first attempt, and a further 15% at the second attempt (6). Since each successive repair produces more scar tissue, successful repair at the first attempt is the goal.Making basic maternity services available to an increasing number of women in the rural areas, where 80-90% of the population lives, and where most patients with VVF originate can reduce the incidence of fistula. Such services should seek to identify women at risk of difficult delivery. The partograph, introduced by Phillpot, was intended to help identify difficult labors and facilitate prompt referral (9). Traditional birth attendants should also be trained to improve the quality of care they provide. After repair the patient should avoid coitus for 3 months post repair. Future deliveries should all be by elective cesarean section. Our patient was counseled post repair on sexual practice and future deliveries

REFERENCES

1) World Health Organization, Maternal Health and Safe Motherhood Programme; April 1989, PP 3-11.

2) Gunratne M, Mati JKG. Acquired fistula of the female genital tract; A comprehensive five-year review. J. Obstet. Gynecol East and Central Africa. 1982; 1:11-15

3) Orwenyo E.A. A retrospective study of 166 cases of acquired urinary genital and/or rectovaginal fistula treated at Kenyatta National Hospital. 1979-1982; M.Med Thesis, U.O.N 1984.

4) Ojanuga OD, Ekwempu CC; An investigation of socio-medical risk factors associated with fistula in Northern Nigeria; Women and Health; 28; 3:103116. 1999.

5) Lawson J.B; Stewart D.B; Obstetrics and Gynecology in the tropics; London, Arnold Publ; 1976.

6) Gebbie D.A.M Obstetrics and Gynecology in Health and Disease in Kenya, Vogel LC (Ed); Nairobi, EARL, 1974, P497.

7) Waaldijk K: Step by Step of vesicovaginal fistulas capion press 1994.

8) Philpott R.H. Graphic records in labour: Brit. Med. J. 1972; **4:** 163.

GYNAECOLOGY CASE NO 4

SYMPTOMATIC UTERINE FIBROIDS: TOTAL ABDOMINAL HYSTERECTOMY

NAME: A.W **AGE:** 44 YRS

IP NO: 1091424

DOA:25/6/07

DOD 29/06/07

HISTORY OF PRESENTING ILLNESS

H.A. was well until 6years ago when she noticed lower abdominal swelling, which was progressively increasing in size. It was firm and nodular. One year later she started experiencing low abdominal pain, which was dull in nature, intermittent, and could occasionally radiate to the back. Three years ago, she developed heavy and prolonged menses which could occasionally come in clots lasting between 2 – 4 weeks duration and she could change her pads 5 -10 times per day, fully soaked. Initially, her menstrual flow could last for 3-4 days in a 28 days cycle. A few months before admission, she started having dizziness, general body weakness and awareness of heartbeat and that's when she decided to seek medical attention. There was no associated history of chest pain, cough, yellowing of the eyes, urinary symptoms or history of weight loss.

PAST MEDICAL HISTORY

She was a known hypertensive patient controlled on medication (Adalat R) but had not been on follow up. There was no other significant history.

OBS/GYN HISTORY

Menarche was 15 years. Her menstrual flow was 3-4 days in 28 days cycle. She had moderate flow changing pads 2-3 times in a day. There was no history of dysmenorrhoea. She had never used any contraceptives in her entire life.

The patient was Para 3+0, last delivery was in 1991.All of them were spontaneous vertex delivery and all the children were alive and well. Last normal menstrual period was 3years ago.

FAMILY SOCIAL HISTORY

She was a widow and stayed at Meru. She did not smoke or take alcohol. Her husband died 3years ago due to a road traffic accident. There was no other chronic illness in the family.

On examination, she was in good general condition, mildly pale, not jaundiced, and not edematous a febrile with no lymphadenopathy.

Vital signs: BP 120/70 mmHg, PR 80/min, RR 20 /min and temperature was 36.5°c

ABDOMINAL EXAM

The abdomen was uniformly distended at the lower part with a pelvic mass. There were no scars or dilated veins and the abdomen was moving with respiration and the umbilicus was not everted. The mass was corresponding to 20 weeks of gravid uterus. It was firm, had nodular surface and was mobile from side to side with dull percussion note.

V/E: The external genitalia was normal. The cervix was long and firm and the cervical os was closed. The mass was found to be continuous with the uterus as when the mass was moved from side to side the cervix was found to move with it. The uterus was bulky at 20 weeks. The adnexae had no masses and were non-tender bilaterally. There was no bleeding or abnormal discharge noted.

An impression of Symptomatic uterine fibroids was made.

Lab investigations

Hb 12.2g/dl WBC 5.2 x 10^9/L, Platelets 156 x 10^9/L

Urea 6.5mmol/l, Creatinine 75 Umol/l

Pap smear: Showed satisfactory smear showing mainly squamous, cells within normal limits. Endocervical cells were present and normal. The uterosacral ligaments were also double clamped, cut and distal portions ligated with vicryl No. 1 suture. The cervico-vaginal junction was identified. Little woods tissue forceps were used to grasp the interior vaginal wall

which was then opened using a knife. A pair of scissors was used to circumcise off the whole cervix. The margins of the vagina were secured with long artery forceps. The vagina vault was closed with interrupted mattress sutures and tied with sutures .from the cardinal and uterosacral ligaments. Peritonization was done with vicryl No. I suture.

Follow up She was seen after 2 weeks and found to be doing well. Her blood pressure was well controlled on Adalat **R** 20mg OD and the wound was well healed. Histology report confirmed of uterine fibroids.

DISCUSSION

H.A. was a 48 year old Para 3+0 with symptomatic uterine fibroids (uterine mass and heavy menses). She did not use any method of contraception. Total abdominal hysterectomy was done with resolution of her symptoms.

Uterine leiomyomas (i.e. fibroids/ leiomyofibroma/ myomas/ myofibroma) are benign clonal turnours, arising from the smooth muscles cells of the uterus and containing an increased amount of extracellular (fibrous connecting tissue) matrix. It is a well

circumscribed tumour that is not encapsulated.[3] They may be asymptomatic or can be associated with uterine bleeding, pain or reproductive problems.

Fibroids are the commonest pelvic tumours being present in about 20-25% of all reproductive age women. By the fifth decade as many as 50% of black women will have fibroids. They are rare before the age of 20 years and are 3-9 times found more commonly in black women than in white women [1]. Fibroids are also more common in nulliparous and relatively infertile women. It is not clear whether sub fertility causes myomas or myomas causes sub fertility or both have a common cause.[2]

At Kenyatta National Hospital, fibroids account for 1.6% of all gynaecologic admissions and 66.7% of all hysterectomies performed are due to uterine fibroids [4] Wanjala found that 30% of

patients with uterine fibroids had parity of 3 and above, but 85% of them had not delivered within the last 6 years.

The cause of uterine fibroids is not known but glucose-6-phospate dehydrogenase studies show that each fibroid is unicellular (monoclonal) in origin'. Oestrogens have been implicated in the growth of fibroids. Evidence of this includes increased oestrogen receptors in myomas compared to the surrounding myometrium and increase in size with oestrogen therapy and during pregnancy but decrease in size and even disappearance following menopause [1, 3]. Leiomyomata growth in pregnancy is related to synergistic activity of oestradiol and human placental lactogen (HPL). Other conditions associated with uterine fibroids include follicular cysts of the ovary, endometrial hyperplasia, endometrial carcinoma and endometriosis.

Classification of myomas is based on anatomical location as follows:-

Submucosal – lie just beneath the endometrium and grow towards the lumen.

Intramural or interstitial – lie within the myometrium.

Subserous or subperitoneal – lie at the serosal surface.

Submucous fibroids may be pedunculated, subjecting them to torsion and infection and may also herniate through the cervical os. Subserous fibroids may also become pedunculated and sometimes acquire extra uterine blood supply. It is called parasitic when its pedicle atrophy and resorb. Subserous myomas may also extend laterally between the two peritoneal layers of the broad ligament to become intra-ligamentary fibroids [1, 2, 3]

Symptomatic fibroids are found in 30-50% of patients. The commonest symptom is abnormal uterine bleeding found in 30% of patients. The bleeding is due to:-

a) Increased endometrial surface area.

b) Increased uterine vascularity

c) Endometrial hyperplasia

d) Endometrial ulceration

e) Dilation and engorgement of the venous plexus.

Menorrhagia is common while metrorrhagia is associated with a tumour with endometrial venous thrombosis and necrosis on its surface and particularly if it is pedunculated and partially extrudes through the cervical canal. At KNH, Wanjala found menstrual disturbances in

54.7% of the patients while 38.3% presented with an abdominal mass[4].

The presenting complains may be inability to conceive, although fibroids may be the primary cause in 2-10% of the patients. Infertility may be due to impaired implantation, impaired tubal motility or interference with sperm transport eg corneal blockage. Spontaneous abortion increases 2-3 fold in the presence of fibroids. This could be due to increased uterine irritability and contractility, altered oxytocinase activity or alterations in endometrial stroma / vasculature leading to reduced blood supply to the developing placenta thus increasing rate of foetal waste wastage [5]. After myomectomy, the incidence of spontaneous abortion reduces from 40% to 20%. Dysemenorrhoea is common in fibroids and may be due to circulatory seclusion, infection, torsion, myometrial contractions to expel a submucous mycoma or sarcomatous changes.

Systematic manifestations of myomas include anaemia (iron deficiency anaemia) which is secondary to menometrorhagia. Paradoxically polycythemia is present in some cases of uterine fibroids particularly in the broad ligament. This is due to production of erythropoietin by the tumour or renal erythropoietin production as a result of ureteric compression. Pressure effects may also occur on the bladder and rectum. On rare occasions a retroperitoneal myoma may cause hypoglycaemia possibly through pancreatic stimulation. Secondary changes of fibroids include atrophy, hyalinization, calcification, cystic changes, sepsis and red degeneration. Malignant changes are rare (0.1 – 0.5%)

Choice of treatment depends on patients' age, parity, general health, symptoms, desire for future fertility, location and state of lyomyomata. Asymptomatic fibroids which are less than 12-14 weeks require no treatment if the patient is post menopausal with no symptoms. The patient is followed up every six months. Medical treatment can also be carried out even though it is not definitive. Gonadotrophin releasing hormone (GNRH) agonists limit the growth or reduce the tumour size to facilitate easier surgery. The use of GNRH agonist is only temporary as they create an artificial menopause [6] Orgametril, a synthetic progestagen active orally is effective in controlling menorrhagia. After treatment 87.5% of patients are symptom free by third treatment cycle [7].

Surgery is indicated in symptomatic uterine fibroids such as those larger than 14 weeks, growing cervical myomas 3-4 cm in diameter, pedunculated submucous and sub serous fibroids[3]. Myomectomy is the operation of choice in patients who wish to retain menstrual and reproductive functions. However recurrence occurs in 15 – 40% of patients and two thirds of these will require further surgery[1].

Hysterectomy is the preserve of patients who do not desire future fertility. It is usually curative as all the tumours are removed with the uterus. It can be done either abdominally or vaginally with the later being more desirable for smaller fibroids or if combined with repair of cystocoele or rectocele.

REFERENCES:

1. Pernoll M. L, Wexler A.S, Benign Disorders of the Uterine Corpus. Current Obstetrics and Gynaecologic Diagnosis and Treatment. 8th Edition, Appleton and Lange 1994 pg 731

2. Tindall V.R. Tumours of Corpus Uteri. Jeff Coates Principles of Gynaecology. 5h Edition, Britterworth and Co 1987 pg 417

3. Thompson J.D, Rock J. A. Leiomyomata Uteri and Myomectomy. Te Lindes Operative Gynaecology. 8h Edition, Lippincott — Raven, 1997 pg 731-85

4. Wanjalla S.N.M. Uterine Fibroids at KNH. MMed Thesis, University of Nairobi, 1980

5. Bultran V, Reiter R. Uterine Leiomyomata. Aetiology, Symptomatology and Management. Fert. Ster. 36.433, 1981.

6. West C.P, William J, Lumsden M.A. Shrinkage of Uterine Fibroids during Therapy with Goserelin. Fert. Ster 48 (1) 1987

7. Muhiu G. Effect of Orgametril (Lynestrenol) on Menstrual blood loss in Uterine Fibroids. MMed Thesis, University of Nairobi 1986

GYNAECOLOGY CASE 5

SEXUAL ASSAULT- EMERGENCY TREATMENT

NAME	M.M
AGE	16 YEARS
CASUALTY. NO.	20735
WARD	1D
DOA	25/04/06
DOD	25/04/06
PARITY	0+0

HISTORY OF PRESENTING COMPLAINTS

M.M, a standard 8 pupil in a local primary school, was walking home when an unknown assailant sexually assaulted her. On the fateful day, she stayed behind in school to complete her homework, starting on her journey back home at 6.00pm. As she attempted to shorten her journey home through a short cut in a coffee plantation, a man that was walking behind her grabbed her from behind into the coffee bushes. He threatened her with a knife he was carrying if she did not cooperate. He sexually assaulted her for about one hour, before disappearing into the coffee farm. Concerned about her lateness, her father met her as he came looking for her.

OBSTETRIC AND GYNECOLOGY HISTORY

She had never been involved in any sexual activity and was a nullipara. Her menarche started at the age of 14 years, and was now regular, lasted 3-4 days with an interval of 26 days. There was no dysmenorrhoea. She had never used any contraceptives.

FAMILY HISTORY

She was the first born in a family of 2 siblings. Her brother, 3 years her junior was now in class seven in the same school. There was no family history of chronic ailments.

PHYSICAL EXAMINATION

She was a young lady in good general condition but looked very apprehensive. She did not have pallor, jaudice, edema or lymphadenopathy. She had bruises on her elbows and knees. Vital signs were a blood pressure of 100/60 mmhg, pulse of 100/min, respiratory rate 14/min and a temperature of 36.4°c.

ABDOMINAL EXAMINATION

The abdomen was scaphoid and moving with respiration. It was non-tender with no masses or organomegaly.

MUSCULOSKELETAL SYSTEM

There were bruises on the elbows and right knee.

PELVIC EXAMINATION

The vulva was bruised, and some blood clots were present at the vaginal introitus. The hymen was broken. There were no vaginal lacerations or tears. Semen was evident in the posterior fornix. A vaginal swab was taken for microscopy, wet mount culture and sensitivity.

DIAGNOSIS

Sexual assault (rape).

MANAGEMENT

After examination, the victim and her parents were explained of the findings and the major risks of sexual assault on the victim. Pre-Test counseling and consent for HIV testing was obtained. They also gave consent for blood to be drawn for laboratory analysis of pregnancy (PDT). VDRL, and Hepatitis B and C. Hemogram was also taken.

RESULTS

Hemogram	Normal blood picture.	Hb level 12.6gidl.
VDRL	NEGATIVE	
HIV	NEGATIVE	

- Pregnancy test NEGATIVE
- Hepatitis NEGATIVE for both B and C.
- Vaginal swab for microscopy was normal.

TREATMENT.

The patient was started on Prophylaxis for sexually transmitted infections (STI's) with oral Ciprofloxacin 500mg start, Metronidazole 400mgs thrice daily and Doxycyline 100mg twice daily for one week. She was put on Postinor II, one tablet start to be followed by a second tablet 12 hours later to protect against pregnancy. It was also decided to start her on Post-exposure prophylaxis for HIV, with Combivir one tablet twice daily for 28 days. She was seen by a counselor and on going supportive counseling arranged. She was referred for follow up in the patient support centre in 2 weeks.

FOLLOW UP

Two weeks later the victim was doing well and continuing with anti-retroviral therapy. She was informed that the culture of her cervical swab did not grow any organisms. She booked for repeat serologic test for syphilis, hepatitis B and HIV in 4 weeks

DISCUSSION; She presented as a 16-year old school girl raped on her way from school by an unknown assailant. She was promptly brought to hospital and received emergency treatment, vis-a-vis, prophylactic antibiotics, antiretroviral therapy and emergency contraception. Ongoing supportive counseling was also initiated.Sexual assault of children and adult women has now reached epidemic proportions in the United States and is the fastest growing; most frequently committed, and most underreported crime (1,2).

In Kenya, no accurate records exist on the incidence of sexual assault by if hospital and police reports are anything to go by, the crime is on the rise. Sexual assault is a crime of violence and aggression, not passion, and encompasses a continuum of sexual activity that ranges from sexual coercion to contact abuse (unwanted kissing, touching, or fondling) to forcible rape. Many states have now adopted the gender-neutral term sexual assault in favor of rape, which has traditionally referred to forced vaginal penetration of a woman by a male assailant (3).

Several variants of sexual assault have been described. Marital raped is defined as forced coitus or related sexual acts within marital relationship without the consent of the partner. Acquaintance rape refers to those sexual assaults committed by someone known to a victim. More than 75% of adolescent rapes are committed by acquaintances of the victim. When the acquaintance is a family member, the sexual assault is referred to as incest. When forced or unwanted sexual activity occurs in the context of dating relationship, it is referred to as a date rape. "Date rape drugs" such as flunitrazepam (Rohypnol) and gamma hydroxybutyrate (GHB), are occasionally used to diminish a woman's ability to consent or remember the assault (3). Child sexual abuse is defined as contact or interaction between a child and an adult when the child is being used for sexual stimulation of the adult or another person.

Childhood sexual abuse has a profound and potentially lifelong effect on the survivor. Although most cases of childhood sexual abuse are not reported by the survivor or her family, it is estimated that as many as one third of adult women were sexually abuse as children (4). Women, who are sexually abused, as children tend to be more likely to experiencedepression, chronic anxiety, anger, substance abuse problems, sleep disorders and personality disorders. They also often experience sexual dysfunction and difficulty with intimate relationships and parenting (4). They may also develop the posttraumatic stress disorder (PTSD), defined as development of characteristic symptoms following a

psychologically traumatic event outside of normal human experience. These include blunting of affect denial of symptoms, intrusive re-experiencing of the incident and agitation in response to reminders of the event.

The world conference on human rights in Vienna (1993), resolved that women and girl rights are an inalienable, integral and indivisible part of universal human rights (5). In Kenya, the law states that any person who unlawfully and carnally knows a girl below 14 years is guilty of a felony and is liable to 14 years imprisonment with hard labor and corporal punishment (6). Rape attracts life imprisonment. Various non-governmental organizations now feel these legal provisions are inadequate and are lobbying for stiffer punishment for sexual offences.

Following sexual assault, women have many concerns, including pregnancy, STI's (including human immunodeficiency virus (HIV) infections), being blamed for the assault, having their name made public and having their family and friends find out about the assault. The initial reactions to sexual assault may be shock, numbness, withdrawal, and possibly denial. It is difficult to predict how any assaulted individual will react. Some nay present with rape trauma syndrome characterized by fear, helplessness, disbelief, shock, guilt, humiliation, embarrassment, anger and self-blame (4). Survivors may experience intrusive memories of the assault experience nightmares and a variety of somatic symptoms. Some fear the assailant will return to retaliate or rape them again.

Examination and treatment should be done in a quiet and supportive environment in order to facilitate a thorough history and examination. Because of the legal ramifications, consent must be obtained from the patient before obtaining the history, performing the physical examination, and collecting evidence. Documentation of the handling of specimens is especially important, and the "chain of evidence" for collected material must be carefully maintained. During the interview and examination family, friends, or a counselor should be

encouraged to accompany the patient. The physician's responsibility in treating sexual

assault survivors includes:

1. Obtaining an accurate gynecologic history, including a recording of the sexual assault.
2. Assessing, documenting, and treating physical injuries.
3. Obtaining appropriate cultures (including samples for forensic tests), treating existing infection and providing prophylaxis for sexually transmitted diseases.
4. Providing therapy to avoid unwanted pregnancy.
5. Providing counseling for the patient and her partner and or family.
6. Arranging for follow up medical care and counseling.
7. Reporting to the legal authorities as provided by the law.

At the KNH, we offer counseling, HIV prophylaxis, emergency contraception as well as treatment and or prophylaxis for sexually transmitted disease. This was done in the case presented.

REFERENCES

1. Sorenson SB, Stein JA, Seigel JM, et al. The prevalence of adult sexual assault. AM. J. Epidemiol 1987; 126:1154-1164.

2.American College of Obstetrics and Gynecology. Sexual assault. Technical Bulletin. Washington. DC, ACOG, 1997:242.

3.Micheal C. LU, Jessica S. LU & Vivian P. Haifin. Domestic Violence and Sexual assault. In; current obstetric & gynecologic Diagnosis & Treatment, 9th edition chapter 60 Pg 1087-1093, 2003.

4.David A Baram. Sexuality, Sexual Dysfunction, and Sexual assault. In; Novak's Gynecology 13th edition, chapter 11 ph 295-317. Lippincott Williams & Wilkins, 2002.

Angela Hawke. UNICEF Innocenti Research Centre. Domestic Violence against women and girls. May 2000.

5.Republic of Kenya, Offences against morality. In: Law of Kenya, penal code cap 63, chapter XV.

GYNAECOLOGY CASE 6

CARCINOMA OF THE VULVA STAGE

RADICAL VULVECTOMY + INGUINAL LYMPHADENECTOMY

NAME	R.Y	PARITY	6+0
AGE	68 YEARS	DOA	22.3.2005
IPNO	1017652	DOD	17.8.2005

Complaints

She was admitted through casualty as a referral from Wajir District Hospital with a 2 year history of painful vulva swelling.

History of presenting complaints

She developed a painful vulval swelling two years prior to admission. The swelling progressively increased in size. There was associated pruritus on the swelling and occasional vaginal discharge which was foul smelling. She experienced lower abdominal pains and dysuria.

Obstetrics and gynaecologic history

She was para 6+0. All her deliveries were spontaneous vertex deliveries. She was 20 years postmenopausal and her last delivery was 30 years ago. All her children were alive and well.

Family and social history

She had married and lived with the husband in Wajir. There was no family history of chronic illness. She neither smoked cigarettes nor drank alcohol. The husband used to chew khat but did not smoke or take alcohol.

PHYSICAL EXAMINATION; She was an elderly lady in fair general condition with no fever, pallor jaundice or oedema. She had bilateral inguinal lymphadenopathy. The nodes were firm, fixed to the underlying structures and measured about 1 x I cm in diameter.

ABDOMINAL EXAMINATION; The abdomen was scaphoid and moved with respiratory. There were no surgical scars or palpable messes.

VAGINAL EXAMINATION

There was an ulcer on the lower half of the left labia majus. Its size was 3 by 4 cm. It was bleeding early. There were no signs of infection. It was not possible to do a digital examination.

Diagnosis

A diagnosis of cancer of the vulva stage III was made.

MANAGEMENT; The diagnosis was explained to her through a translator. She was first to be done examination under anaesthesia and biopsy taken for histology.

An abdominalpelvic ultrasound performed on 30.3.2005 was reported as a normal

Examination under anaesthesia (EUA)

She was taken for EUA on 8.4.2005. At EUA, she was found to have an ulcer arising from the left labia majus and covering half of the left vulva. It was bleeding easily on touch and had raised edges. There were circumferential nodules on the lower 1/3 of the vagina. It was not possible to put a speculum through the intoitus On rectal examinaion the rectal mucosa was freely mobile. A biopsy was taken from the ulcer for histology.

History results showed an anaplastic carcinoma.

The patient was then planned for radical vulvectomy and partial vaginectomy. The nature of the operation was explained and informed consent obtained.

Haemoglobin level and urea and electrolytes were repeated on 14.5.2005 and the results were : Hb 11.2 g/dl, Sodium 142 mmol/L Potassium 4.1 mmol/I and BUN 4.5mmol/l. She was scheduled for operation on 19.5.2005. Blood was taken for grouping and cross — match and three units reserved for the operation. She was done enema at 6.00 pm the previous day and at 6.00 am on the day of the operation. She was starved from midnight. On the morning of the operation she was premedicated with atropine 0.6 mg intramuscularly and wheeled to theatre.

Radical vulvectomy

In theatre, she was put under anaesthesia. She was placed in lithotomy position. The abdomen, vulva and perineum were cleaned with savlon solution and draped with sterile towels. The urinary bladder was catheterised with a Foleys catheter and clear urine drained.

A transverse incision was made above the in[g]uinal ligaments and extended to the anterior superior iliac spines. The incision was deepened to reach the aponeunosis of the external oblique muscle. Two incisions were then made running from the ends of the tranverse incision long the labio-crural folds. The vulva was dissected off en block. Both superficial and deep inguinal lymph nodes were removed bilaterally. Half of the vagina and the area above the anal orifice were removed as well. Haemostasis was achieved by electrocoagulation of the vessel stumps. The resulting raw area was approximated starting from the inguinal area towards the urethra and vagina.

A urethra catheter was left in situ. A firm T bandage was applied. The patient was successfully reversed from anaesthesia. Total blood loss was 700 ml. She was transfused two units of blood intra-operatively. The specimen was taken for histology.

Postoperative care

She was transferred to the recovery ward and observed half hourly until fully awake and then transferred back to the ward. She was put on intravenous fluids normal saline alternating with 5% dextrose 3 litres in 24 hours. She was started on intravenous crystalline Penicillin 2 mega units 6 hourly. Gentamicin 80 mg hourly and Metronidazole

She was given Pethidine 500 mg 6 hourly for 48 hours.

She developed wound sepsis and on the seventh post—operative day the stitched area broke down. She continued on wound dressing twice daily and intravenous antibiotics. On 21.7.2005 she was taken to theatre and skin grafting of the wound done. Post-operatively the graft took well and both the recipient and donor sites healed without sepsis.

Histology result of the specimen taken at vulvectomy confirmed squamous cell carcinoma with lymphnode metastasis. The margins were fee of tumour. She was discharged home on 17.8.2005 to book radiotherapy clinic in four weeks.

Follow up

She came for radiotherapy after the four weeks and was given one course of radiotherapy. She has continued coming for radiotherapy erratically due to financial constaints.

DISCUSSION

R.Y was a 68-year-old postmenopausal patient who presented with carcinoma of the vulva stage 111. Radical vulvectomy was done and she was referred for radiotherapy.Carcinoma of the vulva is an uncommon malignancy accounting for 3-5% of A genital malignancies (1, 2). In Kenya 3.3 % of all genital malignancies are due to vulva carcinoma. It is fourth commonest malignancy after cervical carcinoma, ovarian cancer and choriocarcinoma. An average of four cases are seen annually at KNH. Carcinoma of the vulva accounted for 3.5% of all female cancer deaths ([3]) Vulvar carcinoma may arise from the skin, subcutaneous tissues, urethra, glandular elements of the vulva or the mucosa of the power 1/3 of the vagina (2). Histologically, 90% are squamous cell type, 3.5%

melanoma. 1% basal cell type and 1% originate in the Bartholin's gland. Sarcomas and adenocarcinomas contribute less than 1% (4).

The patient presented had squamous cell carcinoma and it originated in the labia majora.

Aetiology of vulvar carcinoma is unknown. It is predominantly a disease of old women with a median age of 65 years at diagnosis (1, 2, 5, 6). Of the women with vulvar carcinoma 3/4 are aged over 60 years. At KNH 51-70% of the patients were 60 years.

Aetiological factors include long-standing irritative agents (chemical or infective) especially when combined with poor hy[g]iene, chronic inflammatory diseases like venereal granulomas, long–standing vulval warts and chronic vulval dystrophies especially, when associated with epithelial overactivity (8). Associated disorders found most frequently with vulvar carcinoma are obesity, hypertension and chronic vulvar irritation secondary to diabetes mellitus (2).

The patient presented did not have any of the above risk factors.

Vulvar carcinoma in-situ, like carcinoma in-situ of the cervix, is considered a precussor to invasive disease though the risk of progression is lower occurring in about 3% of patients (9). Vulvar carcinoma in-situ tends to be multifocal with a lower risk of invasive disease in younger women but tends to be unifocal with higher of invasive disease in older women (10). For this reason all patients with vulvar carcinoma in-situ should be treated and long term follow—up is mandatory. Patients with carcinoma of the cervix are at increased risk of developing vulvar carcinoma and vice versa (1).This patient had no previous history of a vulvar lesion.

Majority of squamous cell carcinoma's (70%) develop in the labia most commonly the labia majora. 11-12% of cases develops on the clitoris. In about 10% of cases the tumour is so large that the precise site of origin is uncertain (5). Vulvar carcinomas are frequently

ulcerated with raised margins and sloughing base but they can also be fungating papillomatous form or flat plaques (4, 5, 8). The patient presented had a large ulcer coverin[g] half of the vulva on the left side.

The commonest presenting complaints include vulvar lump or ulcer, prunitus, discharge, bleeding and pain (1, 2, 5, 8). The patient ma[y] be aware of a lesion on the vulva but delay in seeking medical help is common. There can also be delay by the physician treating for other conditions without taking biopsy for histology (6).

Spread of vulvar carcinoma can be by local extension to the vagina, urethra, groin and anus. Lymphatic spread which is the commonest route of spread, occurs to the inguinal, femoral and pelvis lymphnodes. Haematogenous spread is rare and occurs late to the lungs, liver and bones (1, 4, 5, 8). About 30% of patients have inguinal nodal metastases at the time of initial diagnosis and 10-20% have pelvic node metastases (5).

In this patient the inguinal lymphnodes were involved at admission.

Staging is done using the modified International Federation of Gynaecologists and Obstetricians (FIGO) staging which is based on prognostic variables and also takes into account the depth of invasion in early stage disease (11).

This patient had III B disease. Diagnosis of vulvar carcinoma depends on biopsy and histologic examination of the tumour. Small tumours or naevi are done excisional biopsy. Colposcopy may be used to identify abnormal areas (after application of 30% acetic acid or 1% toluidine blue) and biopsy of these areas done (4). In this patient biopsy was done and histology confirmed squamous cell carcinoma.

The primary treatment for cancer of the vulva is surgical excision. Radical vulvectomy and bilateral inguinal lymphadenectomy involves a wide excision of the entire vulva and

mo[P]s pubis as well as block dissection and removal of inguinal and femoral nodes on both sides. This is essential even though the lesion is unilateral because the lymphatics of the vulva communicate freely from one side to the other (8). Limited surgery such as simple vulvectomy leads to poor results (4).

The patient presented was done radical vulvectomy with bilateral lymphadenectomy.

Radical vulvectomy leaves a large surgical defect that is associated with marked disfigurement of the genital area. Other complications include increased risk of venous thromboembolism, chronic leg oedema (lymphoedema), high rate of wound complications, psychosexual effects, urinary and stool incontinence and pelvic relaxation which can lead to rectocele and cystocele (1). In this patient the wound became septic and she was done skin grafting.

Use of radiotherapy for carcinoma of the vulva remains controversial but may be the only option when the patient presents with unresectable disease. Morbidity is high and is related to radionecrosis of the tissues (1, 4). Studies have been done, which confirm that radiotherapy can be used to cause tumour regression to a point where a more limited resection can be undertaken with sparing of organ function and improved quality of life (12).

The status of groin lymph nodes is already the most important prognostic factor for patients with invasive squamous cell carcinoma of the vulva (1).Homesley and colleagues in a study found that 65.5% of the patients had negative groin lymphnode involvement and of these the five—year survival was 90.9%. They found that 34.5% had positive lymphnode involvement and of these the five year survival was 57.7% (13).

Other prognostic factors include tumour diameter, tumour thickness, tumour differentiation, presence of vascular invasion and pattern of tumour growth. Low risk cases are stage I patients and stage II patients with tumour thickness up to 5 mm. High-

risk patients are stage II patients with tumour thickness greater than 5 mm and stages III and IV (5).

The patient was in the high risk group because she had stage III disease.

Of the patients with squamous cell carcinoma of the vulva, 15-40 % develop recurrence after treatment (1,2). The incidence of recurrence depends on original stage of the disease depth of invasion and regional lymphnode status. It is managed by resection and radiotherapy (1).

Careful follow—up of the patients should extend over the remaining years of the patient's life in order to detect recurrence early and treating it aggressively. All patients should be examined every three months for two years and every six months thereafter (1).

REFERENCES

1. Hoffman M.S, Cavanagh D. Malignacies of the vulva In : Te Linde's Operative Gynaecology 8th ed Lippincott — Raven Publishers pg 13') 1-1383 1997

2. Barclay D.L et al.Premalignant & Malignant disorders of the vulva and vagina In : Current Obstetric & Gynecologic Diagnosis & Treatment 9'11 ed McGraw- Hill Chp 46 pg 879— 893 2003

3. Kaguta T.K. A 10 year review of carcinoma of the vulva as seen at KNH. M. Med. Thesis UON 1984

4. Peel K.P. Malignant disease of the vulva and vagina In : Dewhurst's Textbook of Obstetrics and Gynaecology for Postgraduates 4'h ed Oxford : Blackwell Scientific Publishers pa 755 — 765 1988

5. Ridley C.M, Buckley C.H, Fox H. Epithelial neoplasms of the vulva In : Haines and Taylor Obstetric and Gynaecologic Pathology 4th ed Churchill Livin[g]stone pg 81-96 1994.

1

6. Cavanagh D, Fiorica J, Hoffman M.S. Invasive carcinoma of the vulva Changing trends in surgical management: Am. J Obstet Gynaecol 163;1007:1990

7. Kaguta T.K, Orero S.0, Rogo K.O. Carcinoma of the vulva in Kenyans East Afri. Med J 1988

8. Tindall V.R. Tumours of the vulva In : Jeffcoate's Principles of Gynaecology

9. Fiorica J.K., Cavanagh D, Roberts W.C. Carcinoma in-situ of the vulva : 24 years experience.

10. Jones R.W, Rowan D.M. Vulvar intraepithelial neoplasia III : A clinical study of the outcome in 113 caseswith relation to the later development of invasive carcinoma of the vulva. Obstet Gynaecol 84 : 741 1994

11. Shepherd J.H. Cervical and vulva cancer. Changes in FIGO definitions of staging. Bri. J Obstet Gynaecol 103 : 405 1996

12. Kotmensh J. Rubin S. J. Sutton H.G. Preoperative radiotheraphy followed by radical vulvectomy with inguinal lymphadenectomy for advanced carcinoma of the vulva. Gynae Oncol 36 : 181 1990

13. Homesley H.D. Bundy B.N. Seblis A. Prognostic factors for groin node metastates in squamous cell carcinoma of the vulva : A Gynaecologic oncology group study. Oncol 49 : 279 1993

GYNAECOLOGY CASE NO 7

PELVIC ABSCESS — LAPARATOMY AND DRAINAGE

NAME	**D. M.**	
IP NO	1085278	AGE 20 Years
DOD	13/04/06	**DOD** 20/04/06

HISTORY OF PRESENTING ILLNESS

Patient had an abortion at 28 weeks gestation on 31/03/06 — was induced with prostaglandin pessaries then put on oxytocin drip then delivered two days later at a private clinic in Kabete. There after she was discharged home on antibiotics. Three days later started vomiting, about 4 times per day associated with abdominal pains and fever. There was history of pus like per vaginal discharge and reduced bowel movements.

PAST MEDICAL HISTORY

She had been admitted to Equator hospital with depression in March 2005 due to some family issues.

OBS / GYN HISTORY: Menarche was at 12 years, her menstrual lasts about 21 days with a flow of 6 to 10 days which is irregular. Her LMP was on 31/07/05.There was no history of contraceptive use She has one previous scar — Emergency C/S in 2004 due to foetal distress.

FAMILY SOCIAL HISTORY: She is single, the last born in a family of four, currently a second year tourism student at Moi University. Her mother is hypertensive.

On examination,

She was found a sick looking obese patient, not pale or jaundiced. Vital signs: BP 110/60 mmHg, PR 82/min Weight 105Kg. Temp 37.2° C

ABDOMINAL EXAM

Abdomen was distended, moving with respiration with marked tenderness over suprapubic region. She had a healed sub umbilical midline incision scar.

VAGINAL EXAM: There was normal external genitalia, cervix was central 2cm dilated, with marked bilateral adnexal tenderness and fullness in the Pouch of Douglas. There was a foul smelling brownish discharge on the examining finger.

Abdominal pelvic ultrasound scan: Showed a normal liver parenchyma and echo-pattern. Spleen, pancreas and both kidneys were normal in size and texture. There is a cystic mass seen with debris above the uterus measuring 13x6x7.07cm. Slight fluid accumulation is seen in the pouch of Douglas: Features suggestive of Pelvic Abscess. An impression of post abortal pelvic abscess was made.

Management; The patient was informed about the diagnosis and the need for her to under go an emergency laparatomy. A written consent was taken from her. An intravenous line was established, blood taken for grouping and cross matching; two units of blood were preserved for her. She was started on an infusion of 5% dextrose and was to remain starved. Half an hour before theatre she was pre-medicated with Atropine 0.6 mg stat, then wheeled to theatre. Intra-operatively found an abscess cavity covering the anterior and superior aspects of the uterus containing about 300mls of pus extending up to the lower uterine segment with omentum covering it. Fallopian tubes, ovaries and bladder were normal. There was a bulky uterus about 12 weeks, grossly normal in appearance. Gut, omentum and uterus were matted together but were easily separated. Para-colic gutters and the intestines were normal.

Drainage of the abscess cavity, noted a perforation on the left lateral extend of the previous uterine transverse incision, peritoneal lavage done with 1.5 litres of warm normal saline mixed with Rifocin. The margins of perforated incision sited were freshened and repaired with catgut 2/0. A corrugated drain was left in situ. Mass closure of abdominal wall was done.

Post operative care.

Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mls 4 hourly for the first 24 hours. She was also on intravenous Zinacef 750 mg and Flagyl 500mgs 8 hourly for five days. She was also on intramuscular Diclofenac 75mg 8 hourly for pain control. On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet. The drain was removed on the third post operative day. Post-operatively patient did well and was discharged home on 6th post operative day on Augmentin 625 mg twice daily for 5 days .

Follow-up

She was given an appointment for review at G.O.P.0 in two weeks time but did not show up.

DISCUSSION

The patient presented was a 20 year old Para 1+1 who had a pelvic abscess as a complication of a procured criminal abortion. She underwent a laparatomy for the drainage of the abscess and did well post operatively.

A pelvic abscess is a collection of pus in the pelvic cavity 1 It is a complication of a pelvic infection. Most cases are due to pelvic inflammatory disease that often is secondary to sexually transmitted illness. Pelvic abscess may also follow abortion sepsis

or puerperal sepsis 3. In some cases infection follow gynaecologic surgery and procedures like intrauterine device insertion and hysterosalpingography (HSG). The abscess in the case presented resulted from induced criminal abortion.

In Kenya criminal abortion is illegal yet the rates of induced abortions are on the rise. Abortion was found by Aggarwal [4] to be the leading indication for admission to the Kenyatta National Hospital acute gynaecology wards and accounted for 60% of admissions.

62.3% of these were induced abortions and were common in single and adolescent girls. At the same hospital Kizza [5] found a 5.4 % infection rate following manual vacuum aspiration (MVA) for incomplete abortion. Aggarwal reported pelvic abscess to complicate 20% of all the cases of induced abortion [4]. Abortion therefore remains a major cause of morbidity and mortality in the female patients. Makokha 6 reported that 22% of maternal deaths at KNH were as a result of complications of abortion. The patient presented was a 20 year old single unemployed lady, who had procured a criminal abortion for her first pregnancy.

Organisms commonly isolated from pelvic abscesses are enterric and anaerobic bacteria like Bacteroides and Escherichia species, Gonococci and Chlamydia. Though common cause of pelvic infection are rarely isolated in pelvic abscesses [2,7,8] Staphylococci, Streptococci and Chlostridia may be found. Atypical organisms are isolated in patients with immunosupression. Cultures in our patient grew E.coli and Klebsiella species. An abscess should be suspected if constitutional symptoms develop or persist after treatment for pelvic inflammatory disease, following an abortion or after delivery, after gynaecologic surgery or procedure. But even a large abscess may be asymptomatic. Symptoms include lower abdominal pains radiating to the back and thighs, fever, general malaise, and lassitude, and vaginal discharge. In some patients, dysuria, frequency of micturition diarrhoea, painful defecation and vomiting may occur . Our patient presented with severe lower abdominal pains, chills, fever, and general malaise and vaginal discharge following a criminal abortion. Examination reveals a sick patient who may be febrile, pale and jaundiced. The abdomen will be tender; there may be guarding and rebound tenderness. Often there is tender abdominal mass. Cervical excitation test is positive, the adnexae are tender and the pouch of Douglas will be full. The white blood cell count is often raised [1,2,3,9] The patient had the above features except jaundice. Her white cell count was $18x10^{9}/L$ with a neutophilia of 90% and the heamoglobin level was only 4.0g/dl. Paracentesis and the culdocentesis when performed may reveal pus. Paracentesis in our patient was negative for pus.

Diagnosis of pelvic abscess therefore is clinical but ultrasonography (US) and Computerized Tomography scan (CT scan) may aid with diagnosis. Laparoscopy may also be employed for direct visualization of the abscess but it carries risk of injury to viscera in case of adhesions [2, 9]. These diagnostic methods are available at KNH, but only U/S is used in our unit. In the case presented diagnosis was clinical, and was confirmed by U/S.

Patients with pelvic abscess require admission. Resuscitation with fluid and electrolyte replacement and blood transfusion may be necessary. Our patient was given intravenous fluids and was transfused 3 units of whole blood. These patients in addition should be started on broad spectrum intravenous antibiotics to cover for both aerobes and anerobes [9,10]. At KNH Formulu reported 90% sensitivity to tetracycline either alone or in combination with gentamycin and metronidazole . Our patient was initially started on a combination of intravenous crystalline penicillin, gentamycin and flagyl. On the secondday she was put on i.v augumentin and flagyl. Culture results showed E. coli and Klebsiella spp sensitive to Rocephin and Amikacin and these were substituted. She did well on this treatment despite the initial wound sepsis and was allowed home on the 140' post operative day.

In some centres a conservative medical approach is practiced and surgery only performed for deteriorating condition, rupture of the abscess, failure to respond in 24-48 hours or in case of recurrence. At KNH laparotomy and drainage as in the case presented is the management of choice. At operation, the uterus and pelvic viscera are inspected thoroughly for injury, the pelvic cavity washed adequately and a drain left in situ 9,10,11

In the past, total abdominal hysterectomy (TAH) and bilateral salpingo-oophorectomy (BSO) were done at laparotomy. Currently a more conservative approach to preserve ovarian function and fertility is practiced where possible. TAH and BSO is reserved for chronic recurrent cases and in cases where fertility is not desired or of poor prospects. TAH and BSO is not routine at

KNH and the patient presented only had drainage of pus done.

In some countries percuteneous drainage and culdotomy through the vagina are practiced. It is reported that only 15-84% of abscesses are fully drained by these procedures and 1/3 will need further laparotomy [9, 10, 11]

Various complications are associated with pelvic abscess. Rupture could lead to peritonitis and endotoxic shock. There are inherent surgical risks at laparotomy; long term complications include chronic pelvic pains, bowel obstruction, infertility, chronic dyspareunia and predisposition to ectopic pregnancy. Prognosis is good in those with early and adequate intervention and in cases where the abscess is well localized 1, 2, 3

Prevention of this condition requires adequate health education, provision of effective contraceptive services, control of and adequate treatment of sexually transmitted illness, effective management of abortion, aseptic delivery procedures and proper gynaecologic surgical procedures. Our patient had had unsafe backstreet abortion hence developed pelvic abscess as a complication.

REFERENCES

1. Rock A.J., Thompson J.P. Pelvic Inflammatory Disease. Te Linde's Operative Gynaecology. 8h Ed, Lippincot-Raven Publishers, Philadeliphia, 1997: 657

2. Hemsell D., Wevdel G.B., Micical A. STD and Pelvic Infection. Current Obst & Gyn Diagnosis And Treatment. 9h Ed .Appleton And Lange, connectcut USA, 2001: 770798

3. Whitfield C.R. Pelvic Infection. Dewhurst Textbook of Obs & Gyn for Postgraduates, Blackwell Scientific Publications London 4' Ed 1986; 596

4. Aggarwal S.P., Mati J.K.G. Epidemiology of Induced Abortion in Nairobi Kenya. J Obs Gyn East Central Africa, 1982: 1; 54

5. Kizza A.P.M. Incomplete Abortion Treated with Kermans Canula & syringe, 1999:68-78

6. Makokha A.E. Maternal Mortality at Kenyatta Hospital 1972-1977. E.A. Med J, 1980:57(7); 451

7. Chow A.W et al. The Bacteriology of Acute Pelvis Inflammatory Disease; Value of cul-de sac cultures and Relative Importance of Gonococci, Aerobic and Anaerobic Bacteria. Am J Obs Gyn (1975); 122; 876

8. Formulu J.N. Bacteriological Sensitivity Pattern in Septic Abortion, Post Abortal Sepsis and Pelvic Abscess in KNH. MMed Thesis UoN 1981

9. Landers D.V., Sweet R.I. Current Trends in the Diagnosis and Treatment of Tubo Ovarian Abcess. AM J Obs Gyn, 2001: 151; 1098

10. Walker C.K., Landers D.U. Pelvic Abscesses; New Trends in Management. Obs Gyn SU (1999) 46; 1001-1062.

GYNAECOLOGY CASE 8

BLIGHTED OVUM — DILATATION AND CURRETAGE.

NAME T. W	DOA	18/5/07
AGE 30 YEARS	DOD	18/5/07
PARITY 1 + 0		

PRESENTING COMPLAINT

T.W presented as a referral from a private clinic with a diagnosis of missed abortion.

HISTORY OF PRESENTING COMPLAINTS

T. W was otherwise well and had her last menstrual period on 31/1/04. She had not done any pregnancy test however. One week prior to admission she visited a private clinic in Nairobi to start her antenatal clinic. She was attended to, and a pelvic scan requested to confirm the pregnancy. To her dismay, the pelvic scan revealed that she had a blighted ovum and was referred to KNH for evacuation.

OBSTETRIC AND GYNECOLOGY HISTORY

T.W was now Para 1 + 0, her last delivery having being 10 years earlier while still in school. The delivery was a spontaneous vertex delivery (SVD), and her son was alive and well.

Her menarche was at 15 years, and her menses were regular, lasting 4 —5 days, and occurring every 28 — 30 days. There was no associated dysmenorrhoea. She had used oral contraceptive pill for over 10 years, having stopped using them about 1 year earlier in order to get a child. She had never had a pap smear done.

PAST MEDICAL HISTORY

She had been admitted about 2 year earlier with malaria and discharged after 3 days. She had never had any surgical operation.

FAMILY AND SOCIAL HISTORY

She was now married for about one and a half years and lived with her husband in Kayole. Both were business people. There were no known chronic ailments in their family.

PHISIAL EXAMINATION

She was a young lady in good condition and well nourished. She did not have pallor, Jaundice,

edema or lymphadenopathy. Her vital signs were normal with a blood pressure of 110170 mmhg; pulse of 74/min; respiratory rate of 15/min and a temperature of 36.7°c.

ABDOMINAL EXAMINATION

The abdomen was scaphoid, non-tender and moving with respiration. There were no masses palpable or any organomegaly.

PELVIC EXAMINATION

There were normal external genitalia. The cervix was posterior, long, firm consistency and closed. The uterus was about 8 weeks. The adnexae and pouch of Douglas were free. There was no blood or abnormal discharge on the examining finger.

PELVIC SCAN

A pelvic scan done at Nairobi hospital showed an empty gestational sac with no fetal pole. The radiologist had concluded that it was a blighted ovum.

MANAGEMENT

The patient was told of the management of the condition. Informed consent was obtained and blood drawn for hemogram, urea and electrolytes.

RESULTS OF INVESTIGATIONS

- Hemogram - Hb 12.8 g/dl - Platelets 356 x 109/1-
- Urea 4.8 mmol/L-. creatinine 68 micromoles/ L. Na^+ 138 mmol/L; K^+ 4.3 mmol/L.

PROCEDURE.

The patient we premedicated with I.M atropine 0.6 mg Y2hour before being wheeled to theatre.

In theatre she was put under general anaesthesia and repositioned to the lithotomy position. Vulvalvaginal toilet was done with chlorohexidine solution and patient draped. Bimanual examination revealed an anterverted uterus about 8 weeks in size. A Sims speculum was introduced into the vagina and the anterior lip of the exposed cervix grasped with a

tenaculum. The cervix was drawn and cleaned with Betadine.

The cervix was gradually dilated with metal Hegar's dilators; till no 10 Hegar's dilator could pass easily. A curette was then introduced into the uterine cavity, and the products of conception scraped away. About 100cc of POC's were evacuated. Once a grating sound was heard, the instruments were removed, patient cleaned and anesthesia successfully reversed.

POST – OPERATIVE CARE

Once awake in the ward, the patient was allowed to eat normally. She was allowed home on Doxycycline 100mg twice daily and Metronidazole 500mg three times a day for one week. Since she wanted a baby, she declined any contraceptive advice.

DISCUSSION

The patient presented was a 30-year-old Para 1 + 0, diagnosed to have a blighted ovum by ultrasound. She underwent dilatation and curettage under general anaesthesia with no complications. Specific anomalies are not readily detected in early embryos but generally disorganized growth is recognized. The vast majority (about 85%) of conceptuses spontaneously aborted in the first 8- 10 weeks of gestation and were abnormal. Among these growth-disorganized conceptuses is the one with a chorionic vesicle without an umbilical cord remnant or embryo commonly referred to as a "Blighted ovum", but which is better termed descriptively as an "empty chorionic vesicle" or "unembryonic sac" (1)

This term is probably a misnomer because the condition appears to arise after implantation as a result of the death or distortion of the embryonic disk after the trophoblast has differentiated. A high level of chromosomal abnormalities have been found in blighted ovum (2); compared with their embryonic counterparts, spontaneous abortion rates linked with blighted ova were 14% higher (67%); exhibited a much higher rate of trisomies (74%Vs 35%), especially in chromosomes 16 and 22; and totally lacked 45 X karyotype (2). It

was therefore speculated that arrest in early embryonic development might be correlated to genes on autosomal chromosomes, particularly 16 and 22.

Blighted oval survive for longer than biochemical pregnancies, usually 30 days or longer, or even through the majority of the first trimester. The patient may be a symptomatic or present with regression of the symptoms and signs of pregnancy. More often a patient has missed her menses and expects to be pregnant only for the pregnancy test to be negative. Falling or abnormally low plasma levels of, B HCG are predictive of an abnormal pregnancy, among them a blighted ovum.

On ultrasound, an abnormal gestational sac, without a yolk sac or embryo, is consistent with a blighted ovum (3). At abortion, no obvious fetal parts are found; and when examined microscopically, the tissue is entirely trophoblastic (2, 3).

REFERENCES

1. Kalousek, D.K, and Neave, C. Pathology of abortion, the embryo and the previable fetus. In: J.S Wigghesworth and D.13 singer (Eds). Textbook of fetal and perinatal pathology. Boston: Black well, 1991. Pg 141.
2. Ervin E. Jones. Abnormal ovulation and implantation. In: Medicine of the fetus and mother. 2nd ed. Edited by E. Albert and John Hobbins. Lippincott — Raven Publishers; Philadelphia, 1999.
3. Sara H. Garmeh. Early pregnancy risks. In: current obstetric and Gynecologic diagnosis & treatment chapter 14, Pg 274, 9th edition. The McGraw — Hill companies, 2003.

GYNAECOLOGY CASE 9

HYDATIDIFORM MOLE — SUCTION AND SHARP CURETTAGE

NAME:	Z. A
IP. NO:	0977963
D.O.A:	24/7/04
D.O.D:	3/7/04
AGE:	34 YEARS
PARITY:	2 + 2; 1 LIVING CHILD
WARD:	ID

PRESENTING COMPLAINTS

Z. A. presented with vomiting, anorexia and vaginal bleeding after manual vacuum aspiration for incomplete abortion at Kisii District Hospital.

HISTORY OF PRESENTING COMPLAINTS

Z.A. last menstrual period was on 24/4/04 and was pleased to be pregnant again. However in Mid June 2004, she developed persistent vaginal bleeding, and lower abdominal pain, and was seen at Kisii District Hospital where a diagnosis of incomplete abortion was made and manual vacuum aspiration done. At that time the vomiting and nausea was said to be due to pregnancy. However about 2 weeks after MVA, the vaginal bleeding recurred, associated with nausea and vomiting. Soon thereafter she developed lower abdominal pain and swelling. She sought help at the KNH.

OBSTETRIC AND GYNECOLOGY HISTORY

Z.A. was now Para 2+2. Her first delivery was in the year 1994, by cesarean section because of cephalopelvic disproportion (CPD). The outcome was a life male infant who was her only living child. In 1997, she had a molar pregnancy diagnosed at the KNH, where evacuation was done. She was however lost to follow up. In 1999 she had her 3rd pregnancy, which was complicated by preterm premature rapture of membranes (PPROM). A cesarean section was done but the baby succumbed to the complication of prematurity – respiratory distress syndrome. Her last

pregnancy was the abortion preceding her current admission which was evacuated at Kisii District Hospital.

Her menarche was at the age of 14 years, menses were regular, occurring at an interval of 26 days and lasting 3-5 days. She had never used any form of contraception. She had never had a pap smear done.

PAST MEDICAL HISTORY

This was non-contributory.

FAMILY AND SOCIAL HISTORY

She was married and lived mainly in Kisii. However she often visited her husband who was a hotel chef and lived in Dandora. She did not drink alcohol nor smoke cigarettes. There was no family history of chronic ailments.

PHYSICAL EXAMINATION

She was a middle-aged lady who was in fair general condition. She was however pale, but did not have jaundice, cyanosis, edema or lymphadenopathy. Her vital signs were a blood pressure of 110/70 mmHg, pulse of 80/minute, respiratory rate of 16/min and a temperature of 36.5°C.

ABDOMINAL EXAMINATION

The abdomen was distended in the supra pubic region. There was a midline sub-umbilical scar. There was tenderness in the lower abdomen. There was a pelvic mass, thought to be the uterus extending to the level of the umbilicus. It felt firm, was mobile from side to side, and was slightly tender. There were normal bowel sounds.

PELVIC EXAMINATION

There were normal external genitalia. The vaginal walls were also normal. The cervix was posterior, closed and long. The uterus was bulky to approximately 20 weeks equivalent and slightly tender. The Adnexae were free and the pouch of Douglas empty. Cervical

excitation test was negative. There was blood on the examining finger.

TENTATIVE DIAGNOSIS

A diagnosis of recurrent hydatidiform mole was made. A pelvic scan, serum B HCG Levels, full hemogram, urea, creatinine and electrolytes were ordered. A chest radiograph was also requested.

RESULTS OF INVESTIGATIONS

Pelvic scan – showed a non-gravid uterus, which was enlarged measuring 182 by 141 by 92 mm. It was seen as a huge almost homogenous mass giving the impression of a 'Snowstorm". No Adnexal masses were seen.

Serum B, HCG levels.	200 000 miu/ml Normal below 5MIU/ML.
Hemogram	WBC 4.7 X ^{106} /L
	RBC 3.54 X ^{106} /L
	Hb 9.5 g/dl
	Urea 12.1 mmol/l; creatinine 53 ummol/l
Chest x-ray	Normal

A diagnosis of recurrent H. mole was confirmed and patient informed of the diagnosis. The management was explained to her and informed consent obtained. Blood was drawn for blood group and cross-match. She was prepared for suction curettage in theatre. She received LM Atropine 0.6mg start.

PROCEDURE; In theatre, she was placed in lithotomy position and general anaesthesia induced. An oxytocin infusion of 40 I.0 was begun before the induction of anaesthesia. Vulvovaginal toilet was done, and the bladder aseptically catheterized. A speculum was introduced to expose the cervix, which was cleaned with Betadine. It was held with a tenaculum. A uterine sound was introduced but the cavity was too long. Progressive cervical dilatation was done using Hank's dilators. When the 8mm dilator was inserted there was a gush of blood from

the uterus. The cannula was introduced into the uterine cavity and connected to the vacuum apparatus. Within about 10 minutes the uterus had decreased dramatically in size. About 2.7 litres of blood and vesicles were evacuated. Bimanual palpation of the uterus was done to stimulate uterine contraction. The bleeding soon subsided and a vaginal pack was left. One unit of blood was transfused. Anaesthesia was reversed.

A day after the suction curettage, the patient was discharged home on antibiotics and hematinic Ranferon to come back for sharp curettage after 10 days.

On 10/8/04, she was taken back to theatre and gentle sharp curettage done. There was minimal residual tissue, which was sent for histology. The uterine size had regressed to about 8 weeks equivalent. She was discharged home on oral contraceptive pills and weekly review in the GOPC, with B HCG levels.

FOLLOW UP

On follow up, the serum, B HCG levels fell to undetectable levels by the 4th week. She was then scheduled for monthly follow up, with B HCG levels.

DISCUSSION

The patient discussed here was a 34-year-old Para 2+2, who had hydatidiform mole in 1997, and presented, yet again, with another mole after an abortion at about 9 weeks. She underwent suction and sharp curettage.

Hydatidiform mole belongs to a spectrum of pregnancy related trophoblastic tumors. It comprises of abnormal proliferation of the syncyti otrophob last and abnormal replacement of normal placental trophoblastic villi by hydropic placental villi. The grapevine vesicles fill and distend the uterus (1). The incidences of molar pregnancies vary in different parts of the world. The incidence in Europe and North America has been reported

to be between 0.6 and 1.1 per 1000 pregnancies (2), compared to 2 per 1000 pregnancies in Japan.

Case control studies have identified how dietary intake of carotene and thus vitamin A to be associated with complete molar pregnancy (3). Dietary factors may therefore explain regional variations in the incidence of molar pregnancy. This may also be related to socioeconomic factors. Maternal age older than 35 years has consistently been shown to be a risk factor for complete moles. Ova from older women are thought to be more susceptible to abnormal fertilization. In one study, the risk of a complete mole was found to be increased 2.0 fold for women older than 35 years, and 7 — 5 fold for women older than 40 years (4). Limited information is available concerning the risk factors for partial molar pregnancy. However, these have been shown to differ. For instance, there is no association between maternal age and risk of partial mole (4). The risk of partial mole has been reported to be associated with the use of oral contraceptives and a history of irregular menstruation, but not with dietary factors (5). Hydatidiform mole may be categorized as either complete or partial moles on the basis of gross morphology, histopathology, and karyotype (1). In histopathology, complete moles lack identifiable embryonic or fetal tissues and the chorionic villi exhibits generalized hydatidiform swelling and diffuse trophoblastic hyperplasia. Cytogenetic studies have demonstrated that complete hydatidiform moles usually have a 46, XX karyotype, and the molar chromosomes are entirely paternal in origin (6). It appears that complete moles usually arise from an ovum that has been fertilized by a haploid sperm, which then duplicates its own chromosomes. The ovum nucleus may either be absent or inactivated (7). Although most complete moles have a 46, XX chromosomal pattern, about 10% have a 46 XY karyotype (8). Chromosomes in a 46, XY complete mole also appear to be entirely of paternal origin, although mitochondria) DNA is of maternal origin (9).

Partial hydatidiform moles are characterized by the following pathologic features (10): Chorionic villi of varying size with focal hydatidiform swelling, cavitations and trophoblastic hyperplasia; marked vinous scalloping: prominent stromal trophoblastic inclusions, and identifiable embryonic or fetal tissues. Partial moles are generally triploid

in karyotype (69 chromosomes); the extra haploid set of chromosomes usually being derived from the father (11). The patient discussed had a compete hydatidiform mole for the $_2$na time in her life, having had a similar condition 6 years earlier (1997).

The classical and current clinical features of a complete mole include:

- Vaginal bleeding — most common symptom. Currently reported to occur in 84% of patients (12). Because vaginal bleeding may be considered and prolonged, about one half of these patients have anemia (Hb <10.Ogldl).
- Excessive uterine size — excessive uterine enlargement relative to gestational age is one of the classic signs of a complete mole, although its present in only half of the patients.
- Preeclampsia — preeclampsia almost exclusively develops in patients with excessive uterine size and markedly elevated HCG levels. Hydatidiform mole should always be considered whenever preeclampsia develops early in pregnancy.
- Hyperemesis gravidarum — This may lead to electrolyte disturbances which require treatment with parenteral fluids.
- Hyperthyroidism — This develops almost exclusively in patients with very high HCG levels. Some investigators have suggested that HCG is the thyroid stimulator in women with GTD; because positive correlations between serum HCG and total T3 or T4 concentrations have been observed.
- Other rarer presentations include trophoblastic embolization and Theca Lutein ovarian cysts. The former may lead to respiratory distress characterized by chest pain, dyspnoea, tachynea, and tachycardia. The patient discussed presented with vaginal bleeding, excessive uterine enlargement and hyperemesis gravidarum.

Patients with partial Hydatidiform mole usually do not have the dramatic clinical features characteristic of complete molar pregnancy. In general, these patients have the signs and symptoms of incomplete or missed abortion, and partial mole can be diagnosed after histologic review of the tissue obtained by curettage (13).

Ultrasonography is a reliable and sensitive technique for the diagnosis of complete molar pregnancy. Because the chorionic villi exhibit diffuse hydropic swelling, complete moles

produce a characteristic vesicular, ultrasonographic pattern, the so called "snow storm". This was found in the ultrasound done in the patient discussed. Ultrasound may also contribute to the diagnosis of partial molar pregnancy, by demonstrating focal cystic spaces in the placental tissues and an increase in the transverse diameter of the gestational sac (14). When both of these criteria are present, the positive predictive value for partial mole is 90%.

After molar pregnancy is diagnosed, the patient should be evaluated carefully for the presence of associated medical complications, including pre eclampsia, hyperthyroidism, electrolyte imbalance, and anemia. After the patients' condition has been stabilized, a decision must be made concerning the most appropriate method of evacuation.

If the patient desires surgical sterilization, a hysterectomy may be performed with the mole in situ. The ovaries may be preserved at the time of surgery, even though prominent theca lutein cysts are present. They may be decompressed by aspiration. Hysterectomy does not prevent metastasis. Therefore, patients still require follow — up with assessment of HCG levels.

Suction curettage is the preferred method of evacuation, regardless of uterine size of the patient Our patient underwent suction curettage. The use of prophylactic chemotherapy at the time of molar evacuation is controversial. However in a prospective study in the United States, It was shown to prevent metastasis and reduce the incidence and morbidity of local uterine invasion (15). Indeed, the author recommends that prophylaxis may be especially useful in the management of high-risk complete molar pregnancy, especially where hormonal follow-up is unavailable or unreliable. After molar evacuation, patients should be monitored with weekly determinations of the R, sub unit of HCG levels, until they are normal for three consecutive weeks, followed by monthly levels until normal for six consecutive months. At the completion of follow up, pregnancy may be undertaken.

Patients are encouraged to use effective contraception during the entire interval of HCG follow – up. If the patient does not desire sterilization, the choice is to use oral contraceptives

or barrier methods

1. Palmer J R. Advances in Epidemiology of Gestational Trophoblastic Disease. J Rep. Med 1994, 39: 155 – 162.
2. Berkowitz RS, Cramer D W, Bernstein MR, et al. Risk factors for complete molar pregnancy from a case – control study. AM. J. Obstet. Gynecol. 1985; 52: 1016 -1020
3. Perazzini F, La Vecchia C, Pampallona S. Parental age and risk of complete and partial Hydatidiform mole. Br J. Obstet. Gynecol. 1986: 93: 582 – 585.
4. Berkowitz RS, Bernstein MR, Harlow BL, et al. Case control study of risk factors for partial molar pregnancy. A. M. J. Obstet. Gynecol. 1995; 173: 788-794
5. Kajii T, Ohama K. Androgenic origin of hydatidiform mole. Nature 1977; 268: 633 –634
6. Yamashita K, Wake N, Araki T, et al. Human lymphocyte antigen expression in Hydatidiform mole: Androgenesis following fertilization by a haploid sperm. A. M. J. Obstet. Gynecol. 1979; 135: 597-600
7. Pattilio R A, Ssaki S, Katayama KP, et al. Genesis of 46 x y Hydatidiform mole. AM J Obstet. Gynecol 1981: 141: 104 – 105
8. Azuma C, Saji F, Tokugawa Y, et al. Application of gene amplification of polymerase chain reaction to genetic analysis of molar mitochondria) DNA: the detention of a nuclear empty ovum as the cause of complete mole. Gynecol. Oncol. 1991; 40: 29-33
10. Szulman A. E, Surti U. The syndromes of Hydatidiform mole. Cytogenetic and morphologic correlations. AM. J. Obstet. Gynecol. 1978: 131: 665 – 671
11. Lawler SD, Fisher RA, Dent J. A prospective genetic study of complete and partial Hydatidiform moles. AM. J. Obstet. Gynecol. 1991: 164: 1270 – 1277
12. Goldstein DP, Berkowitz RS. Current management of complete and partial molar pregnancy. J. Reprod. Med 1994: 39: 139-146
13. Szulman A. E., Surti U. The clinicopathologic profile of the partial Hydatidiform mole. Obstet gynecol 1982; 59: 597 – 602

14.Fine C, Bundy AL, Berkowitz RS, et al. Sonographic diagnosis of partial Hydatidiform mole. Obstet. Gynecol. 1989; 73: 414 – 418

15.Kim DS, Moon H, Kim KT et al. Effects of prophylactic chemotherapy for persistent trophoblastic disease in patients with complete Hydatidiform mole. Obstet. Gynecol. 1986; 67: 690-694

GYNAECOLOGY CASE NO 10

OVARIAN CANCER STAGE IIIB – TOTAL ABDOMINAL HYSTERECTOMY, UNILATERAL SALPINGO-OOPHORECTOMY, RADIOTHERAPY AND CHEMOTHERAPY

NAME: J. C. **AGE:** 28 YEARS

IP NO : 1009234 D.O.A: 18/02/05 **DOD:** 15/03/05

28 year old Para 4+0 LD 2000, referred from Maua Hospital with C/o

Abdominal distension, Constipation and Weight loss x 1 year

HISTORY OF PRESENTING ILLNESS

J.0 gave history of progressively increasing abdominal distension for the last one year which was associated with anorexia and generalized body weakness with no diarrhea, vomiting or urinary symptoms. There was marked constipation with associated weight loss. She had been amenorrhoeic for the last six months.

PAST MEDICAL HISTORY

Had been admitted to Mau Hospital for two times, during which she underwent laparatomies – in 2001 and 2003, however there was no histology report or a summary of infra-operative findings which was available.

OBS / GYN HISTORY

Menarche was at 15 years, her menstrual cycle lasts 28 days with a menstrual flow of 4 days and is regular. She had used DMPA for 11/2 years since 2000.Para 4+0, LD 2000 with 4 living children.

FAMILY SOCIAL HISTORY

She is a housewife, while the husband is a farmer. She does not smoke or take alcohol. There is no chronic illness in the family.

EXAMINATION, She was sick looking, not pale, jaundiced, or cyanosed. Vital signs

ABDOMINAL EXAM: Markedly distended, moving with respiration. Sub umbilical mid line incision scar noted, there was a tender mass on right hypochondrium.

Abdomino-Pelvic ultrasound Scan done on 18/02/05 – showed a large multi-lobulated mixed echo-pattern mass arising from the pelvis and extending up to the epigastric region. The mass exceeds 17 cm in its greatest dimension. The uterus is normal in site and size and shows no myometrial lesion. Both ovaries cannot be identified. There is fluid collection noted around the uterus.

Liver, gall bladder, and both kidneys were normal. The pancreas and pars-aortic regions could not be identified due to a massive abdomino-pelvic mass. Ascites is demonstrated. Conclusion: Massive infra-abdominal mass of ?ovarian origin with associated ascites TBC – Hb 12.5 gm/dl, WBC 7.9X10^9/L, Plat 305x109/L

U/E/C – Na 147 K4.4 Cl 110 U 3.3 Creat 60

LFTS – T prot 57 Alb 30 T Bil 3.4 D Bil 2.5 AST 22 ALT 13 ALKP 34.2 yGT 21 An impression of Cancer of the Ovary was made.

Management.

The patient was informed about the diagnosis and the need for her to under go a laparatomy. A written consent was taken from her. Blood was taken for grouping and cross matching; two units of blood were preserved for her. Half an hour before theatre she was pre-medicated with Atropine 0.6 mg star, then wheeled to theatre.

Infra-operatively on 24/02/05 a diagnosis of Cancer of the Ovary stage III B was made. Found a grossly huge and well vascularised tumour mass covering the abdominal cavity arising from the pelvis, it was peeled off from the abdominal & pelvic organs (huge tumour about I Okgs removed)

There was a normal right tube, ovary & uterus noted. Left tube and ovary were missing. There was a bloody ascitic fluid noted. Multiple tumour seedlings were noted on anterior abdominal wall. There was a grossly normal liver in appearance but with enlarged gall bladder.

Did: Total abdominal hysterectomy with unilateral salpingo–oophorectomy, inferior omentectomy & adhesionolysis.

Post operative care.

Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mls 4 hourly for the first 24 hours. She was also on intravenous Crystalline penicillin 2mu 6 hourly and Gentamycin Wing 8 hourly for 48 hours then oral Amoxil and Flagyl for five days. She was also on intramuscular Diclofenac 75mg 8 hourly for pain control. On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet.

Histology exam of the specimen showed a high grade fibrosarcoma. She was reviewed by a Radio-oncologist who advised that patient was to receive 25 courses of radiotherapy then chemotherapy. This was done daily for 25 consecutive days.

J. C. was readmitted on 17/04/05 for chemotherapy: Cisplatin 100mg & Adriamycin 60 mg stat and cyclophosphamide 500 mg once daily for five days, after every three weeks for six courses.

DISCUSSION

The patient presented was a 28 year old multiparous woman who presented with advanced carcinoma of the ovary. She had laparotomy with total abdominal hysterectomy, unilateral salpingo-oophorectomy and inferior omentectomy and was put on 25 courses of radiotherapy then later started on chemotherapy with Cisplatin, Adriamycin and Cyclophosphamide.

Ovarian cancer is the seventh most common malignancy worldwide. It is the fifth most common form of cancer in American women. In the United States it is the leading cause of death from any gynaecologic malignancy. Ovarian cancer has the highest fatality-tocase ratio of all the gynaecologic malignancies. For women in the United States, lifetime risk of developing the disease is approximately 1 in 70 or 1.4%. (1,1,1,4)

The causes of ovarian cancer are poorly understood, but several factors have been associated with an increased or decreased risk of the disease. Age over 40 years, white race, nulliparity, infertility, history of endometrial or breast cancer, and family history of ovarian cancer consistently have been found to increase the risk for invasive epithelial cancer. Parity, oral contraceptive use, history of breastfeeding, tubal ligation, and hysterectomy has been

associated with decreased risk of ovarian cancer. (1, 2, 3, 4) An association between ovarian cancer and a family history of ovarian cancer and other malignancies including breast, endometrial, colon and prostate cancer has been reported in the literature. Three main hypotheses have been proposed to explain the pathogenesis of ovarian cancer; the incessant ovulation, gonadotrophin and pelvic contamination theories. (1,2,3)

- The incessant ovulation hypothesis postulates that repeated minor trauma to the epithelial surface of the ovary caused by continuous ovulation increases the likelihood of ovarian cancer.
- The gonadotrophin hypothesis postulates that exposure of the ovary to continuously high levels of circulating gonadotrophins increases the risk of malignancy.

- The pelvic contamination theory suggests that carcinogens may come into contact with the ovary after passing through the genital tract.

Ovarian cancer may be divided into three major categories based on the cell type or origin; epithelial, germ cell and sex cord and stromal. The ovary may also be the site of metastatic disease by primary cancer from another organ sites.

- Epithelial neoplasms are derived from the ovarian surface mesothelial cells and include six cell types. Serous, mucinous, endometrioid, clear cell, transitional cell and undifferentiated.
- Germ cell neoplasms arise from the germ cell elements of the ovary and include dysgerminoma, endodermal sinus tumour, choriocarcinoma, teratoma, polyembryoma and mixed germ cell tumours.
- Sex cord and stromal neoplasms include granulosa cell tumour fibroma, thecoma, sertoli leydig cell and gynadroblastoma.
- Neoplasms metastatic to the ovary commonly arise from the breast, colon, stomach, endometrium or lymphoma. ti, 2,4)

Epithelial ovarian cancer accounts for 90% of all cases of ovarian cancer. The tumours are derived from the coelomic epithelial or mesothelium Ovarian cancer typically

develops as an insidious disease, with few warning signs or symptoms. Most neoplastic ovarian tumours produce few symptoms until the disease is widely disseminated throughout the abdominal cavity. A history of non-specific complaints, including nausea, dyspepsia and altered bowel habits, is particularly common. Early satiety and abdominal distension as a result of ascites are generally signs of advanced disease. A change in bowel habits, such as constipation and decreased stool calibre, is occasionally noted. Large tumour may cause a sensation of pelvic weight or pressure. Rarely, an ovarian tumour may become incarcerated in the cul-de-sac and cause severe pain, urinary retention, rectal discomfort, and bowel obstruction [9]. Abnormal vaginal bleeding may occur in patients with ovarian cancer in the presence of synchronous endometrial carcinoma. Granulosa theca cell tumours are classically oestrogen-producing tumours The most important sign of epithelial ovarian cancer is the presence of a pelvic mass on physical examination. A solid, irregular, fixed pelvic mass is highly suggestive of an ovarian malignancy. Ascites or malignant pleural effusion may also be present.

Postmenopausal woman are at greatest risk for developing a pelvic mass that subsequently proves to be a malignant ovarian neoplasm. The reproductive age woman is more likely to have functional ovarian cyst or endometriosis.

The work-up should involve physical examination, radiographic evaluation, ultrasound, CT scan, MRI, and laboratory evaluation. Ultrasonography is the most important radiographic test to evaluate an adnexal mass. Malignant masses are likely to be solid, with septations, bilateral and to have ascites [2].CA 125 is an antigen expressed by 80% of non mucinous epithelial ovarian cancers. A level greater than 35 U/ml is considered abnormal. In premenopausal women, however, CA 125 levels may also be elevated in a number of benign conditions, including pelvic inflammatory disease, endometriosis, pregnancy and hemorrhagic ovarian cysts. In addition, approximately 50% of women with early ovarian cancer have a normal CA 125 level (". Other markers for ovarian causes include CA 19-9, CA 15-3, OVXI, Her -2/neu and human chronic gonadotrophin (HCG) [0]-Surgery is the cornerstone of therapy for ovarian cancer. For a patient suspected of having ovarian cancer, primary surgery accomplishes the following goals, (a) confirmation of the diagnosis of ovarian cancer, (b) Precise determination of extent of disease; (c) maximum cytoreductive surgery in patients with

advanced stage disease. Procedures in the surgical staging of ovarian cancer include the following:

- Samples of ascites or peritoneal washings from the pars colic gutters and pelvic and sub diaphragmatic surface for cytology.
- Complete abdominal exploration
- Intact removal of tumour
- Hysterectomy
- Infra colic omentectomy
- Biopsies of abdominal peritoneal implants; if present random biopsies from the paracolic gutter peritoneum, pelvic peritoneum, and right sub diaphragmatic peritoneal surface.

- Pelvic and pars-aortic lymph node biopsies
- Cytoreductive surgery to remove all visible disease.

Ovarian cancer is surgically staged according to the staging system developed by the International Federation of Gynaecology and Obstetrics (FIGO).1-5

Stage I: Growth limited to the ovaries

IAGrowth limited to one ovary: no ascites, no tumour on the external surfaces; capsule intact.

IB Growth limited to both ovaries: no ascites, no tumour on the external surface, Capsule intact

IC Tumour either stage I A or IB but with tumour on surface of one or both ovaries; or with capsule ruptured; or with ascites present containing malignant cells or with positive peritoneal washings

Stage II: Growth involves one or both ovaries with pelvic extension

II A: Extension or metastases to the uterus or tubes

II B : Extension to other pelvic tissues

II C: Tumours either stage 11A or 11B, but with tumour on surface of one or

both ovaries, or with capsule ruptured; or with ascites present containing malignant cells, or with positive peritoneal washings.

Stage III: Tumour involves one or both ovaries with peritoneal implants outside the pelvis and/or positive retroperitoneal or inguinal nodes. Superficial liver metastases equal stage III. Tumour is limited to the true pelvis but with histologically proven malignant extension to small bowel or omentum.
111A: Tumour grossly limited to the true pelvis with negative nodes but with histologically confirmed microscopic seedling of abdominal peritoneal surfaces.

IIIB: Tumour of one or both ovaries with histologically confirmed implants of abdominal peritoneal surfaces, non exceeding 2 cm in diameter nodes are negative.

IIIC: Abdominal implants > 2cm in diameter or positive retroperitoneal or inguinal nodes.

Stage IV: Growth involves one or both ovaries, with distant metastases, if pleural effusion is present, there must be positive cytology to alot a case to stage IV. Parenchymal liver metastases equal stage IV.

The patient presented had stage IIIB disease. In patients with stage IA disease and grade I tumours, chemotherapy following initial surgical treatment has no influence on survival. Therefore, this group of patients if selected carefully does not require chemotherapeutic treatment. However, all other patients should undergo systemic chemotherapy. Agents shown to be active against epithelial ovarian cancer include cisplatin, carboplatin, cyclophosphamide, and paclitaxel. Combination therapies have been demonstrated to be superior to single agent treatment currently the most effective regimen uses a combination of paclitaxel and cisplatin or carboplatin. Six courses are given every 3 to 4weeks Our patient had combination chemotherapy with cisplatin, cyclophosphamide and adriamycin. Assessment of response to combination chemotherapy is based on physical examination, changes in size of palpable or radiographically measurable lesions,

and changes in the CA125 level. Although the preoperative CA125 level does not correlate with tumour burden, changes in response to chemotherapy appear to be of some prognostic benefit [1-4] .After completion of initial therapy for ovarian cancer, patients without clinical evidence of disease may undergo a second-look operation to determine the therapeutic response and assess the persistence of tumour [2].The prognosis for patients with ovarian cancer is primarily related to the stage of the disease. The 5 year survival rate for patients with stage I epithelial ovarian cancer, is depending on tumour grade, between 76% and 93%. The 5 year survival rate for those with stage II disease is 60% - 74%. Stage III ovarian cancer is associated with 5 year survival rate of approximately 23 - 41%. The survival rate fora patient with stage IV disease is about 11% (2)

REFERENCES

1. Duska L. Bicher A. Ovarian cancer. The John Hopkins Manual of Gynaecology and Obstetrics, Lippincott Williams and Wilkins, Philadelphia, 1999: 370-386.

2. Dongo 0, Baker V.P. Premalignant and Malignant Disorders of the Ovaries and Oviducts. Current Obstetric and Gynaecologic Diagnosis and Treatment 9h edition, McGraw Hill, 2003: 933-946.

3. Ivan-Nagel J.F, Gershenson D.M. Ovarian Cancer: Etiology, Screening, and Surgery. Te Linde's Operative Gynaecology, 9th edition, Lippincott Williams and Wilkins, Philadelphia, 2003: 1487 - 1515.

4. Berek J.S. Ovarian Cancer. Novak's Gynaecology, 13th edition, Lippincott Williams and Wilkins, Philadelphia, 2002: 1245 - 1307.

5. Stabile I., Chard T, Grudzinskas G. (Eds). Tumours of the Ovary. 2nd edition, Springer, 2000: 183 - 188.

GYNAECOLOGY CASE 11

CHORIOCARCINOMA ; CHEMOTHERAPY — REMISSION

NAME	A.N	PARITY	3+0
AGE	30 YEARS	LMP	4.8.2004
IPNO	1024960		
DOA	12.5.2005		
DOD	17.6.2005		

Presenting complaint

She was admitted to the acute gynaecologic ward from casualty as a referral from Matuu Cottage Medical Centre with a nine months history of vaginal bleeding on and off.

History of presenting complaint

She had been well until September 2004 when she developed prolonged vaginal bleeding. There was no associated abdominal pain. She was on oral contraceptives when the bleeding started and she was advised to stop taking them. The bleedin[g] persisted in spite of the stoppage. [In] January 2005 she came to KNH and MVA was done as she was thought to have incomplete abortion. The bleeding continued and in March 2005, she sought treatment at the Cottage Medical Centre where MVA was repeated. The bleeding continued and two weeks later a pregnancy test done was positive and she was referred to KNH.

Obstetric and gynaecologic history

She was Para 3+0. All her deliveries were SVD and the children were alive and well. Her last deliver[y] was in March 2002. Her LMP was on 4.8.2004. Her menarche was at 15 years. Menses lasted 4 days and the cycles were regular every 28 days. She had been on oral contraceptives since January 2003.

Physical examination

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

Abdominal examination Normal

Pelvic examination Normal

A tentative diagnosis of choriocarcinoma was made.

Investigations done: Ultrasound showed a bulky uterus with a mass containing areas of mixed echogenicity and cystic changes. The endometrium was thickened (about 2 cm thick). The impression was molar pregnancy.

1.Haemogram

2.Hb	9.84 g/dl	RBC	4.22 x 1012/1
3.WBC	3.8 x 10^9/1	Platelets	258 x 109/1

4. Urea and electrolytes normal
5. Liver function tests were within normal
6. Blood group B positive
7. Chest X ray normal
8. Beta hCG 317, 570
9. Head CT scan was normal

After the results of investigations the diagnosis was revised to high risk choriocarcinoma.

Management

The diagnosis was explained to the patient and she was started on chemotherapy. Methotrexate 25 mg daily intravenously for five days. Anctinomycin D 50 mg daily intravenously for five days and Cyclophosphamide 500 mg daily for five days. She tolerated the medication well and was dischar-ed home on the fifth day. She was to be admitted in two weeks for the second course. She was to have haemogram, urea and electrolytes and liver function tests done before readmission. She remained in the ward and on 30.5.2005 the results of blood investigations were acceptable and PhCG level was 21.6 IU/1. She was given the second course which she tolerated well.

BHCG LEVELS DURING TREATMENT (NORMAL RANGE 0-10)

ıte	BhCG (IU/1)	Course given
.5.2005	317,570	Pre- treatment I st course
.5.2005	21.6	2nd course
30.6.2005	7.0	3"course
30.7.2005	5.6	4"course
29.8.2005	5.8	5" course

The patient went home after the third course of chemotherapy. She came to the ward with blood results and was given the remaining courses of chemotherapy. She was advised on family planning with oral contraceptives during follow-up.

DISCUSSION

Gestational trophoblastic neoplasms include the tumour spectrum of hydatidiform mole, invasive mole and choriocarcinoma. Thev arise from foetal tissue within the maternal host and are composed of both syncytiotrophoblastic and cytotrophoblastic cells. They have properties of the placenta including invasion and liberation of human chorionic

Choriocarcinoma is rare and is reported in 2-5% of all cases of gestational trophoblastic neoplasms (1) .The incidence is about 1 in 5000 pregnancies in oriental countries and 1 in 50,000 in Europe and North America At KNH Fongoh repoted an incidence of 1 in 1118 pregnancies (4). In about 50% cases choriocarcinoma follows as molar pregnancy, 25% follow a term pregnancy while the rest follow other forms of pregnancy e.g ectopic, abortion (1, 3, 5).

The patient presented had choriocarcinoma following a term pregnancy.

The primary growth is usually in the uterine wall but may be in the cervix or vaginal, or in the tube or broad ligament following ectopic pregnancy. The tumour is soft, necrotic,

haemorrhagic, dark red or purple in colour and rugged or friable (3). Histologically, choriocarcinoma is characterised by sheets of anaplastic syncytiotrophoblast and cytotrophoblast without chorionic villi (1, 5). The tumour is unusual in that it does not stimulate any stromal reaction and is therefore essentially a mixture of haemorrhage and necrosis with tumour cells scattered within the mass (1). In choriocarcinoma the predisposition of normal trophoblast to invasive growth and erosion of blood vessels is greatly exaggerated (5).

The leading symptom is irregular uterine bleeding coming sooner or later after expulsion of a mole or a normal pregnancy ([3]), 5, 6). As the condition advances an offensive vaginal discharge develops and cachexia with pyrexia supervenes (3). In many cases the first indication may be a metastatic lesion. Gestational trophoblastic disease has a non—gynaecological presentation in more than 1/3 of cases (7). There can be vaginal or vulval tumours, cough and bloody sputum from pulmonary metastates, or headache and visual disturbance from brain metastates (5).
The patient presented with vaginal bleeding after a normal delivery.

Metastases often develop early and are generally blood borne because of the affinity of trophoblast for blood vessels. The most common sites of metastases are lungs (75%) and vagina (50%). The vulva. kidneys, liver, ovaries, brain and bowel are also common sites of metastates (5, 6). Cerebral metastases are usually a poor prognostic sign for the patient(8)
The patient presented did not have evidence of metastases outside the uterus.

Diagnosis relies on history. Any case of unusual bleeding after a term pregnancy or abortion should be investigated by curettage and measurements of chorionic gonadotrophin level (hCG). Persistent or rising gonadotrophin level in the absence of pregnancy is indicative of trophoblastic tumour. Ultrasound is done to evaluate the degree of uterine involvement. Chest X-ray detects lung metastases (cannon ball appearance). CT scan should be done to evaluate the brain, lung, liver and pelvis (5).
Several other investigations are done after diagnosis is made and before treatment.

In this patient ultrasound showed echogenic and cystic changes in the uterus, PhCG level was 317,570 IU/1, chest X-ray was normal, blood group was B positive, haemogram, urea and electrolytes and liver function tests were normal.

The patient presented had stage IC disease because hCG level was 317,570 IU/I , she presented with vaginal bleeding two and half years after normal pregnancy, ultrasound showed disease confined to the uterus and chest X-ray was normal.

In additional to anatomic staging, it is important to consider other variables to predict the likelihood of drug resistance and to assist in selecting appropriate chemotherapy. The scoring system is based on prognostic factors. It has been proposed by the World Health Organization (WHO) (10).

A total score <5 = low risk, 5-7 medium risk, > 7= High risk

The patient presented had PhCG level 317,570 IU/1, blood group B positive, the choriocarcinoma followed a term pregnancy and had not received prior chemotherapy. She had a score of 9 which put her in the high risk group.

Treatment depends on stage, level of BhCG, duration of the disease, the specific site of metastases and the extent of prior treatment (11). Of utmost importance in treating these patients is institution of therapy as quickly as possible and continuing therapy at very close intervals until normal BhCG titres are obtained. The interval between courses should rarely exceed 7-10 days depending on the treatment regimen toxicity. Patients should receive 1-3 courses of chemotherapy after the first normal BhCG depending on the extent of disease (3, 10).

In the patient presented BhCG level was normal after the ^{2nd} course of treatment and she continued up to the ^{6th} course.

This patient was treated with combination therapy with good results.

The EMA-CO regime consisting of Etoposide, Methotrexate, Actinomycin D, Cyclophosphamide and Vincristine is the preferred primary treatment of patients with metastases and high prognostic score (> 8) as well as MAC treatment failure (12). If the patient does not desire fertility hysterectomy with adjuvant chemotherapy may be performed as primary treatment.

The patient desired future fertility so hysterectomy was not performed.

Cerebral metastases usually spell a grave prognosis for the patient (13, 14). When chemotherapy for primary disease is started these tumours undergo haemorrhage and necrosis (13). Better results are got with total brain irradiation with 3,000 — 4,000 rads at a rate of 200 rads/day for 5 days over a period of 2 weeks combined with chemotheraphy (I. 1-)).

The patient presented had no cerebral metastases.

After completion of chemotherapy, patients are followed up by serial PhCG estimations. The patient must use an effective contraceptive (combined pill is preferred) to avoid pregnancy for at least 12 months (1). This period allows for full metabolism and excretion of the chemotherapeutic agents, mature ova affected by chemotherapy to be eliminated as well as monitoring for relapse. Pregnancy is achieved once contraception is stopped in most patients. There is no increase in risk of abortion or congenital malformations (1. 5. 15). Recurrent Hydatidiform mole develops in 1:50-1:150 pregnancies and women with previous gestational trophoblastic disease (GTD) are at 10 — 20 times increased risk of GTD than women who have never had GTD (15). Occasionally GTD can occur or recur after a subsequent normal pregnancy (2).

REFERENCES

1. O'Quinn A.G, Markusen T.E. Gestational Trophoblastic Diseases In : Current Obstetrics Gynecologic Diagnosis and Treatment 7[th] ed McGraw-Hill Chp 50 pg 947-958

2. Tidy J.A, Rustin G. J. S, Newlandi E.S. Presentation and management of choriocarcinoma after a molar pregnancy Bri. J Obstet Gynaecol 102 : 715 1995

3. Tindal V.R. Trophoblastic tumor In : Jeffcoates Principles of Gynaecology 5 " ed Butterworths &, Co. Chp 14 pg 226 – 2')7 1987 m

4. Fongoh F.B. Clinical and laboratory evaluation of choriocarcinoma at KNH 1980 – 1983 M. Med Thesis UON 1984

5. Cunningham F.G, leaves K.J. Gant N.F. et al : Diseases and abnormalities of the placenta In : Williams Obstetrics 21[st] ed McGraw-Hill Chp 32 pg 827-852 2001

6. Onadeko B.O. Anotendu A, Aimakhu V.E. Malignant trophoblastic disease with special reference to pulmonary involvement in Ibadan. East Afri. Med J 58 303 1982

7. Goldberg C.L. Yon D.A. Block B The significance of vaginal metastases. Gynaecol Oncol 24 : 155 1986

8. Newlands E.S. Bagshave K.D, Begent R.H. Results with the EMA-CO regimes in high risk GTD (1979 – 1989) Bri. J Obstet Gynaecol 98 : 550 1991

9. Petterson F. Staging rules for gestation trophoblastic tumour. Acta Obstet Gynaecol Scand. 71 : 224 1992

10. WHO Scientific group. Gestational trophoblastic diseases. WHO technical report series 692 : 1 1983)

11. Lurain J.R. Gestational trophoblastic tumours. Seminars in Surg. Oncol 6. 347 1990

12. Berkiwitz R.S, Goldstein D.P, Bernstein M.R. 10 years experience with Methotrexate and Folinic acid as primary theraphy for GTD (1974 — 1983) Gynaecol Oncol 23 : It 1986

13. Ilancheran A. Ratnam S.S, Barakan G. Metatastic cerebral choriocacinoma with primary neurological presentation. Gynaecol Oncol 29 : 361 1988

14. Bahemuke M. Neurological complications of choriocarcinoma at KNH East Afri. Med J 58 ; 117 1981

15. Ngan H.Y.S, Won[g] L.C.. Ma H.K. Reproductive performance of patients with GTD in Hong Kong. Acta Obset Gynaecol Scand. 67 ; 11 1988

GYNAECOLOGY CASE 12 IMPERFORATE HYMEN CRUCIATE INCISION

DOA 12/7/2007

DOD 13/7/2007

WARD 1D

CHIEF COMPLAINTS

She presented with cyclical lower abdominal pain and back pain for 6 months.

HISTORY OF PRESENTING COMPLAINTS

She was well till about 6 months prior, when she developed some discomfort in the lower abdomen. Every month, the discomfort increased to become cramping lower abdominal pain and back pain. Subsequently she noted a swelling in her lower abdomen. She had never had any menses. She did not have any urinary complaints. There was no change in the bowel habits.

OBSTETRICS AND GYNECOLOGY HISTORY

She was a nulliparous lady who had never had menses.

PAST MEDICAL HISTORY

She had never been admitted to hospital for any illness or surgical operation. She had no known chronic illness.

FAMILY AND SOCIAL HISTORY

She was the $_{2}$nd born in a family of 3 siblings. She was currently in class 7 in a local primary school. Her father was a primary school teacher while her mother was a businesswoman. There were no known chronic ailments.

PHYSICAL EXAMINATION

She was a young lady in good general condition and well nourished. She was not pale, did not have edema, cyanosis, or lymphadenopathy. Her vital signs were normal with a blood pressure of 100160 mmhg, pulse rate of 76/min, respiratory rate of 14/min and a temperature of 36.6°C.

ABDOMINAL EXAMINATION

The abdomen was distended in the suprapubic region. There was a mass, which you could not go under approximately 18 weeks equivalent. It was tender and mobile side to side. Bowel sounds were present and normal.

PELVIC EXAMINATION

There were normal external genitalia. There was protrusion of the hymen as a mass that was dark in color. The mass was tender and further pelvic examination was abandoned.

RECTAL EXAMINATION

The rectum was full. The uterus could be felt, below which was a cystic mass thought to be the vagina.

DIAGNOSIS

Imperforate hymen with hematocolpos and hematometra. A pelvic ultrasound was requested.

MANAGEMENT

The pelvic ultrasound confirmed examination findings. The diagnosis was explained to the patient and her mother and they accepted to go to theatre for incision of the hymen.

A hemogram, urea, creatinine and electrolytes were requested. The mother signed a consent form.

RESULTS OF INVESTIGATIONS

- Hemogram
  - Hb 12.6 g/dl
  - RBC 4.2 x 106/MM3
  - WBC 4.8 x 103/mm3
  - Platelets 370 x 106/mm3
- Urea 6.2 mmol/l
- creatinine 50 micromoles/L
- NA+ 140 mmol/L. k+ 4.1 mmol/L

PROCEDURE

Half-hour before being wheeled to theatre, she was premedicated with 0.6mg I.M atropine. On the theatre table, she was put under general anaesthesia. She was repositioned to the lithotomy position, Vulvo vaginal toilet and aseptic catherisation done. Vaginal examination confirmed previous findings. The hymen membrane was incised, at 2, 4, 8 and 10 O'clock. The quadrants of the membrane were excised after drainage of about 2 litres of chocolate brown altered blood. The patient was cleaned and anaesthesia successfully reversed.

Post-operative care

She was put on Tramadal 50mg 8 hourly for 24 hours. 6 hours after coming from theatre she was started on oral sips. On the 2nd post-operative day she was discharged home on Augumentin 375 mg TID x 5/7, Brufen 400mg TID x 5/7 and follow-up in the Gynecology clinic in two weeks.

FOLLOW UP

In the gynecology clinic 2 weeks later, the patient was doing well and the abdominal swelling had disappeared. She was discharged from the clinic unless in need.

DISCUSSION

Presented is a 14-year-old schoolgirl with hematocolpos and hematometra due to imperforate hymen. Cruciate incision was done with good results.

The hymen is the junction between the sinovaginal bulbs and the urogenital sinus. It is a thin membrane sometimes cribriform in appearance composed of endoderm derived from the epithelium of the urogential sinus (1). They hymen is usually perforated in embryonic life to establish a connection between the lumen of the vaginal canal and the vestibule. It is usually torn in pre-pubertal years. When there is no perforation through this membrane it is called imperforate hymen (1, 2, 3).

Imperforate hymen is perhaps the most common obstructive anomaly of the female genital tract, with an incidence of between 0.01 and 0.1% among female newborns. Most of the

cases are sporadic with no evidence to suggest any genetic factor (4). It is almost always an isolated finding. Associated anomalies, including urinary tract anomalies, are rare. Some of these anomalies may include: imperforate anus, bifid clitoris, hypo plastic kidney and vascular abnormalities (4).

Imperforate hymen has no symptoms if the uterus is absent or functionless (5). If the uterus is present, symptoms usually occur at puberty. Occasionally in early childhood, placental hormones may stimulate the uterus and cervix leading to the collection of mucus in the vagina, causing mucocolpos, at birth (2, 5).

Symptoms in imperforate hymen are due to the accumulation of menstrual blood. The blood of the first period or two is collected in the vagina. The vagina can hold blood from one or two cycles without undue stretching and with no other symptoms. Accumulation of menstrual blood in the vagina is termed hematocolpos. The patient may feel a little fatigue and have crampy discomfort suggesting menstruation, but no blood appears at the vaginal outlet. As menstruation recurs, the vagina becomes greatly over distended, and the cervical canal also dilates. Hematometra, which is the accumulation of menstrual blood in the uterine cavity, may form. This was the presentation in our patient. When the intrauterine pressure reaches a certain point,

there is retrograde passage of blood into the tubes, forming a hematosalpinx. Adhesion formation within or at the fimbriated end of the tubes can seal them, and little or no blood may enter the peritoneal cavity. If it enters the peritoneal cavity it forms hematoperitoneum (1, 2).

Diagnosis of imperforate hymen is rare before puberty. Most patients are brought to the doctor at 13 to 15 years of age when their mothers begin to notice symptoms and the girls appear not to have begun menstruating. At puberty, primary amenorrhea and cyclic cramping lower abdominal pain are the most common complaints. Other symptoms may include discomfort in the pelvis and how back pain (1, 2, 3). Urination can also be difficult, because pressure of the distended vagina on the urethra may compress the urethra and preventing

emptying of the bladder. Cramps like pains recur in the supra-public region, a long with the common urologic symptoms of dysuria, frequency, and urgency. Overflow incontinence may develop eventually (1).

When the patient is examined, a tender mass is often palpable supra-pubically, the result of uterine enlargement and upward displacement, or bladder distension, or both. If hemoperitoneum occurs, the irritation of the free blood may cause the patient to experience all the symptoms and demonstrate signs of peritonitis. Our patient did not have these symptoms and/or signs. Vulva) inspection reveals the bulging pink imperforate hymen, which may or may not be bluish in color depending on its thickness. On rectal examination, the vagina is palpable as a large cystic mass (6).

The management of imperforate hymen is usually surgical. This involves excision of the membrane to allow flow of the accumulated blood. The hymenal membrane is incised preferably at 2,4,8 and 10 O'clock. The quadrants of the hymen are then excised and the mucosal margins are approximated with fine delayed absorbable suture. If hematocolpos has already developed all unnecessary intrauterine instrumentation should be avoided because of the risk of perforating the thin overstretched uterus wall. To prevent scarring and stenosis, which could result in dyspareunia, the hymenal tissue should not be excised too close to the vaginal mucosa. Should the uterine mass fail to

regress in 2 - 3 weeks, then inspection and dilatation of the cervix should be performed to make certain that drainage from the uterus is satisfactory.

REFERENCES

1. Rock A., Horowitz I.R. Surgical conditions of the vagina and urethra. Te Linde's Operative Gynecology, 8^{n} edition. Lippincott - Raven, 1997; 911-938
2. Martin A.S., William D., Arthur L.H., MischelL D.R. Imperforate hymen Comprehensive gynecology, 4th edition. Mosby Inc. 2001; 253 - 265.
3. Wendy J., Scherzer, Howard M.C., Chamrock. Amenorrhoea. Novak's Gynecology. 12th

edition 2000. Pg. 819

4. Usta I.M., Anward J.T., Usta J.A. Imperforate hymen: Report of unusual, Familial occurrence. Obstet. Gynecol. 1993; 82: 665.
5. Tindal V. R. Malformations and Maldevelopments of the Genital Tract. Jeff Coates Principles of Gynaecology, 5th edition, Butter works & Co; 1987, 138 - 158.
6. Murram D. Congenital anomalies of the female genital tract. Current obstetric and gynecologic diagnosis and treatment. 8th edition 2001 pg. 640-41.

GYNAECOLOGY CASE NO 13

INCOMPLETE ABORTION – MVA DONE

NAME: A. S. AGE: 25 YEARS

IP. NO.: 1003542

DOA: 13/01/05 **DOD:** 22/01/05

25 year old pars 0+0 G 1 LMP 15/09/04 She was 17 weeks pregnant.

HISTORY OF PRESENTING ILLNESS

A. S. gave history of having tripped and fell down while going up the stairs one day earlier hitting the floor on her abdomen. Later she started experiencing intermittent low abdominal pains accompanied by bleeding in clots. She changed her pads 4 times in 24hrs which were fully soaked with blood.

PAST MEDICICAL HISTORY

Not significant

OBS/GYN HISTORY

Menarche was atl6 years, her menstrual cycle lasts 28 days, while the flow takes 3 days, and is regular. She has never been on any contraception.

FAMILY SOCIAL HISTORY:-

She is a recently married housewife while her husband is a businessman. There is no chronic illness in the family.

On examination: - She was in fair general condition, not pale, jaundiced, cyanosed or oedematous.

Vital signs:- BP 100/80 mmhg, TEMP. 36.8°C, PR 76/ min, RR 14/ min

P/A: The abdomen was slightly distended with an enlarged uterus at 14/40 which was non tender.

V/E: She had normal external genitalia, Cervix was central, 3 cm dilated with products of conception felt at the cervical os, both adnexa and Pouch of Douglas were free.

An impression of Incomplete Abortion was made.

Lab investigations:

TBC: Hb 9.8gm/dl, WBC 18.8X10^9/L, Neut 81.9%, Platelets 321x109/L UEC:

Na 140, K4.0, Cl 106, Urea 5. 1, Creat 84.

Management

The procedure and its importance were explained to the patient and informed consent obtained. She was taken to the procedure room and was placed in lithotomy position, vulval vaginal toilet was performed using savlon as an antiseptic and the patient was draped with sterile towels. A vaginal examination revealed the same findings as above. A Cusco's bivalve speculum was inserted into the vagina and the cervix exposed. It was cleaned using sterile cotton swabs soaked in antiseptic solution. The cervix was normal without lacerations or tears. The walls of the vagina were found to be normal; the cervical os was dilated with products of conception visible at the cervical os.

The anterior lip of the cervix was held with a tenaculum forceps with prior warning to the patient that there would be some pain. A size 12 cannula was chosen and the manual vacuum aspiration performed as explained in the introduction. 100 mls of non foul smelling products of conception were evacuated from the uterus, the bleeding after the procedure was minimal. The instruments were removed and the patient taken to the ward. Throughout the procedure, the assisting nurse spoke to the patient and explained the steps as they were taken and reassuring the patient, urging her to be cooperative and to bear the discomfort.

Post operative management

The patient was observed in the ward for 4 hours for per vaginal bleeding which was monitored using pads, this was minimal and her repeat vital signs were as follows:-BP range 100-110 mmHg systolic and 70-80 mmHg diastolic, pulse 70-80 beats per minute, respiratory rate averaged 20 breaths per minute. She was on brufen for pain.

She was discharged home on empirical treatment of Doxycycline 100mg BD for seven days, Flagyl 400mg TDS for seven days and Brufen 400mg PRN for pain and Ranferon syrup for two weeks as an haematenic. She was to be seen in the GOPC in two weeks time.

She was advised to report in the outpatient department urgently if she noted any of the following symptoms:-Increased or persistent per vaginal bleeding.Foul smelling per vaginal bleeding.Fever, chills, rigors or persistent headaches.

Follow up

She was seen in the GOPC after two weeks and was in good general condition, she had no complains. She was discharged from the clinic and advised to start her ante natal clinic early in the next pregnancy.

DISCUSSION:

The patient presented was a 30 year old Para 0+2 who had an abortion of her first pregnancy at about two months for which an MVA was done, then later on developed a ruptured tubal ectopic pregnancy for which she underwent salpingectomy. During subsequent follow up, she was found to have secondary infertility due to blockage of the remaining fallopian tube for which she had to under go open tuboplasty. Her post operative recovery period was uneventful.

GYNAECOLOGY CASE NO 14 LOST IUCD RETRIVAL

DOA 12/04/08

DOD12/04/08

Low abdominal pains x 4/12

Had also reported lost IUCD.

HISTORY OF PRESENTING ILLNESS

The low abdominal pains were intermittent, dull in nature and radiating to the back. There was no associated per vaginal discharge or bleeding. Patient gave history of having had an IUCD inserted 4 years ago and wanted it to be removed. However when she went to the Family Planning clinic the strings could not be seen at the cervical os and so she was sent fora pelvic U/S scan which showed a translocated IUCD which she wanted to be retrieved.

OBS/GYN HISTORY

She had her menarche at 14 years; her menstrual cycle was 3-4/28, regular. Para 7

+ 0 Last delivery was 5 years ago

All her deliveries were spontaneous vertex deliveries; all the children are alive and well.

Contraceptive history: had a IUCD inserted in 2001 after the last delivery.

FAMILY SOCIAL HISTORY

She is married, a house wife, and lives in Nyeri. She does not smoke or take alcohol. There is no

EXAMINATION,

She was in fair general condition, not pale, jaundiced, cyanosed or oedematous.

P/A The abdomen was scaphoid with no palpable mass, non tender. Normal external genitalia, Cervix was closed, there were no IUCD threads felt

Uterus was normal in size; there was no adnexae tenderness or mass felt. The Pouch of Douglas was free.

Pelvis Ultrasound Scan done on 7/6/06:- Showed a normal sized uterus, 9.45 cm in length with normal echo pattern with no uterine wall masses noted. There was an empty endometrial cavity. An IUCD was noted, which appeared posterior to the uterus just above the cervical part, one end appears to be within the uterine wall while part of it is in the Pouch of Douglas. There appears to be some adhesions above it but no free fluid is noted in the Pouch of Douglas. No pelvis mass was noted.

Conclusion:

Normal sized uterus. IUCD in the Pouch of Douglas with part of it within the uterine wall posteriorly Impression; Lost IUCD (within the peritoneal cavity adjacent to the uterus)

Lab investigations done on 7/08/06

Haemogram: Hb – 12.2g% RBCs 5.10 x 10 12/1, urea was 2.2 mmol/l

Management

The patient was explained the above findings and the need for her to undergo a laparatomy to have the translocated IUCD removed. She then gave a written informed consent for the operation to be done on 10/08/06. Blood for grouping and cross matching was taken and a theatre list prepared. She was to remain nil per oral from midnight on the day of operation. She was pre-medicated with intramuscular Atropine 0.6mg stat half hour before the operation then wheeled to theatre.

In theatre, patient under general anesthesia, in semi-lithotomy position, vulvo-vaginal toiletry was done, bladder aseptically catheterized and drained about 80mls of clear urine, then the patient was put in supine position. The abdomen was cleaned and draped. The abdomen was opened via a sub umbilical midline incision. Infra-operatively, the IUCD was found sticking through the posterior uterine wall in the Pouch of Douglas with few flimsy adhesions covering it. It was pulled it out, but no significant bleeding was observed. The abdomen was closed in layers and patient was successfully reversed from general anaesthesia.

Post operative care.

Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mls 4 hourly for the first 24 hours. She was also on parenteral Crystalline Penicillin 2MU 6 hourly, Gentamycin 80 mg 8 hourly and Pethidine 100mg 8 hourly for 48 hours then oral Amoxil 500mg, Flagyl 400mgs and Brufen 400mgs for the next 3 days.

DISCUSSION: A.W. was a 40 year old Para 7+0 who presented with a IUCD that had perforated through the posterior uterine wall and was retrieved via laparatomy.Intrauterine contraceptive devices (IUCDs) are flexible metal or plastic devices that are inserted into the endometrial cavity through the cervix for contraception. In general, devices are of two varieties. Those that are chemically inert are composed of a non-absorbable material, most often polyethylene, and impregnated with barium sulphate for radiopacity e.g. Lippes loop. In those that are chemically active, there is continous elution of copper or a progestational agent. Copper containing IUCDs include Copper T 380A (Paragard), Nova T and Multiload 375 while hormone releasing IUCDs include the progesterone releasing T (Progestasert) and the levonorgestrel releasing T (Mirena). [1,2,3] Our patient had a Cu. T 380A.The exact mechanism of action of IUCDs is not precisely understood. The postulated mechanisms of action include: [3,4] Induce intense local inflammatory response that leads to lysosomal activation and other inflammatory actions that are spermicidal. Inhibits fertilization by the spermicidal action and by speeding up ovum transport, Immobilizes sperms; interferes with migration of sperms from the vagina into the fallopian tubes, Speeds up the transport blastocyst from the endometrium; increases local production of prostaglandins: inhibits implantation; disrupts the proliferative-secretory maturation process (progesterone elaborating IUCDs) thus impairing implantation, Progesterone producing IUCDs produce an atrophic endometrium in long-term use and may interfere with sperm penetration through thickened cervical mucus. The effectiveness of IUDs is similar to the overall effectiveness of oral contraceptives. The Copper T-380A and the levonorgestrel T have remarkably low pregnancy rates, less than 0.2 per 100 woman years. The Progestasert has a high failure rate, about 3 pregnancies per 100 women per years Importantly, the unintended pregnancy rate decreases progressively after the first year. Complications of IUD insertion include cramping, uterine perforation (<1 per 1000 insertions)

usually when sounding the uterus or during insertion of the device, and abortion of an unsuspected pregnancy. [2-6] Fainting or collapse of the patient at the time of insertion of the IUCD is due to dilataion of the cervix. Intermenstrual spotting and menorrhagia occurs in 50% of cases. They are mostly seen during the first few days and months and tend to disappear later. Dysmenorrhoea and intermenstrual pain are due to uterine colic's. [2,6]

REFERENCES

1. Robert A.H. et al. Intrauterine devices. The Essentials of Contraceptive Technology. John Hopkins Population Information Programme, 1998: 12-1 to 12-28.

2. Grewal M, Buckman R.T. Contraception and Family Planning. Current Obstetric and Gynaecologic Diagnosis and Treatment, 9h edition, Lange medical Books/ McGraw Hill, 2003: 641-644.

3.Cunningham FG, Gant NF, Leveno KJ, Gilstrap LC, Hauth JC, Wenstrom. KD (Eds) Contraception. Williams Obstetrics, 1st edition McGraw-Hill, 2001:1535 - 1541.

4. Hatcher RA, Kowal D, Guest F, et al. (Eds).Intrauterine Devices (IUDs) Contraceptive Technology, International Edition, Printed Matter Inc, Atlanta GA, USA, 1989: 309- 333.

5. Stubblefield P.G. Family Planning. Novak's Gynaecology, 13[0]. edition, Lippincott Williams and Wilkins, Philadelphia, 2003:242-247.

6. Martin L Pemoll. Contraception. Handbook of Obstetric and Gynaecology, 101' edition, McGraw Hill, 2001: 739-741.

Tindall V.R. Contraception. Jeffcoate's Principles of Gynaecology, 5th Edition, Butterworth

GYNAECOLOGY CASE NO 15

SECONDARY INFERTILITY DUE TO TUBAL FACTOR – OPEN TUBOPLASTY DONE

NAME: M. I. **AGE:** 30 years

IP NO : 0933617

DOA: 12/10/04 **DOD:** 21/10/04

30 years old Para 0+2 LMP 25/09/04 admitted with C/O:

Inability to conceive x 2 years

HISTORY OF PRESENTING ILLNESS

M.I gave history of inability to conceive for two years despite having regular un-protected intercourse about 4 times per week. She stays together with her husband. There was no history of per vaginal discharge or low abdominal pains. She has normal vision and has never experienced any nipple discharge. The two pregnancies she has lost were from her current husband. She has never been treated for any sexually transmitted infection / pelvic infection before.

PAST MEDICAL HISTORY,

She had been admitted to KNH in December 2003 with a ruptured left tubal ectopic pregnancy for which salpingectomy was done.

OBS/GYN HISTORY

She attained her menarche at the age of 12 years. Her menstrual cycle lasts 28 days with a menstrual flow of 3 days and is regular. There is no history of dysmenorrhoea. She has never used any contraception.

- Had an abortion in 1994 at 8 weeks – MVA was done.
- Had a ruptured ectopic pregnancy in 2003 – left partial salpingectomy was done.

FAMILY SOCIAL HISTORY

She has been married since 1994, is a housewife. There is no history of chronic illness in the family.

On examination, she was in fair general condition, not pale, jaundiced, cyanosed or oedematous. Vital signs: BP 100/60 mmHg PR 90/min Temp 36.7°c

ABDOMINAL EXAM: The abdomen was not distended, was moving with respiration. There was a Pfannenstiel incision scar, abdomen was non tender.On vaginal exam she had a normal external genitalia, cervix central, closed, adnexa and pouch of Douglas free, uterus not enlarged.

Investigations

TBC - Hb 12.2gm/dl, WBC $3.9X10^9/1$,, Plat 188x109/1,U/E/C - Na 145 mmol/1 K4.4 mmol/L, Urea 5.2 mmol /L, Creat 56pmol/L,HIV –Negative. Semen-analysis: Volume 2.3 mls, Appearance : creamy white, Motility 3% rapidly progressive, 2% slowly progressive, immotile 95%, Vitality 8% alive, Total sperm count 6.4x I 06/mil, WBC count 1.2x I $^{06/ML}$ Morphology :Normal 4% Abnormal 96%. Impression: Aestheno-oligozoospermia with elevated WBC count. Comment: Treat infection and repeat semenalysis. HSG: Showed a right tube with peri-fimbrial adhesions with no spill over of dye into the peritoneal cavity with left tubal occlusion.

DIAGNOSIS; An impression of Secondary infertility due to tubal factor was made and patient was admitted for open tuboplasty on 18/10/04.

Management.

The patient was explained about the diagnosis and the need for her to under go an open tuboplasty. Her husband was to be put on a course of antibiotics then a repeat semenalysis was to be done after three months. She then gave a written informed consent for the operation to be done on 18/10/04. Blood for grouping and cross matching was taken and a

theatre list prepared. She was to remain nil per oral from midnight on the day of operation. She was pre-medicated with intramuscular Atropine 0.6mg stat half hour before the operation then wheeled to theatre.

The patient was taken to theatre. Under general anaesthesia, vulvo-vaginal toilet was done, bladder catheterized and the posterior vaginal wall packed with gauze roll to elevate the uterus. Abdominal wall cleaned and draped, then opened through a Pfannenstiel incision. Intra-operatively, found a left tubal salpingectomy stump. The right fallopian tube was visualized with numerous peri-fimbrial adhesions to the right ovary. Uterus, both ovaries and bladder were visualized and normal.

Adhesionolysis of the fimbrial adhesions was done, uterine cervix was clumped and dye introduced to the uterine cavity. There was no free flow of the dye to peritoneal cavity achieved. A massive hydrosalphinx formed on the middle 1/3 of the isthmic portion of the right tube. No dye noted flowing on the left fallopian tube stump. Gutter salpingiostomy was performed at the hydrosalpinx site on the right tube, stitching done with propylene 5/0 suture. Subsequently dye spill was achieved at salpingiostomy site, pelvic cavity was irrigated with 500mg hydrocortisone in 250mls of normal saline, then Iml of heparin instilled in to the pelvis. The abdomen was closed in layers and patient was successfully reversed from general anaesthesia.

Post operative care.

Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mis 4 hourly for the first 24 hours. She was also on parenteral Crystalline Penicillin 2MU 6 hourly, Gentamycin 80 mg 8 hourly and Pethidine 100mg 8 hourly for 48 hours then oral Amoxil 500mg, Flagyl 400mgs and Brufen 400mgs for the next 3 days.

On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet. She was discharged home on the third post operative day (21/10/04).

Follow up

M.I. was initially seen at GOPC after two weeks on 10/11/04. She did not have any complaints. Her LMP had been on 6/11/04. She was given an appointment to be reviewed after three months. She was reviewed again onl0/02/05. She still did not have any complains at that time and the incision site had healed completely. She was reassured and discharged from follow up.

DISCUSSION:

The patient presented was a 30 year old Para 0+2 who had an abortion of her first pregnancy at about two months for which an MVA was done, then later on developed a ruptured tubal ectopic pregnancy for which she underwent salpingectomy. During subsequent follow up, she was found to have secondary infertility due to blockage of the remaining fallopian tube for which she had to under go open tuboplasty. Her post operative recovery period was uneventful.

Infertility is usually defined as the failure to conceive after one year of intercourse without contraception. Primary infertility applies to those who have never conceived, while secondary infertility refers to those who have conceived at some time in the past. 80% of couples experiencing unprotected intercourse will achieve pregnancy within one year; an additional 10% will achieve pregnancy in the second year [1]

In Kenya the exact statistics on infertility is not clear, but about 60% of all new gynecology clinic attendance at Kenyatta National Hospital is by patients with infertility [2]. It is further estimated that two thirds of all gynaecology consultation is taken by patients complaining of infertility [3]

The causes of infertility are varied. Globally, the causes are attributable to male factors in 30%, combined male and female factors in 30% and unknown factors in 10%. In Africa, the female factors are contributory in about 72% of the cases.Male factors can be broadly categorized into:-

1) Endocrine disorders: - Hypothalamic dysfunction (Kallman's Syndrome), Pituitary Failure (tumour, surgery), Adrenal hyperplasia, Hyperprolactinaemia.
2) Anatomic disorders: Obstruction of vas deferens, congenital absence of vas deferens abnormal ejaculatory system.
3) Abnormal spermatogenesis.
4) Abnormal motility: absent cilia (Kartageners Syndrome), Antibody formation.
5) Sexual dysfunction: Retrograde ejaculation, impotence, decreased libido.

Ovulatory factors include: -

1) Central defects: chronic hyperandrogenic anovulation, hyperprolactinaemia, hypothalamic insufficiency, pituitary insufficiency.
2) Peripheral defects: gonodal dysgenesis, premature ovarian failure, ovarian tumour, ovarian resistance.
3) Matabolic factors: Thyroid disease, liver disease, obesity.

Pelvic factors include:-

1) Infections: Pelvic inflammatory disease, uterine adhesions (Ashermans Syndrome), endometriosis, structural abnormalities, myomas, Mullerian duct abnormalities.
2) Immunologic incompatibility – like sperm immobilizing and agglutinating antibodies.

In Kenya, studies have shown that 73% of female patients with infertility have tubas occlusions secondary to pelvic inflammatory disease (PID). The micro-organisms concerned could be Chlamydia trachomatis, Neisseria gonorrhoea and Escherichia cols. Although infertity involves the loss of the denial of expectations, it defies categorization as an illness. There are few symptoms and definitive tests. A thorough clinical history and physical examination of the couple is important in arriving at a diagnosis. Male factors are evaluated by semen analysis and post coital examination. Female factors are evaluated by doing hormonal profile for LH, FSH, prolactin, serum progesterone, thyroid function tests, basal body temperature determination, endometrial biopsy, hystero-salpingogram (HSG), and diagnostic laparascopy and hysteroscopy. Post coital mucus test done 1-2 days before ovulation evaluates cervical factors. Antibody testing is done for immunologic factors. [5, 6, 7]

Treatment of infertility is cause specific where applicable. The ultimate therapy for male factor infertility as shown by un-favourable sperm parameters, a negative sperm penetration assay, or both, is in vitro fertilization or gamete infra-fallopian transfer (GIFT).

In offering treatment to induce ovulation, the premise that normal fertility should result from the correction of ovulation implies that pregnancy should occur within the first 6 cycles for the majority of patients and with in one year for up to 80%. Ovulation can be induced by using clomiphene citrate, human chorionic gonadotraphin (HCG), human menopausal gonodotrophin (HMG), pulsatile gonadotrophin release hormone (GNRH) or when appropriate by reducing serum prolactin levels by using bromocriptin 5, 6, 8

Tubal blockage may be corrected by surgery although the results may be disappointing if the tubal ciliary action is irretrievably damaged. The most successful surgery is salpingiolysis is in a case of adhesions. Other forms of tubal surgery are salpingostomy, tubal anastomosis and re-implantation fimbrioplasty. Tubal surgery can b performed either laparascopically or by open laparatomy [6, 7].

If reconstruction surgery fails or is not feasible, in vitro fertilization and embryo transfer can be taken. If everything else fails adoption or surrogate motherhood can be offered as alternative options. Since majority of our patients have tubal occlusion, as sequale of infection, revention by health education and prompt treatment of STI should be our goal.

REFERENCES:

1) Martin M.C. Reproductive Endocrinology and infertility. Current Obstetric and Gynecologic diagnosis and treatment. 7th Edition, Appleton and Lange ch 51 pp1036, 1991.

2) Mati. J.K.G et al. A study on infertility in Kenya: Results of investigations of the infertile couple in Kenya. E.Afri. Med J. 58:288, 1981

3) Mati J.K.G. Infertility in Africa: Magnitude, major causes and approaches to management. J.Obster. Gynaecol. East Cent. Afr: 5:65, 1986

4) Van-na T. Female infertility, clinical gynecology. Clinical Gynecology. 1St edition, pg 645, 1991.

5) Precis III. An update in Obstetrics and Gynaecology, infertility pp 303-310 American college of Obstetrics and Gynecology 1986.

6) Tindal V.R. nfertility and sub-fertility. Jeffcoates principles of gynecology 4th edition, 1987 pp578-597

7) Gomel V. Reconstructive tubal surgery. Te- Lindes' operative gynecology 8th edition, Lippincot – Raven Publishers Philadelphia 1997, pp 549- 584

8) Lewis UI, Chamberlain G. Infertility. Gynecology by Ten Teachers 15th Edition, pp 222-232, 1989.

RESEARCH DISSERTATION

KNOWLEDGE AND PRACTICE OF CONTRACEPTION AMONG SEXUALLY ACTIVE BREASTFEEDING WOMEN IN THE FIRST YEAR AFTER DELIVERY AT THE MCH CLINIC OF MBAGATHI DISTRICT HOSPITAL.

ABSTRACT

Background: Breastfeeding women who resume sexual activity after childbirth are at risk of pregnancy even though they experience lactational amenorrhea which usually last for 6 to 12 months. Lactational amenorrhea is only effective as a method of contraception if the mother is exclusively breastfeeding which is usually not the case. It is possible that sexually active breastfeeding women may mistakenly believe that since they have not resumed their menses they are not at risk of pregnancy and do not need to use any contraceptive method. As majority of these women are in contact with health care workers in Maternal Child Health (MCH) clinics where family planning services are also provided, it would be expected that knowledge and utilization of family planning would be high. Nevertheless high pregnancy rates have been reported among sexually active breastfeeding women especially within the first year after childbirth. It is therefore important to know whether these women have adequate knowledge of family planning and the extent of utilization of family planning services after childbirth.

Study Objective: To determine the knowledge and use of contraception among sexually active breastfeeding women within the first year after delivery at the MCH clinic of Mbagathi District Hospital.

Study Population: Two hundred and six sexually active breastfeeding women were interviewed.

Study Design: A prospective descriptive study.

Study Setting: The study was conducted at the MCH clinic of Mbagathi District Hospital, an urban district hospital situated in Nairobi, Kenya.

Study methodology: Sexually active breastfeeding women attending the MCH clinic within the first year after delivery who consented to participate in the study were recruited. Interviews were conducted using an interviewer administered semi structured questionnaire to assess knowledge about family planning, use of family planning methods and factors influencing their use. The

information obtained was then analyzed using the SPSS statistical software package for personal computers. Discrete variables were expressed as percentages and presented as frequency tables and cross tabulations. Chi square was employed as the test of association of knowledge and use of contraception.

Study results: The awareness of family planning was high (100%) while the current family planning use was low (13.8%). The respondents who desired to use a method of family planning in future were higher (64.1%). Among the current no non users (86.5%) believed that lactational amenorrhea offered them natural protection from getting pregnant. Parity and the number of living children were the sociodemographic variables that were significant in influencing the utilization of family planning.

Conclusions: There is a high level of awareness of family planning but a low utilization of family planning among sexually active breastfeeding mothers. Most mothers believe in lactational amenorrhea as a form of family planning.

Recommendations: It is recommended that breastfeeding mothers be targeted for an urgent behavior communication intervention with regards to contraceptive acceptance which should not be limited to the health facilities alone but should be population based with community involvement. A much larger interventional study involving a larger population should be undertaken in order to emphasize on the need to address family planning among sexually active breastfeeding mothers. Health education on the need to adopt family planning methods should be clearly emphasized in the antenatal clinics, immediately after delivery and in the maternal/ child healthcare clinics.

INTRODUCTION

Definition

The term contraception includes all measures temporary or permanent designed to prevent a pregnancy. Ideal contraceptive methods should fulfill the following criteria:-widely acceptable, inexpensive, simple to use, safe, highly effective and requiring minimal motivation, maintenance and supervision. However no one single universally acceptable method of family planning has yet been discovered.[1, 2] Use of contraception among sexually active breastfeeding mothers is of great importance. It is a known fact that women are less fertile when breastfeeding their babies. The delay in recurrence of ovulation after delivery is due in part to the hypophyseal and hypothalamine stimulation from nursing during amenorrhea.

Lactational amenorrhea

Nonetheless the duration of suppression of ovulation is variable and this method of contraception (lactational amenorrhea) can only be practiced for up to six months after birth for most women.[2] Lactational amenorrhea is the use of breastfeeding as a temporary family planning method. Lactational amenorrhea provides natural protection against pregnancy and gives the mother enough time to start another family planning method at a proper time. A woman is naturally protected against pregnancy when her baby gets 85% of his or her feeds as breast milk, she breastfeeds her baby often day and night, her menses have not returned and her baby is less than six months old. [3] If she keeps breastfeeding very often her protection from pregnancy may last longer than six months and perhaps as long as nine to twelve months. If lactational amenorrhea is used effectively failure rate is two pregnancies per a hundred women in the first six months after childbirth. A woman who uses lactational amenorrhea should therefore be encouraged to breastfeed often at least eight to ten times a day, including at least once at night. Day time feedings should not be greater than four hours apart and night time feedings not less than six hours apart. There should be good breastfeeding techniques and good maternal diet. If these conditions cannot be met she is encouraged to start a modern family planning method.[3] Prolonged and sustained breastfeeding offers a natural protection against pregnancy especially in women who have amenorrhea.[3] However lactational amenorrhea can only be effective as a method of contraception if the mother is exclusively breastfeeding since supplemental feeding may alter the pattern of lactation and the intensity of infant suckling which may secondarily affect suppression of ovulation.[4, 5]

Menstruation

There is no way of predicting when menstruation or ovulation will resume following a delivery. On average after childbirth, fertility will return four to six weeks for those not breastfeeding and twenty four weeks for those breastfeeding.[5] It is estimated that 50% of pregnancies occur within six months of resumption of intercourse after childbirth for those not on a method of family planning. [5] Nearly 80% of breastfeeding mothers ovulate before the return of their first menstrual period and this may even occur as early as two months after delivery making sexually active breastfeeding mothers to be at risk.[5]

This study therefore aims at assessing the contraceptive knowledge and use among sexually active breastfeeding mothers within the first year after child birth at the MCH clinic of Mbagathi District Hospital in Nairobi, Kenya.

LITERATURE REVIEW

Family planning is a primary health care strategy that has important benefits for both maternal health and child health. The practice of birth control (contraception) by most couples is due to personal reasons. Many couples wish not to have children at a particular time (spacers) or to have no children or no more children (limiters). Others desire to avoid childbearing because of the effects of pre-existing illness on the pregnancy or because pregnancy may be life threatening to the mother due to chronic medical conditions.[2] Family planning allows women to determine the number and timing of their children and empowers them to manage their lives with respect and dignity.

Family planning in the postpartum period has been of great importance in many countries. This is because many women resume sexual activity immediately after childbirth especially in developing countries. There are various contraception methods available for postpartum mothers who desire to commence a certain method. These include barrier methods, intrauterine contraceptive devices (IUCD's), hormonal methods, (combined and progesterone only methods) lactational amenorrhea method (LAM) and permanent sterilization method.[2]

BIRTH CONTROL IN BREASTFEEDING WOMEN

Barrier methods

Barrier methods prevent pregnancy by blocking sperm from entering the uterus. Barrier methods are widely used by nursing mothers because they are not associated with complications known to occur with other methods in which the medication could pass into the breast milk or affect milk supply. Male and female condoms, contraceptive sponges and contraceptive gels are readily available and sold over the counter. The diaphragm is a cap that fits over the cervix and is usually fixed by the doctor or a midwife at least six weeks after the baby is born. The diaphragm must be refitted after every pregnancy and after any weight loss or weight gain of greater than 15 pounds. Nevertheless barrier methods are not as reliable in preventing pregnancy compared to birth control pills and other hormonal contraceptives.[2, 6] Diaphragms and cervical caps have been used very little for family planning purposes in Kenya and they are not available in most public health facilities.

Non hormonal intrauterine contraceptive devices (IUCD's)

Copper IUCD's are reliable long term revisable methods of birth control. Unlike hormone containing IUCD's, copper IUCD's have no effect on breast feeding.[6] It releases copper causing the endometrial to be shed more often than normal therefore blocking the implantation of fertilized eggs. Compared to non nursing mothers breastfeeding mothers experience less pain during the insertion of copper IUCD's and have decreased removal rates due to bleeding or pain.[6] Copper IUCD's can be inserted up to 48 hours after delivery including immediately after delivery of the placenta. If delivery is by caesarean section the copper IUCD's can be placed near the fundus after delivery of the placenta before closing the uterus.

Hormonal methods

Combined oral contraceptives contain estrogen and progesterone. These have not been found to be harmful to nursing mothers and their babies. Some studies have shown a 41.9% drop in milk volume in nursing mothers who are using combined oral contraceptives due to their effect on milk supply. They should therefore be avoided until the baby is six months old and is eating solid foods.[7]

Progesterone only contraceptives

These include the minipill, injectables and some progesterone containing intrauterine contraceptive devices. They are good choices for breastfeeding mothers who wish to use birth control medications and they are highly effective in preventing pregnancy. They work by keeping the ova from implanting in the endometrial lining of the uterus. Unlike other progestins, the progesterone intrauterine contraceptive device delivers its hormone directly to the endometrial lining of the uterus. As a result it is very effective and has fewer side effects. The progesterone intrauterine contraceptive device can be used four to six weeks after the baby is born. There is controversy whether progesterone only contraceptives affect breast milk supply. In one study women taking the minipill actually had a higher than average milk supply.[6] However, other studies have showed the minipill caused a 12% drop in milk supply. [6] There is some concern that even progestin only birth control can cause low milk supply in certain women. This is because physiologically a natural drop in the hormone progesterone after child birth stimulates the milk making process. It is therefore recommended that women wishing to commence on progesterone only pills are advised to wait for three days after the baby is born.[6] If desired waiting for six weeks may prevent even more milk let down complications.

Lactational amenorrhea method (LAM)

LAM is a good option for mothers who do not want to take birth control pills during the early postpartum period after the baby is born. LAM has been found to be 98% effective [8] as long as;

i. The baby is less than six months old.
ii. The baby is exclusively breastfeeding.
iii. The baby does not exceed four hours in the daytime and six hours at night in between feeds.
iv. The mother has not had her first menstrual period after delivery.[8]

When these conditions are not all met another birth control method should be used otherwise the mother might get pregnant. LAM cannot be used as a method of contraception if breastfeeding and formula feeding are combined. Breastfeeding must therefore be exclusive and regular. The return of fertility varies from mother to mother. Even with 100% breastfeeding some have their first period within a few months after childbirth. Others will not have their period for twelve months or longer even if the baby does not breastfeed regularly. Vaginal bleeding/discharge after delivery (lochia) usually lasts two to four weeks followed by a period of amenorrhoea. The first menstrual bleeding afterwards usually is a sign that fertility has returned. No matter the age of the baby once a mother has had her menstrual period after delivery she must use another form of contraception to avoid getting pregnant. [9]

Permanent sterilization

This method is highly effective. They are considered if the couple have achieved the desired family size and do not want to have any more children. Women who have undergone bilateral tubal ligation are able to continue nursing their infants as soon as they are able to do so after surgery.[9]

Contraceptive use in Kenya and other regions

Although fertility rates have declined during the last few decades the unmet need for family planning still remains substantial.[10] It is estimated that over 120 million women in developing countries who do not want to have any more children are not using any family planning method even though they are sexually active.[11] While the contraceptive prevalence for developed countries has been estimated to be as high as 72% it has remained less than 20% in Sub-Saharan African countries.[11] Sub-Saharan Africa has the lowest rate of contraceptive use in the world.

Several factors have contributed to this low rate. These include difficulty in getting contraceptive supplies, inadequate family planning clinics, largely rural population, low socioeconomic levels, high rates of infant and child mortality and high values many cultures place on large family size. The percentage of married women of reproductive age using contraception ranges from 4% in the Niger to 48% in Zimbabwe and 75% in Mauritius.[12] As contraceptive use increases total fertility rates decreases. In Kenya the contraceptive use increased three fold during the 1980's. The rise was associated with a decrease in family size from 8.3 to 6.5 children per woman. [12] The national contraceptive prevalence in Kenya has stagnated at 39%. This was after the contraceptive prevalence rose from 32% to 39% between 2003 and 2008.[13] Kenya still has a large unmet need for family planning services estimated at around 25% in 2008. [13] Nearly 42% of Kenya's population (16.5 million) was estimated to be under fifteen years in 2008 and an estimated 100.000 young people turn 16 years every year. This pattern will continue for the next decade thereby putting a heavy demand on reproductive health including family planning. [13]

In Kenya many women have limited access to family planning during the immediate postpartum period. Few studies have been done to assess the knowledge and use of family planning on sexually active breastfeeding mothers. In a study done in 1992 on missed opportunities in postpartum family planning in Kenya a total of 1000 mothers were recruited. 800 of the mothers were interviewed at the prenatal clinic and the child welfare clinic while 200 subjects had just given birth in hospital. 90% of the mothers expressed the desire to use a contraceptive method in the postpartum period despite the fact that majority had not used any family planning method at all.[14] Only 20% of the women had been exposed to family planning practices before and only 2% were discharged with a family planning method. [14]

In the Kenya demographic health survey 2003, it was estimated that more than 27% of the 20 to 29 year old women had less than 2 years between their last two pregnancies. 23% of the births were at intervals of greater than 24 months and many of these births were unintended. 68% of postpartum mothers had unmet need for family planning during the first year with only 23% using a method. [15] However, currently the average fertility rate in Kenya has been approximated to be about 4.9 births per woman.[15] The fertility rate has not changed much in the last 15 years. It is also estimated that almost 1 in 4 births occur less than two years after the preceding birth.[13]

The high number may be due to the fact that the prescribed time of resuming sexual activity after childbirth is one to six weeks even though most couples will resume sexual activity immediately after childbirth.[13] Integrating family planning with other health services during and after pregnancy can help increase access to and use of contraception thereby decreasing unmet need to prevent unintended pregnancies.[13, 15]

Data from twenty seven surveys in developing countries were conducted as part of the demographic and health surveys between 1993 and 1996 to assess intentions to practice contraception and to determine if there was any unmet need concerning the use of contraception among breastfeeding mothers in the first year after birth. Unmet need in the survey focused on future wishes on births and pregnancies rather than past pregnancies. Across the twenty seven countries there was much unsatisfied interest in contraception and unmet need for contraception in two thirds of women who were within one year of the last birth.[16] 40% of the women said they planned to use a method in the next 12 months but they were not currently doing so. In conclusion it was felt that women who had recently given birth needed specialized attention from family planning and reproductive health programmes if they wanted to decrease the number of unwanted births and abortions and to lengthen subsequent birth intervals.[15] Several other studies demonstrated that the unmet need for contraception was highest among postpartum mothers in the first year after delivery. On average two thirds of these women did not want to become pregnant but were not using a contraceptive method and nearly 40% planned to use a method but had not done so.[17] There was a clear need to increase the availability and access to family planning services during the extended postpartum period (i.e. for the first twelve months after delivery). It was not clear on how it was best to do this. While some women were able to obtain family planning from a dedicated family planning service delivery outlet many women were not able to understand the contraceptive options available during this period and how to access them.[17]

The Kenya Demographic Health Survey 2008/2009 reported that 96% of sexually active women interviewed knew at least one modern method of family planning, while 61% had ever used a modern method.[13] However only 39% of the married women were currently using a modern method.

In a cross sectional descriptive study conducted in Nigeria in 2005 among 157 sexually active breastfeeding mothers six to nine months after childbirth, the current contraceptive use was quite low (13%) while contraceptive awareness was quite high (95.5%).[11]This demonstrated that even though the awareness of family planning was high there was limited use of family planning among the sexually active breastfeeding mothers. The proportion of mothers who planned to use family planning methods in future among the non users was also quite high (64%). All the current non users (86.6%) met the criteria for unmet need for family planning because they were sexually active and either did not want to have any more children or preferred to delay their births until a later date. Majority of the non users of contraception depended only on the perceived contraceptive effects of breastfeeding.

Due to the fact that sexually active breastfeeding women are at risk of getting unplanned pregnancies especially if they are not on an effective contraceptive method, it is important to determine knowledge and factors influencing utilization of family planning methods among these women in order to develop targeted programs to increase their use.

RATIONALE/ JUSTIFICATIION OF THE STUDY

Many breastfeeding women depend on lactational amenorrhea as a means of contraception. However, lactational amenorrhea is only effective if the baby is less than six months old, exclusively breastfeeding, and duration between feeds does not exceed four hours in the daytime and six hours at night.[8] nonetheless the duration of suppression of ovulation during breastfeeding is variable. Nearly 80% of breastfeeding mothers ovulate before the return of their first menstrual period which may even occur as early as two months after delivery making sexually active breastfeeding mothers to be at risk of unintended pregnancies if they are not a contraceptive method.[5] It is estimated that among women not using contraception after childbirth, 50% of pregnancies occur within six months of resumption of intercourse.[5]

Sexually active breastfeeding mothers are therefore at risk of unintended pregnancies after childbirth unless they are on an effective contraceptive method. Majority of women visit the Maternal and Child Health clinic in the first year after delivery for infant vaccination and weighing. Family planning services are also provided in the same clinics and usually by the same health care providers. It would be expected that since the mothers come into contact with health care workers providing family planning services during these clinic visits, knowledge and use of contraception would be high. However, the high pregnancy rates reported among these women during the first year after delivery indicates that this is not usually the case. It is not clear whether breastfeeding mothers are fully aware of the need to utilize family planning services and or have adequate knowledge about family planning methods they can use. To our knowledge, no study has been conducted among sexually active breastfeeding mothers within one year of delivery to determine knowledge about family planning and to determine reasons that limit the utilization of family planning services. The findings of this study will therefore be used to promote use of family planning services among sexually active breastfeeding women and thereby avoid unintended pregnancies.

RESEARCH QUESTION

Does the level of knowledge of family planning methods among sexually active breastfeeding mothers after delivery determine the extent of utilization of family planning methods?

CONCEPTUAL FRAMEWORK

Narrative:

Breastfeeding women who resume sexual activity after childbirth are at risk of pregnancy even though they experience lactational amenorrhea which may last for 6 to 12 months. Sexually active breastfeeding women after delivery may mistakenly believe that since they have not resumed their menses they are not at risk of pregnancy and do not need to use any family planning method.

This study was a prospective descriptive study to determine the knowledge and use of contraception among sexually active breastfeeding women within the first year after delivery at the MCH clinic of Mbagathi District hospital.

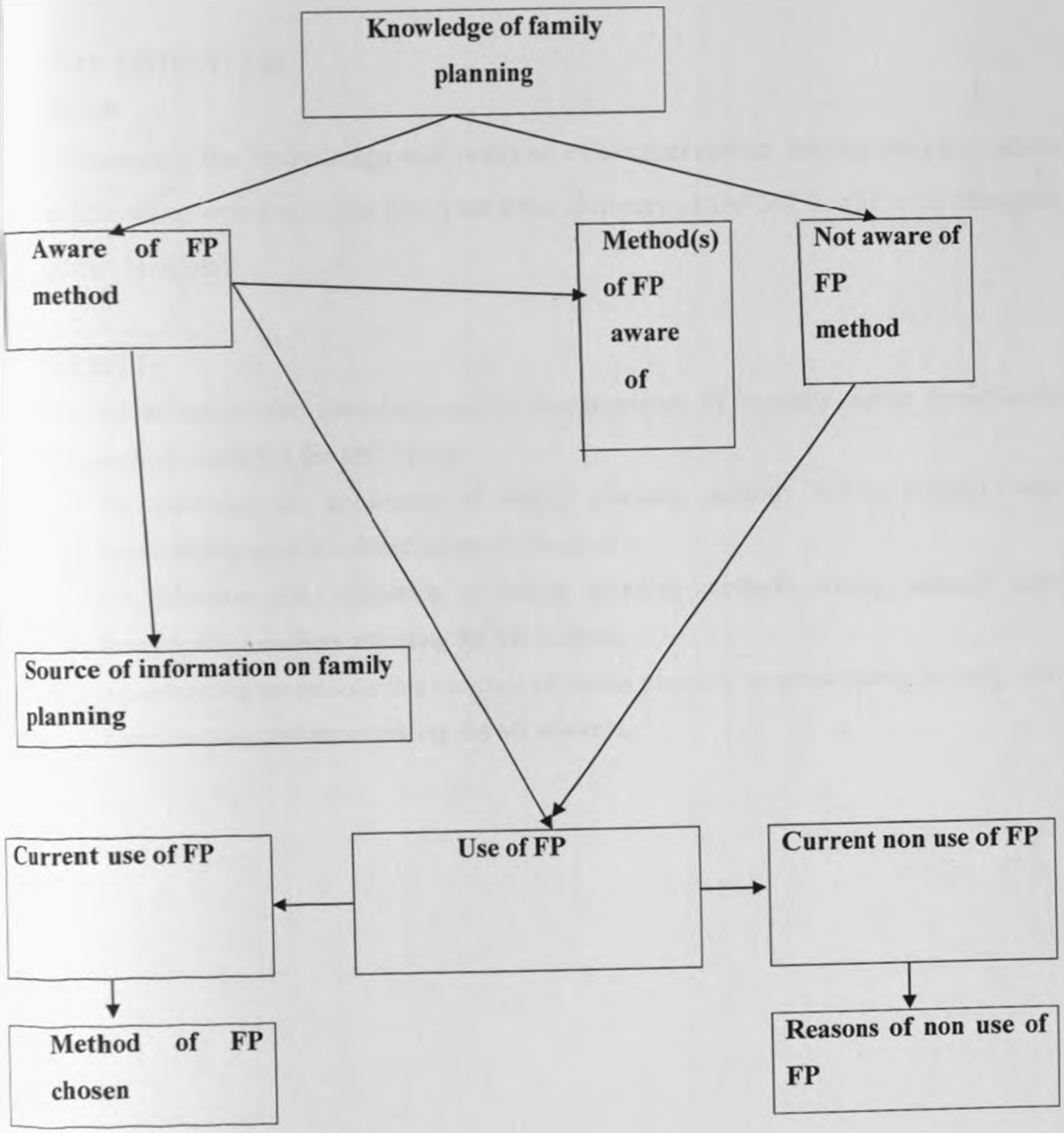
Outcome variables that were measured included the knowledge of family planning methods. The respondents were asked if they were aware or not of family planning methods they were aware of. The method(s) of family planning they were aware of and the source of information of family planning methods.

The use of family planning methods was measured by determining the current and previous use of family planning methods. Among the current users the type of family planning method adopted was measured. The influence of sociodemographic characteristics among the current users and non users was also measured.

The reasons for non use of family planning method were determined among the current non users of family planning.

The results drawn from these findings would give rise to conclusions and recommendations that will promote the use of family planning services among sexually active breastfeeding women and thereby avoid unintended pregnancies.

Diagramatic conceptual framework



STUDY OBJECTIVES

BROAD:

To determine the knowledge and practice of contraception among sexually active breastfeeding women in the first year after delivery at the MCH clinic of Mbagathi District Hospital.

SPECIFIC:

i. To determine the sociodemographic characteristics of sexually active breastfeeding mothers attending the MCH clinic

ii. To determine the knowledge of family planning methods among sexually active breastfeeding mothers attending the MCH clinic.

iii. To determine the utilization of family planning methods among sexually active breastfeeding mothers attending the MCH clinic.

iv. To determine the reasons that limit use of family planning services among sexually active breastfeeding mothers attending the MCH clinic.

METHODOLOGY

STUDY SETTING:

Location

The study was conducted at the MCH clinic at the Mbagathi District Hospital in Dagoretti Division, Nairobi West District of Nairobi County in Kenya.The hospital is located about four kilometers from the city centre along Mbagathi Road which is off Ngong Road.

Administrative areas

Administratively the hospital is under Ministry of Medical Services. Its main catchment area is the Kibera slums. It is the only general district hospital in Nairobi County and therefore it serves people from other areas as well.

Structural organization

The hospital is under the ministry of medical services which is headed by the minister of medical services. Being in Nairobi County it falls under the provincial director of medical services, Nairobi. The medical superintendent is the overall head of the hospital. Under the medical superintendent there are heads of department who are in charge of the various units in the outpatient and inpatient areas. There is an overall chief nurse under the medical superintendent who is in charge of all the nursing services.

Functions/Health services offered

The hospital offers curative, palliative and rehabilitative services. The outpatient department has the Maternal and Child Health/ Family Planning clinic (MCH/FP), Antenatal clinic, Dental clinic, Eye clinic, Pediatric clinic, HIV clinic, Surgical clinic, Skin and Tuberculosis (TB) clinic. In the MCH/FP clinic the services offered include growth monitoring and promotion, immunization and integrated management of childhood illness. The family planning clinic offers all methods of family planning.

Other outpatient services offered include HIV counseling and testing, antiretroviral therapy and prevention of mother to child transmission of HIV. The tuberculosis clinic is a centre of tuberculosis diagnosis with laboratories and tuberculosis drugs. There is also a radiology

department where radiology services are offered. In the outpatient department there is also a centre where rural health training is carried out.

The inpatient department is made up of six wards. These include the maternity, medical, pediatric, surgical, eye and T.B wards. The hospital has a total bed capacity of 200 beds and ten baby cots. In the maternity wards basic/comprehensive emergency obstetric care are offered. Emergency and elective caesarean sections are also done in the hospital.

Maternal and Child Health clinic/FP (MCH)

The services offered at the clinic include family planning, antenatal services, postnatal services, child immunization and prevention of mother to child (PMTCT) services. The total number of revisits and new cases seen in the clinic yearly is about 10758 and 2973 respectively as per 2009 data. The MCH clinic is divided into six rooms were each of the services is offered. There is also an administrative room where the nurse in charge of the clinic is stationed.

The MCH/FP clinic was chosen as the site of this study because the location of the hospital serves a large catchment area with mothers from both poor and middle socioeconomic background utilizing the hospital facilities. This because the hospital is located next to the populous Kiberia slums. There are also various estates surrounding the hospital with a large population who are able to utilize the hospital facilities easily. The hospital is also near the city centre and is easily accessible. The costs of the services at the hospital are also affordable being a government hospital. These reasons made the site suitable for the study since the respondents who were interviewed were from different socioeconomic backgrounds.

STUDY DESIGN:

A prospective descriptive study design was used to determine the knowledge and utilization of family planning methods and reasons that limit the use of family planning services among the sexually active breastfeeding mothers attending the MCH clinic.

STUDY POPULATION/ RESPONDENTS

In this study two hundred and six sexually active breastfeeding mothers who had attended the MCH/FP clinic were interviewed. Only those who were one year and below after delivery were considered for the study.

SAMPLE SIZE DETERMINATION

The minimum size required in this study was calculated according to the WHO formula: [18]

$$n = \frac{N * Z^2 * p * (1-p)}{d^2 * (N-1) + Z^2 * p * (1-p)}$$

Where n was the minimum sample size required

N was the expected eligible number of MCH attendants in a year

Z was the standardized score at 95% two tail level to 1.96.

P was the contraceptive prevalence taken at 13%. According to K T Ijadunola et.al the current contraceptive use among breastfeeding mothers attending an infant welfare clinic in Nigeria was found to be 13 %.[11]

d was the level of precision taken at 0.05

Calculating the sample size:

$$n = \frac{10758 * 1.96^2 * 0.13 * 0.87}{0.05^2 * (10758-1) + 1.96^2 * 0.13 * 0.87}$$

$$n = \frac{4674.20}{0.05^2 * (10757) + 1.96^2 * 0.13 * 0.87}$$

$$n = \frac{4674.20}{26.89 + 0.43}$$

$$n = \frac{4674.20}{27.32}$$

$$n = 171$$

Adding 20% expected non response rate a sample size of **206** mothers was obtained which was taken as the number of breastfeeding mothers to be interviewed in this study.

DATA COLLECTION

Data was collected by means of a questionnaire with closed and open ended questions. The structured questionnaire was administered to the women by the researcher and a trained research assistant using a face to face interviewing technique. Sexually active breastfeeding women in their first year after delivery bringing their infants for immunization or seeking various other services in the clinic were targeted for the study. A trained research assistant assigned to the study and a nurse stationed at the MCH clinic assisted the researcher to carry out the study. The registration clerk at the clinic assisted by the nurse assigned to the study identified mothers with infants who were one year or below and had resumed sexual activity. The selected mothers were then invited to participate in the study.

To avoid bias a random selection was done whereby every third mother presenting to the clinic with an infant one year or below were selected to participate in the study. At the MCH clinic approximately forty to fifty mothers attend the clinic daily, therefore only eight mothers were interviewed on each day to avoid bias after immunization of their infant or after receiving the services they came for in the clinic. The purpose of the study was then introduced to the selected mothers individually. It was clarified to each of them that only those mothers who had resumed sexual activity after child birth were eligible for the research study. At this point the consent of the research study was introduced to the mother in detail. The mother then gave her consent by signing the consent form. Once the consent had been obtained the mother was enrolled into the study. The questionnaire was then introduced to the mother. Study numbers were used in the questionnaire in order to conceal the mother's identity. The researcher and his assistant then introduced each question step by step for the mother to answer. The questionnaire was divided into four main areas. These were study identification, socio demographic characteristics, and respondents' knowledge about of family planning utilization of family planning services. To minimize contamination or re-interviewing of mothers the registration number on the cards the

mother used to attend the clinic were noted down and kept confidential. On each interview day the registration numbers were counter checked to ascertain that a mother was not interviewed more than once. Two hundred and six mothers met this criterion during the period of data collection from 15th May to 15th June 2011. Six mothers declined to participate in the study citing lack of time for the interview.

DATA MANAGEMENT

The data obtained was coded and entered into a microcomputer using the EPI – INFO system and proportions used to describe the data. Data was analyzed using SPSS statistical software package for personal computers. Variables were expressed as percentages and presented as frequency tables and cross tabulations. Chi square was employed as the test of association between proportions of respondents.

ETHICAL CONSIDERATIONS:

The study was considered safe and friendly since no invasive procedures were involved.

The mothers were assured of confidentiality of the information given.

STUDY LIMITATIONS

The study did not take into account the sexually active breastfeeding mothers who were not attending the clinic but had resumed sexual activity after delivery with no contraceptive method since they were not interviewed.

The data that was obtained from the mothers could not be independently verified.

RESULTS

The principle investigator carried out a descriptive prospective study at Magahi District Hospital between March to and May 2011. This was in an urban set up. A total of 206 respondents were interviewed to in order to determine their knowledge and use of family planning in the first year of delivery after having resumed sexual intercourse. The sociodemographic characteristics of the sexually active breastfeeding mothers are shown in table one below.

Table 1 Socio demographic characteristics of study participants (N=206)

Socio demographic variable	Number (%)
Age (years) (N=206)	
15-20	81 (39.3)
21-30	39 (18.9)
31-40	59 (28.6)
41-44	46 (13.1)
Marital status (N=206)	
Single	78 (37.9)
Monogamous married	84 (40.7)
Polygamous married	8 (3.9)
Divorced/separated	36 (17.5)
Educational level (N=206)	
None	0 (0.0)
Primary	6(2.9)
Secondary	73(35.4)
College/university	127(61.7)
Parity (N=206)	
1-2	116(56.3)
3-4	71(34.5)
5+	19(9.2)
No. of children alive (N=206)	
1-2	120(58.3)
3-4	68(33.0)
5+	18 (8.7)
Religion (N=206)	
Catholics	54 (26.2)
Protestants	104 (50.4)
Islam	48 (23.3)

The respondent's age ranged from 15 to 44 years with a mean 24.7± 2.4 years. Majority (58.2%) were aged less than 30years while 28.6% were aged between 31 to 40 years. Mothers who were aged between 41 to 44 years were 13.3%. The youngest respondent was aged 15 years while the oldest was aged 44 years.

Most of the respondents came from a monogamous marriage (40.7%). Single mothers constituted 37.9% of the respondents. Only 3.9% of the respondents interviewed came from a polygamous marriage while 17.5% of the respondents were separated from their spouses.

All the respondents interviewed had obtained some formal education. Primary level of education had been attained by 2.9% while 35.4% had reached secondary level. The respondents who had attended either college or university education were 61.7%. Therefore the educational level of the respondents was therefore adequate.

Most of the respondents had been pregnant either once or twice i.e. 56.3% while only 9.2% of the respondents had been pregnant five times. The rest 34.5% had gotten pregnant either thrice or four times. Majority of the respondents who were interviewed i.e. The number of respondents having one or two living children was 58.3% with 18(8.7%) of the respondents having at least five or more living children. The respondent with the highest number of living children was six.

The most common religion was Protestant i.e. 50.4%, while 23.3% came from the Islamic faith. The rest 26.2% were Catholics.

The awareness of family planning methods among the respondents was determined and the results are shown in table two below.

Table 2: **Family planning awareness/knowledge among respondents (N=206)**

VARIABLE	NUMBER (%)
Awareness (N =206)	
Aware	206(100.0)
Not aware	0(0.0)
***Source of information about family planning (N=206)**	
Healthcare facilities	181(87.9)
Radio and television	106(51.5)
Friend/relatives	49(23.8)
Posters/billboards	17(8.3)
Newspapers/magazines	10(4.8)
***Types of family planning respondents aware of**	
Oral pills Injectables	206(100)
IUCD's	205(99.5)
Condoms	204 (99.0)
Implants	188(91.3)
Natural method	199(96.6)
Permanent sterilization	193(93.7)

*Some respondents gave multiple responses

All the respondents interviewed (100%) were aware of at least one or more method of family planning, which they had received from various sources. Majority of the respondents obtained their information from health care facilities i.e. 87.9%, while 51.5% had obtained information on family planning from the radio and television. 8.3% of the respondents obtained information on family planning from posters and billboards. Only 4.8% of the respondents obtained information on family planning from newspapers and magazines. All the respondents were aware of at least one or more method of family planning. In this study all the respondents i.e. 100% were aware of oral contraceptives as a method of family planning.

The principal investigator then sought to find out how many of these respondents were currently on family planning method, since they were sexually active breastfeeding mothers. The results are depicted as shown in table three.

Table 3: Current family planning use among respondents

Variable	Number (%)
Current use of family planning (N= 206)	
Users	28(13.8)
Non users	178(86.2)
Family planning methods among current users (N=28)	
Oral pills	10(35.7)
Injectables	5(17.8)
IUCD's	6(21.4)
Male condom	4(14.2)
Natural method	2(7.1)
Permanent sterilization	1(3.5)

Despite the high level of awareness (100%) only (13.8%) of the respondents were current users of a family planning method. There awareness of family planning was therefore high and the use low.

Among the 28 respondents who were current users of a family planning method, majority of them 35.7% were on oral contraceptive pills. 21.4% of the respondents were using IUCD's while 17.8% were on inject able contraceptives. 7.1% of the respondents had opted to use the natural method of family planning. One of the respondent i.e. 3.5% had undergone bilateral tubal ligation (permanent sterilization).

The use of family planning methods previously before their last pregnancy was also determined. This was to compare the previous and current use of family planning among the respondents and to determine if there were complications associated with family planning previously. The results are shown in table three below.

Table 3: Previous family planning use and complications among respondents

Variable	Number (%)
Previous use of family planning (n=206)	
Users	59(28.6)
Non users	147 (71.4)
Complications of family planning among previous users (n=59)	
Irregular menses	12(20.3)
Heavy menses	22(37.3)
High blood pressure	8(13.6)
Persistent headache	14(23.7)
Conceived on family planning method	3(5.1)

28.6% of the mothers were previous users of family planning while 71.4% had not used any method of family planning previously. This was less than the current use of family planning because only 13.8% of the mothers were on a family planning method currently.

Most of the respondents (37.3%) that had used a family planning method previously reported that they had experienced heavy menses while on a family planning method. 5.1% of the respondents. 20.3% while 23.7% of the respondents experienced headache while on a family planning method. 20.3% of the respondents reported having had irregular menses while 5.1% of the mothers had conceived while they were on a family planning method.

The principle investigator also determined the desire to use a family planning in the future among the respondents. The response is as shown in table four.

Table 4: Future family planning use among respondents (N=206)

Variable	Number (%)
Response to future use of family planning	**(N=206)**
Yes	132(64.1)
No	74 (35.9)

Majority of the respondents 64.1% said they would desire to use a family planning method in the future. This was despite the fact that only 13.8% were current users of a family planning method.

The principal investigator sought to determine the reasons that limit the use of family planning among the current non users of family planning. The respondents gave several reasons why they

were not on a family planning method. Table 5 shows the reasons for non use of a family planning among the respondents.

Table 5: Reasons for non use of family planning methods among current users (n=178)

*Reasons for non use of FP	Number (%)
Believed in breastfeeding as form of protection	154 (86.5)
Waiting for resumption of menses	112 (62.9)
Feared side effects	54 (30.3)
Spouse declined	23(12.9)
Religious reasons	12(6.7)
No reason given	12 (6.7)

*Some respondents gave multiple responses

The reasons for not using a method of family planning method among the current non users were several. Most of the respondents interviewed (86.5%) believed that breastfeeding acts as a form of protection and therefore prevents them from getting pregnant. Even though they had resumed sexual activity they believed as long as they were breastfeeding they would not get pregnant. 62.9% of the respondents believed that they were safe because they had not resumed their menses after delivery. 30.3% feared that use of family planning method will expose them to undesirable side effects among the current non-users of family planning methods, 12.9% of the respondent reported that their spouse had declined to allow them to commence on a family planning method after delivery. 6.7% of the respondents were not using any family planning methods due to religious reasons. 6.7% of the respondents gave no reason for not using a method of family planning currently.

The family planning acceptance (use) by socio-demographic characteristics of respondents was further analyzed to determine their association with family planning use. The results are shown in table six below.

Table 6 Family planning acceptance (use) by socio-demographic characteristics of respondents

Socio demographic characteristics	F P use (Number)			Df	X^2	P -value
Age (years) (N=206)	Yes (N=27)	No(N=179)	Total (N=206)	3	0.926	0.819
≤20	1	12	13			
20-29	16	117	133			
30-39	9	45	54			
≥40	1	5	6			
Marital status(N=206)	Yes (N=37)	No(N=169)	Total (N=206)	3	0.028	0.868
Single	20	58	78			
Monogamous married	15	69	84			
Polygamous married	1	7	8			
Divorced/separated	1	35	36			
Educational level (N=206)	Yes (N=36)	No(N=170)	Total (N=206)	3	0.083	0.994
None	1	15	16			
Primary	11	33	44			
Secondary	20	102	122			
College/university	4	20	24			
Parity(N=206)	**Yes (N=28)**	**No(N=178)**	**Total (N=206)**	**2**	**8.117**	**0.015**
1-2	9	108	117			
3-4	18	52	70			
5+	1	18	19			
No. of children alive(N=206)	**Yes (N=30)**	**No(N=176)**	**Total (N=206)**	**2**	**7.630**	**0.026**
1-2	10	104	114			
3-4	18	55	73			
≥5	2	17	19			
Religion (N=206)	Yes (N=28)	No(N=178)	Total (N=206)	3	0.578	0.911
Catholics	3	22	25			
Protestants	17	96	113			
Islam	8	60	68			

Table 6 depicts family planning acceptance by selected socio-demographic characteristics of the respondents. The age, marital status, educational level, parity, number of children alive and religion were used to determine if they influenced the acceptance (use) of family planning among the current users. The likelihood ratio chi-square was quoted wherever there was a cell frequency that was less than 5. There was no association between the acceptance (use) of family planning and the age, marital status, educational level nor religion ($p > 0.05$). The parity and the number of children alive were significantly associated with current family planning use ($p < 0.05$).

On parity, the mothers who had delivered between three to four children were more likely to adopt a conventional method of family planning compared with mothers with less than three children or more than four children ($p = 0.015$). On the number of living children, the mothers who had 3 to 4 living children were more likely to be current users of family planning compared to mothers with less than three children or more than four children ($p= 0.026$).

While family planning acceptance (use) was not significantly related to religion, ($p< 0.911$) it was noted that the use was higher among Protestants compared with catholics and Muslim women.

DISCUSSION

The respondent's age ranged from 15 to 44 years with a mean 24.7± 2.4 years. Majority (58.2%) were aged less than 30years while 28.6% were aged between 31 to 40 years. Mothers who were aged between 41 to 44 years were 13.3%. The youngest respondent was aged 15 years while the oldest was aged 44 years. These results show that most of the mothers who were attended the MCH clinic were of the younger age group i.e. they were aged less than 30 years. Generally it is assumed that mothers who are older tend to have more children and therefore they are expected to attend the MCH clinic more frequently for various services including family planning services.

Most of the respondents came from a monogamous marriage (40.7%). This was expected considering the fact that due to difficult economic challenges it would be difficult to maintain a polygamamous family set up. The study was also conducted in an urban setup were most of the families generally tend to be monogamous unlike in a rural setup. Single mothers who constituted 37.9% of the respondents were also considered to be of a high number. Since most of the mothers in this study were of the younger age group it was expected that majority of them preferred to raise their children as single mothers due to changing sociocultural values.

All the respondents interviewed had obtained some formal education with majority of the respondents attaining either college or university education i.e. 61.7%. These results show that the significance of education in the society has been accepted. With the high level of education the knowledge and practice of family planning was therefore expected to be also high among the respondents.

The results showed that 58.3% of the respondents had one or two living children, while 8.7% having at least five or more living children. Therefore most of the mothers who participated in this study were from households with smaller families. This could be explained by the fact that majority of the study participants were aged thirty years and below.

In this study most of the study participants were Christians i.e. Protestants and Catholics constituted 76.6%. This was expected because the study site is located in a predominantly Christian community. However Muslims who constituted 23.3% was also considered to be a high number and therefore the catchment area in the community had people from various interdenominational backgrounds.

The study revealed that the awareness/knowledge of family planning was 100%. The respondents had obtained information on family planning from various sources. Most of the respondents had obtained their information healthcare facilities i.e. 87.9% which are readily available and accessible in an urban setup. The study participants also came from an area with adequate telecommunication availability and therefore access to radio and television was within reach to all of them. The high awareness of family planning could therefore be attributed to the fact that being in an urban setup information was readily available. Most of the respondents that were interviewed had attained college /university status 61.7%. Their educational status could have exposed them to various sources of information on family planning. The study setting was also located in an urban setup were information was readily available.

Despite the high level of awareness (100%) only 13.8% of the respondents were current users of a family planning method. There was a wide gap between the awareness and use of family planning among the study participants. These results showed that the level of knowledge of family planning methods among sexually active breastfeeding mothers after delivery does not determine the extent of utilization of family planning methods.

Similar findings have been documented by other authors who have done research on contraceptive use among sexually active breastfeeding mothers. In a cross sectional descriptive study conducted in Nigeria in 2005 among 157 sexually active breastfeeding mothers six to nine months after childbirth, the current contraceptive use was noted to be quite low (13%) while contraceptive awareness was quite high (95.5%).[11]

Majority of the current users of family planning i.e. 35.7% preferred oral contraceptive pills. Oral contraceptive pills are readily available and cheaper. 7.1% of the current users of family

planning had opted to use the natural method of family planning which is unreliable with a high failure rate and therefore they were exposed to getting pregnant. From the results only one of the respondents had undergone bilateral tubal ligation (permanent sterilization). This could be explained by the fact that most of the mothers that had been interviewed either had not achieved the desired family size or preferred the temporary method of family planning.

The previous users of family planning were 28.6% of the mothers while only 13.8% were current users. These results show that the gap between family planning knowledge and use has even widened since more respondents were using a method of family planning previously as compared to the current situation. Maybe this could also be explained by the fact that most of the respondents had experienced some complications with the methods that they were previously using with most of the previous usres experiencing heavy menses.

However it was noted that majority of the respondents 64.1% said they would desire to use a family planning method in the future. It was not clear why most of the mothers desired to use a method of family planning in the future while currently most of them were not on a conventional method.

The current nonusers of family planning gave several reasons why they were not on a family planning method. Majority of them (86.5%) believed that breastfeeding provided them natural protection against pregnancy. Studies done have shown that for a mother to effectively benefit from lactation amenorrhea as a form of natural family planning the infant feeding practices are rigorous. Some studies have shown a woman is naturally protected against pregnancy when her baby gets 85% of his or her feeds as breast milk, she breastfeeds her baby often day and night, her menses have not returned and her baby is less than six months old. [3] If she keeps breastfeeding very often her protection from pregnancy may last longer than six months and perhaps as long as nine to twelve months. If lactational amenorrhea is used effectively failure rate is two pregnancies per a hundred women in the first six months after childbirth.

A woman who uses lactational amenorrhea should therefore be encouraged to breastfeed often at least eight to ten times a day, including at least once at night. Day time feedings should not be

greater than four hours apart and night time feedings not less than six hours apart. There should be good breastfeeding techniques and good maternal diet. If these conditions cannot be met she is encouraged to start a modern family planning method.[3] Other studies have suggested that for lactational amenorrhea to be effective the mother has to practice exclusive breastfeeding for six months since supplemental feeding may alter the pattern of lactation and the intensity of infant suckling which may secondarily affect suppression of ovulation.[4, 5]

The mothers who had not resumed menses and believed that they were safe from getting pregnant since they had not resumed their menses were 112(62.9%). Several studies have shown that the lack of return of menses does not offer protection against pregnancy since there is no way of predicting when menstruation or ovulation will resume following a delivery. On average after childbirth, fertility will return four to six weeks for those not breastfeeding and twenty four weeks for those breastfeeding.[5] It is estimated that 50% of pregnancies occur within six months of resumption of intercourse after childbirth for those not on a method of family planning. [5] Nearly 80% of breastfeeding mothers ovulate before the return of their first menstrual period and this may even occur as early as two months after delivery making sexually active breastfeeding mothers to be at risk.[5] Therefore these mothers were at risk of getting pregnant.

From the results of this study spousal influence i.e. 12.9% also contributed to the non use of family planning among the current users. This showed that male spouses play an important role in the use of family planning because of their influence. Religion also affects family planning use to some extent and due to their beliefs, some mothers decline to utilize conventional method of family planning. This therefore acts as a hindrance. In this study 6.7% of the current non users were not a method of family planning due to their religion.

In this study the parity of the mother and the number of living children were the only socio demographic variables that were found to be significantly to the use of family planning among the respondents i.e. p=0.015 and 0.026 respectively. Mothers who had delivered between three to four children were likely to adopt a conventional method of family planning. This could be explained by the fact that the mothers who were of a lesser parity (> 3) were less motivated to

use a method of family planning because of the small family size. On the other hand mothers who had more than five or more children were of the older age group and may have perceived that they were at less risk of getting pregnant again and therefore tended not to adopt a conventional method of family planning. Other sociodemographic variables such as age, marital status, educational background and religion were not significantly related to family planning among the current users.

CONCLUSIONS

1. The results revealed a high level of contraceptive awareness but a low level of contraceptive use among recently delivered mothers of breastfeeding infants with majority of the non users depending on the contraceptive effects of breastfeeding. The high level of awareness of family planning therefore had no impact on the use of family planning.
2. Sexually active breastfeeding mothers believe that lactational amenorrhea offers them natural protection against getting pregnant which puts them at risk to get pregnant.
3. There is also an unmet need concerning future use of family planning due to the fact that most mothers desired to use a family planning method in the future but were not using one currently.
4. Parity and the number of living children are significantly related to the utilization of Family planning services.

RECOMENDATIONS

1. It is recommended that breastfeeding mothers be targeted for an urgent behavior communication intervention with regards to contraceptive acceptance and use. The intervention should not be limited to the health facilities alone but should be population based with community involvement. During such interventions the unreliability of breastfeeding as a primary form of family planning should be emphasized.

2. Health education on the need to adopt family planning methods should be clearly emphasized in the antenatal clinics, immediately after delivery and in the maternal/ child healthcare clinics.

3. Mothers should be encouraged to adopt a conventional method of family planning regardless of their parity and the number of living children

4. A much larger interventional study involving a larger population should be undertaken in order to emphasize on the need to address family planning among sexually active breastfeeding mothers. This should involve both the urban and rural populations

REFERENCES:

1. Dutta. D.C, 2001, Textbook of Gynaecology; **Contraception,** 3rd Edition, Chapter 28 pp 435.
2. Alan H. De Cherney and Martin L. Pernoll, 1998, Current Obstetrics and Gynecology, **Contraception and family planning**; 8th Edition, Chapter 33 pp 670.
3. Robert H et al, **The essentials of contraceptive technology. A handbook for clinical staff.** July 1997.
4. Kennedy K I, Visness C M, 1992 **Contraceptive Efficacy of Lactational Amenorrhea.** Lancet. pp 227.
5. Rustein S O, 1991. **The Impact of Breastfeeding on Fertility; In Demographic and Health Survey:** 1: pp 403- 428.
6. Koetsawang S et.al, **The effects of contraceptive methods on the quality and quantity of breast milk**. Int J Gynaecol & Obstet 1987;25 suppl; pp115-127.
7. Croxatio H B, et.al **Fertility regulation in nursing women and the long term influence of low dose COC's initiated at day thirty postpartum upon lactation and infant growth.** 1983: 27(1): pp 27-28.
8. Labbock M H et.al, **Multicentre study in Lactational amenorrhea method: efficacy, duration and implications for clinical application. Contraception** 1997.
9. Nichols Johnson, **Breastfeeding and Contraception:** 2001, 21(2): pp 11-12.
10. Metha S, 1994, **Contraception and Women's Health**. Int. J. Gynaecol. Obstet, 46 pp 165-171.
11. K.T. Ijadunola, E.O. Orji and F.O. Ajibade. 2005. **Contraceptive Awareness and Use among Breastfeeding Mothers in Ife Ife, Nigeria,** EAMJ Vol 82.pp 4-7.
12. Manual of CDC, **Family planning methods and practice in Africa**. 1999 Edition: pp14.

13. Kenya Demographic Health Survey (KDHS), 2008/2009. **Fertility and Family Planning.** Central Bureau of Statistics.

14. J Bradley, **Missed opportunities: Postpartum family planning in Kenya**. AVSC (1993), vol 31; issue 3 page 7.

15. Kenya Demographic Health Survey (KDHS), 2003/2004. **Fertility and Family Planning.** Central Bureau of Statistics.

16. Joera A Ross& William L Winfrey, **Internal Family Planning Perspectives**. 2001:27(1) pp 20-27.

17. Ricardo Vernon, Ian Askew; **Delivery and Family Planning Information and Services to postpartum women in clinic based settings. A review of evidence from operations research,** 2009.

18. World Health Organization (W.H.O), 1986, **Sample Size determination; Users Manual,**W.H.O/H.S.T./E.S.M./86.1 (Revised).

APPENDIX ONE:

PARTICIPANT'S CONSENT FORM

Principal Investigator: Dr Julius Ondigo

Address: Department of Obstetrics & Gynaecology, University of Nairobi, P. O Box 19676,

Nairobi, Kenya

Introduction

We are health workers currently stationed both at Kenyatta National Hospital and Mbagathi District hospital. We would like to invite you to take part in a research study. In order to be sure that you understand what it means to be involved in this study please read the information in this consent form carefully. If there is anything you do not understand in this consent form please ask us and we shall explain.

Reasons for the Research

This research study will assess the **family planning knowledge and use in the first year after delivery among sexually active breastfeeding mothers** at the Maternal and Child Health clinic of Mbagathi District Hospital. It is hoped that the study will contribute to the quality improvement in family planning service provision at the Maternal and Child Health clinic.

Your part in the research

If you agree take part in the research study you will be asked to fill in a questionnaire. Your name will not be recorded in the questionnaire. The questionnaire will be marked with study numbers for the purposes of analysis only. The information you give will be treated as confidential. Filling of the questionnaire will only take about ten minutes only. We have obtained permission from the Kenyatta National Hospital Research and Ethics committee and the Medical Superintendent of Mbagathi District Hospital to carry out the study.

Possible Benefits of the study

Your participation in the research study will help us to make suggestions to Hospital and the Ministry of Health on how to improve on the quality of family planning services that is provided.

Possible Risks of the study

We do not think you will face any risk if you decide to participate in the study. Your reply to the questionnaire will not be used against you but solely for the study research purposes.

Decision to participate/Not to participate in the Research

You are free to decide if you want to participate in the study or not. Your decision will not be used against you if you decide not to participate in the study.

Confidentiality

The information we collect will be kept confidential. The research reports and publications will not reveal your identity.

Compensation

We will not be able to provide you with any payment or gift for being in the research but we will appreciate your participation.

PARTICIPANT'S AGREEMENT

The above document describing the benefits, risks and procedures for the research study on "**family planning knowledge and use in the first year after delivery among sexually active breastfeeding mothers**" has been explained to me. I have been given an opportunity to have any questions answered about the research to my satisfaction; I have agreed to participate in the study as a volunteer.

Signature of participant _____ Date ____/____/____

I certify that I have explained the nature and purpose of the study to the participant whose study number is _____

Signature of person obtaining consent_____ Date ____/___/____

STUDY QUESTIONNAIRE:

KNOWLEDGE AND PRACTICE OF CONTRACEPTION AMONG SEXUALLY ACTIVE BREASTFEEDING WOMEN IN THE FIRST YEAR AFTER DELIVERY AT THE MCH CLINIC OF MBAGATHI DISTRICT HOSPITAL.

A. IDENTIFICATION:

Patient's study number _____ _____ _____

B. SOCIO DEMOGRAPHIC DATA.

1. What is your age in years? _____ _____

2. What is your marital status?

1. Single
2. Monogamous marriage
3. Polygamous marriage
4. Divorced/ separated
5. Widowed

☐

3. What is your level of education?

1. None
2. Primary
3. Secondary
4. College/ University

☐

4. What is your religion?

1. Catholic
2. Protestant
3. Islam
4. Others (specify) _____

☐

5. What is your parity? _____ + _____

6. Number of living children? _____

7. When was the date of your last delivery? _____

8. When did you resume sexual activity after delivery? _____

C. FAMILY PLANNING AWARENESS AMONG RESPONDENTS:

9. Do you know any method(s) of family planning?

1. Yes
2. No

☐

10. If yes, what was your source of information about family planning?

1. Healthcare facilities
2. Radio and television
3. Newspapers /magazines
4. Posters/billboards
5. Friends/relatives
6. Other sources (specify) _____

☐

12. What methods of family planning do you know? **(Fill in as many as possible)**

1. Oral contraceptive pills
2. Injectables contraceptives
3. I.U.CD's
4. Sub- dermal implants
5. Male condom
6. Natural methods
7. Other methods (specify) _____

13. Since delivery have you been given any family planning option available? **(If No go to question 15)**
    1. Yes
    2. No
14. What family planning options were you offered after delivery?
    1. Oral contraceptive pills
    2. Inject able contraceptives
    3. Intrauterine contraceptive devices
    4. Sub- dermal implants
    5. Permanent sterilization
    6. Other methods (specify) _____
15. Since you started attending the MCH clinic have you been informed about the family? planning options available to you for use?
    1. Yes
    2. No
16. When do you think one should start using family planning methods after delivery?
    1. Immediately after delivery
    2. 2 to 6 weeks
    3. 6 weeks to 6 months
    4. 6 months to 12 months
    5. Other (specify) _____

D. FAMILY PLANNING USE AMONG RESPONDENTS:

17. Were you using any family planning method previously before your last pregnancy? **(If Yes go to question 19)**
    1. Yes
    2. No
18. Did your spouse have any influence on the decision you made not to use any family planning method previously before your last pregnancy? **(Go to question 26)**
    1. Yes
    2. No
19. What method were you using?
    1. Oral contraceptives pills
    2. Inject able contraceptives
    3. Intrauterine contraceptive device
    4. Sub-dermal implants
    5. Other methods (specify) _____
20. Did you have any problems with the method you were using before your last pregnancy?

(If no go to question 22)

1. Yes
2. No

21. What problems did you have with the last method of family planning you used?
    1. Irregular menses
    2. Heavy menses
    3. High blood pressure
    4. Persistent headaches
    5. Conceived while on family planning method
    6. Other (specify) _____

22. Are you using any family planning method currently since your last delivery? **(If yes go to Question 24 and 25)**
    1. Yes
    2. No

23. Did your spouse have any influence on the decision you made not to use any family planning method? **(Go to question 26)**
    1. Yes
    2. No

24. What method of family planning are you using currently?
    1. Oral contraceptive pills
    2. Injectable contraceptives
    3. Intrauterine contraceptive device
    4. Sub-dermal implants
    5. Permanent sterilization (BTL/vasectomy)
    6. Other methods (specify) _____

25. Where did you get the current method of family planning you are currently using?
    1. Government health facility
    2. Private health facility
    3. Local purchase from chemist/shop
    4. Other sources (specify) _____

26. Reason for not using any family planning method since your last delivery?
    1. No reason
    2. Breastfeeding will give enough protection
    3. Am waiting for menses to start
    4. Family planning methods have harmful side effects
    5. Family planning methods are expensive
    6. Other reasons (specify) _____

OBSTETRIC CASE NO 1:

URINARY TRACT INFECTION IN PREGNANCY, NORMAL DELIVERY.

NAME: E. W. AGE: 23 YEARS

IP.NO.1118789 DOA-.24/09/06 **DOD:** 30/09/06

23 years old primigravida, LMP 25/01/05 EDD 30/10/06 GBD 35 weeks. Was admitted with vomiting, fever and low abdominal pains for 2 days.

HISTORY OF PRESENTING ILLNESS.

E. W. had been experiencing low abdominal pains which were intermittent, dull in nature, arising from the suprapubic region and radiating to right iliac fossa. / right lumbar region. There was no history of dysuria. She also gave history of having vomited two times on the day of admission .There was positive history of fever associated with rigors and chills, however there was no history of recent travel outside Nairobi.

OBS/GYN HISTORY

She had attained her menarche at 17 years. Her menstrual cycle used to last 28 days with a flow of 4 days and was regular

ANTENATAL HISTORY

She had attended clinic three times. Profile — only HIV test had been done, which was negative.

FAMILY SOCIAL HISTORY.

She is married, a house wife, and husband is a business man.

EXAMINATION

Patient was in obvious pain, not pale, jaundiced, cyanosed or oedematous. Vital signs: BP 118/73 mmHg, PR 93/ minute, Temperature 37.8°C

ABDOMINAL EXAM

She had a grid iron incision scar on right iliac fossa, fundal height 34/40, longitudinal lie,

Cephalic presentation, head was 5/5 up, foetal heat was rate was 140/minute, regular. There was marked right iliac fossa and renal angle tenderness.

Vaginal examination: Normal external genitalia, cervix was posterior, 2 cm long, closed.

An impression of Acute Pyelonephritis was made.

Management

Patient was admitted and given IM Pethidine 100mg stat.

Urine was taken for microscopy, culture and sensitivity, blood was taken for TBC, VDRL, blood group, U/E/Cr. She was then started on intravenous fluids 500mls normal saline alternating with 5%dextrose for rehydration within the first 24 hours. Patient was also empirically started on antibiotics, intravenous Augmentin 1.2gm TDS 8 hourly and an antispasmodic Buscopan 40mg 8 hourly intravenously.

The lab results were as follows:-

TBC: HB 13 gm/dl, WBC 10.7 x $I0^{L}$ Neut 78.9% L19.3%, Platelets 448 x 1094- URINE MCS - PH 6.0 SG 1.015 Protein Nil Ketone 2+ Leucocytes + Puscells 0-5/ HPF Culture — No bacterial growth obtained.

VDRL — Negative HIV-Negative, Blood group 0 Rhesus positive-

Progress

The initial treatment was continued and her symptoms and general condition improved markedly. Fever subsided, lumbar pains lessened and vomiting stopped. Her temperature normalised within 24hrs and renal angle tenderness was less. On the fifth day post admission, all the symptoms had resolved and she was discharged home on oral Augmentin 625mg 12 hourly for 5 days. She was to continue with her routine ante natal follow up.

She was readmitted to Labour ward two weeks later in labour. On examination she was having 2 contractions every 10 minutes each lasting 30 seconds, foetal heart was regular at 140/min and the presenting part was 3/5 up. On vaginal examination the cervix was 4cm dilated, and the membranes were bulging but the cord was not presenting. The lower segment was swept and on artificial rupture of membranes, the liquor was clear. She progressed well and after an uneventful labour, she had a spontaneous vertex delivery to a live female infant weighing 2850g with an Apgar score of 9 and 10 in one and ten minutes respectively. She was allowed to room in.

She was discharged home on the second post natal day to be reviewed in the post natal clinic after six weeks.

Postnatal follow up

By the time of writing this case 6 weeks had not yet elapsed for the patient to be reviewed in the post natal clinic.

DISCUSSION

E. W. was a 23 year old primigravida who was admitted with acute pyelonephritis at a gestation by dates of 35 weeks and was successfully treated with parenteral followed by oral antibiotics. She was discharged home and was readmitted in labour three weeks later in labour and delivered SVD to a live female infant who weighed 2850g who scored 9/1, and 10/5.

Asymptomatic bacteriuria, acute cystitis, and acute pyelonephritis are common renal disorders in pregnancy. Urinary tract infections (UTIs) account for approximately 10 percent of office visits by women, and 15 percent of women will have a UTI at some time during their life. In pregnant women, the incidence of UTI can be as high as 8 percent [1, 3]. The incidence of acute pyelonephritis in pregnancy is I – 2%.

Pregnant women are at increased risk for UTIs beginning at week 6 and peaking during weeks 22 to 24. Approximately 90 percent of pregnant women develop ureteral dilatation, which will remain until delivery (hydronephrosis of pregnancy). Increased bladder volume and decreased bladder tone, along with decreased ureteral tone, contribute to

Increased urinary stasis and ureterovesical reflux [3] Up to 70 percent of pregnant women develop glycosuria, which encourages bacterial growth in the urine. Increases in urinary progestins and estrogens may lead to a decreased ability of the lower urinary tract to resist invading bacteria. This decreased ability may be caused by decreased ureteral tone or possibly by allowing some strains of bacteria to selectively grow [1, 3] factors may all contribute to the

development of UTIs during pregnancy. Acute pyelonephritis is more common after mid pregnancy. It is unilateral and right sided in more than half of cases, and bilateral in a fourth [13].

The organisms that cause UTIs during pregnancy are the same as those found in nonpregnant patients. Escherichia coli accounts for 80 to 90 percent of infections. Other gram-negative rods such as Proteus mirabilis and Klebsiella pneumoniae are also common. Gram-positive organisms such as group B streptococcus and Staphylococcus saprophyticus are less common causes of UTI. Group B streptococcus has important

implications in the management of pregnancy and will be discussed further. Less common organisms that may cause UTI include enterococci, Gardnerella vaginalis and Ureaplasma ureolyticum [3], [4], [5]. Between 75 -90% of renal infections are caused by bacteria that have P- fimbriae adhesions [13] Th ere was no organism which was isolated from the urine culture of this patient; however the patient had been on antibiotics for 12 hours by the time the specimen for culture was being collected..

Asymptomatic bacteriuria is defined as the presence of actively multiplying bacteria in the urinary tract excluding the distal urethra in a patient without any obvious symptoms. Significant bacteriuria has been historically defined as isolation of organisms with a colony count of more than 10^5 organisms per ml, of urine in two consecutive clean catch specimens [7]. Asymptomatic bacteriuria is twice as common in pregnant women with sickle cell trait and three times as common in pregnant women with diabetes as in normal pregnant women.

Asymptomatic bacteriuria is common, with a prevalence of 10 percent during pregnancy [8]. Thus, routine screening for bacteriuria is advocated. Untreated asymptomatic bacteriuria leads to development of symptomatic cystitis in approximately 30 percent of patients and acute pyelonephritis in about 25-30 %(7). Asymptomatic bacteriuria is associated with an

increased risk of intrauterine growth retardation and low-birth-weight infants [9].

The American College of Obstetrics and Gynecology recommends that a urine culture be obtained at the first prenatal visit. A repeat urine culture should be obtained during the third trimester, because the urine of treated patients may not remain sterile for the entire pregnancy [10]. The recommendation of the U.S. Preventative Services Task Force is to obtain a urine culture between 12 and 16 weeks of gestation [11]. By screening for and aggressively treating pregnant women with asymptomatic bacteriuria, it is possible to significantly decrease the annual incidence of pyelonephritis during pregnancy [8, 12] Rouse and colleagues [14] performed a cost-benefit analysis of screening for bacteriuria in pregnant women versus inpatient treatment of pyelonephritis and found a substantial decrease in overall cost with screening. The gold standard for detection of bacteriuria is urine culture, but this test is costly and takes 24 to 48 hours to obtain results. The accuracy of faster screening methods (e.g., leukocyte esterase dipstick, nitrite dipstick, urinalysis and urine Gram staining) is variable [15]. The increased number of false negatives and the relatively poor predictive value of a positive test make the faster methods less useful; therefore, a urine culture should be routinely obtained in pregnant women to screen for bacteriuria at the first prenatal visit and during the third trimester [10].

Acute cystitis is distinguished from asymptomatic bacteriuria by the presence of symptoms such as dysuria, urgency frequency and supra pubic discomfort. An acute febrile illness with nausea, vomiting and chills is usually absent " [9]. The characteristic cloudy malodorous urine should be cultured for confirmation of diagnosis.

Acute pyelonephritis during pregnancy is a serious systemic illness that can progress to maternal sepsis, preterm labor and premature delivery. The diagnosis is made when systemic symptoms or signs such as fever, chills, nausea, vomiting and flank pain accompany the presence of bacteriuria. Symptoms of lower tract infection (i.e., frequency and dysuria) may or

may not be present. Pyelonephritis occurs in 2 percent of pregnant women; up to 23 percent of these women have a recurrence during the same pregnancy [20]. Our patient had a single episode of acute phelonephritis. Early, aggressive treatment is important in preventing complications from pyelonephritis.

Group B streptococcal (GBS) vaginal colonization is known to be a cause of neonatal sepsis and is associated with preterm rupture of membranes, and preterm labour and delivery. GBS is found to be the causative organism in UTIs in approximately 5 percent of patients [22, 23]. Evidence that GBS bacteriuria increases patient risk of preterm rupture of membranes and premature delivery is mixed . It is unclear if GBS bacteriuria is equivalent to GBS vaginal colonization, but pregnant women with GBS bacteriuria should be treated as GBS carriers and should receive a prophylactic antibiotic during labor [26]

The initial antibiotic selection should be empiric. Based on the fact that the most common offending pathogen is E. coli, sulfonamides, nitrofurantoin, Ampicillin or cephalosporins could be selected. The antibiotic should also be safe for the mother and foetus. Historically, ampicillin has been the drug of choice, but in recent years E. coli has become increasingly resistant to ampicillin [16]. Ampicillin resistance is found in 20 to 30 percent of E. coli cultured from urine in the outpatient setting [17, 18]. Nitrofurantoin is a good choice because of its high urinary concentration. Alternatively, cephalosporins are well tolerated and adequately treat the important organisms. A seven- to 10-day course of antibiotic treatment is usually sufficient to eradicate the infecting organism(s). Some authorities have advocated shorter courses of treatment-even single-day therapy. Conflicting evidence remains as to whether pregnant patients should be treated with shorter courses of antibiotics. After patients have completed the treatment regimen, a repeat culture should be obtained to document successful eradication of bacteriuria, [0].

Women with asymptomatic bacteriuria should be treated with antibiotics such as Nitrofurantoin 100mg daily, sulphonamide, cephalospiorin, Ampicillin, or amoxicillin for 3 days. Single dose antimicrobial therapy for bacteriuria has also been used with success (Andriole and Patterson 1991).

Any woman with acute pyelonephritis in pregnancy should be hospitalized for therapy. Antibiotics should be given parenterally and dehydration corrected. Antipyretic therapy is given where indicated and vital signs and urinary output are monitored closely. The choice of drug is empirical and ampicillin plus gentamycin, cefazolin, or ceftriaxone have been shown to be 95% effective in randomized trials (Wings and colleagues, 1998, 2000). Parenteral treatment of pyelonephritis should be continued until the patient becomes afebrile. Most patients respond to hydration and prompt antibiotic treatment within 24 to 48 hours. The most common reason for initial treatment failure is resistance of the infecting organism to the antibiotic. If fever continues or other signs of systemic illness remain after appropriate antibiotic therapy, the possibility of a structural or anatomic abnormality should be investigated. Persistent infection may be caused by urolithiasis, which occurs in one of 1,500 pregnancies [21], or less commonly, congenital renal

abnormalities or a perinephric abscess. Diagnostic tests may include renal ultrasonography or an abbreviated intravenous pyelogram. Even the low-dose radiation involved in an intravenous pyelogram, however, may be dangerous to the fetus and should be avoided if possible. This patient responded to the antibiotic therapy.

UTIs recur in approximately 4 to 5% of pregnancies, and the risk of developing pyelonephritis is the same as the risk with primary UTIs. A single, post coital dose or daily suppression with cephalexin or nitrofurantoin in patients with recurrent UTIs is effective preventive therapy [27]. A postpartum urologic evaluation may be necessary in patients with recurrent infections because they are more likely to have structural abnormalities of the

renal system [20, 21, 28]

The maternal and neonatal complications of a UTI during pregnancy can be devastating. Thirty percent of patients with untreated asymptomatic bacteriuria develop symptomatic cystitis and up to 50 percent develop pyelonephritis [7]. Asymptomatic bacteriuria is also associated with intrauterine growth retardation and low-birth-weight infants [9]. Schieve and associates [29] conducted a study involving 25,746 pregnant women and found that the presence of UTI was associated with premature labor (labor onset before 37 weeks of gestation), hypertensive disorders of pregnancy (such as pregnancy-induced hypertension and preeclampsia), anemia (hematocrit level less than 30 percent) and amnionitis. While this does not prove a cause and effect relationship, randomized trials have demonstrated that antibiotic treatment decreases the incidence of preterm birth and low-birth-weight infants [13] Pyelonephritis can be a life-threatening illness, with increased risk of perinatal and neonatal morbidity.

REFERENCES

1. Lucas M.J, Cunningham F.G. Urinary infection in pregnancy. Clin Obstet Gynecol 1993; 36:855-68.

2. Mikhail MS, Anyaegbunam A. Lower urinary tract dysfunction in pregnancy: a review. Obstet Gynecol Sur[y] 1995; 50:675-83.3.

3. Patterson TF, Andriole V. Bacteriuria in pregnancy. Infect Dis Clin North Am 1987; 1:807-22.

4. Barr JG, Ritchie JW, Henry 0, el Sheikh M, el Deeb K. Microaerophilic/anaerobic bacteria as a cause of urinary tract infection in pregnancy. Br J Obstet Gynaecol 1985;92:506-10.

S. McDowall D.R, Buchanan J.D, Fairley K.F. Anaerobic and other fastidious microorganisms in asymptomatic bacteriuria in pregnant women. J Infect Dis 1981; 144:114-22.

6. Kass E.H. Pregnancy, pyelonephritis and prematurity. Clin Obstet Gynecol 1970; 13:239-54.

7. Danoj K.B, Dorothee P. Renal and urinary tract disorders in pregnancy: Current obstetrics

and Gynaecologic Diagnosis and Treatment. 9 h Ed. Mc Graw Hill Comp. 2003, p460-464.
8.Gratacos E, Torres PJ, Vila J, Alonso PL, Cararach V. Screening and treatment of asymptomatic bacteriuria in pregnancy prevent pyelonephritis. J Infect Dis 1994; 169:1390-2.

9. Harris R, Thomas V, Shelokov A.
Asymptomatic bacteriuria in pregnancy: Antibody-coated bacteria, renal function, and intrauterine growth retardation.
Am J Obstet Gynecol 1976; 126:20-5.

10. Antimicrobial therapy for obstetric patients. ACOG educational bulletin no. 245. Washington, D.C.: American College of Obstetricians and Gynecologists, March 1998; 245:8-10.

11. U.S. Preventive Services Task Force. Guide to clinical preventive services: report of the U.S. Preventive Services Task Force. 2d ed. Baltimore: Williams & Wilkins, 1996.

12. Harris R.E. The significance of eradication of bacteriuria during pregnancy. Obstet Gynecol 1979; 53:71-3.

13. Cunnigham F.F; Gant N.F; Leveno K.J; Gilstrap L ; Hauth C.J; Wenstrom K.D. Renal and urinary tract disorders in pregnancy. 21St ed. Mc Graw Hill Comp. 2001: p 1254-1258.

14. Rouse D.J, Andrews W.W, Goldenberg R.L, Owen J. Screening and treatment of asymptomatic bacteriuria of pregnancy to prevent pyelonephritis: a cost-effectiveness and cost-benefit analysis. Obstet Gynecol 1995; 86:119-23.

15.Bachman J.W, Heise R.H, Naessens J.M, Timmerman M.G. A study of various tests to detect asymptomatic urinary tract infections in an obstetric population. JAMA 1993; 270:1971-4.

15. Duff P. Antibiotic selection for infections in obstetric patients. Semin Perinatol 1993; 17:367-78.

16. Peddie B.A, Bailey R.R, Wells J.E. Resistance of urinary tract isolates of Escherichia coli to cotrimoxazole, sulphonamide, trimethoprim and ampicillin: an 11 - year survey. N Z Med J 1987; 100:341- 2.

17. Sanders C., Sanders W.E Jr. Beta-lactam resistance in gram-negative bacteria: global trends and clinical impact. Clin Infect Dis 1992; 15:824-39.

18. Harris R.E, Gilstrap L.C. Cystitis during pregnancy: a distinct clinical entity. Obstet Gynecol 1981; 57:578-80.

19. Gilstrap L.0 , Cunningham F.G, Whalley P.J. Acute pyelonephritis in pregnancy: an anterospective study. Obstet Gynecol 1981; 57: 409-13.

20. Loughlin K.R. Management of urologic problems during pregnancy. Urology 1994; 44:159-69.

21. Mead P.J, Harris R.E. The incidence of group B beta hemolytic streptococcus in antepartum urinary tract infections. Obstet Gynecol 1978; 51:412-4.

22. Pass MA, Gray BM, Dillon HC Jr.
Puerperal and perinatal infections with group B streptococci. Am J
Obstet Gynecol 1982; 143:147-52.

23. Moller M, Thomsen A.C, Borch K, Dinesen K, Zdravkovic M. Rupture of fetal membranes and premature delivery associated with group B streptococci in urine of pregnant women. Lancet 1984; 2(8394):69-70.

24. McKenzie H, Donnet M.L, Howie P.W, Patel N.B, Benvie D.T. Risk of preterm delivery in pregnant women with group B streptococcal urinaryinfections or urinary antibodies to group B streptococcal and E. coli antigens. Br J Obstet Gynaecol 1994; 101:107-13.

25. Prevention of perinatal group B streptococccal disease: a public health perspective. Centers for Disease Control and Prevention. MMWR Morb Mortal Wkly Rep 1996; 45(RR-7):1-24 [published erratum in MMWR Morb Mortal Wkly Rep 1996; 45(31): 679].

26. Pfau A, Sacks T.G. Effective prophylaxis for recurrent urinary tract infections during pregnancy. Clin Infect Dis 1992; 14:810-4.

27. Diokno A.C, Compton A, Seski J, Vinson R. Urologic evaluation of urinary tract infection in pregnancy. J Reprod Med 1986; 31:23-6.

28. Schieve LA, Handler A, Hershow R, Persky V, Davis F. Urinary tract infection during pregnancy: its association with maternal morbidity and perinatal outcome. Am J Public Health 1994; 84:405-10.

29. Gruneberg R., Leigh D.A, Brumfitt W. Relationship of bacteriuria in pregnancy to acute pyelonephritis, prematurity, and fetal mortality. Lancet 1969; 2(7610): 1-3.

OBSTERIC CASE NO. 2

POST DATISM – SUCCESFUL INDUCTION OF LABOUR WITH LIVE BIRTH

Name: N. A. **Age:** 25years **DOA:** 1/5/06 **DOD:** 5/4/06

Para 1 + 0 G2 LMP 7/07/05 EDD 14/04/06 GBD 42/40 plus, quickening was in early October 2005, admitted with abdominal pains for two days.

HISTORY OF PRESENTING ILLNESS N. A. was admitted with two days history of low abdominal pain which was not intermittent, not radiating to the back and had been having much pain for the last two weeks. There was no per vaginal bleeding, discharge or drainage of liquor. She did not also have any urinary symptoms. She reported experiencing normal foetal movements.

OBS/GYN HISTORY She attained her menarche at 16 years, while her menstrual cycle used to last 28 days, and was regular with 3 days of menstrual flow with no associated history of dysmenorrhoea. Had used DMPA from 2004 to early 2005 when she stopped due to irregular bleeding. She was Para 1 + 0 G 2 at 42 weeks her LMP was on 15/7/05 and EDD - 22/4/06. Her last delivery was in 1999, term SVD at home 3 Kg and was alive and well.

ANTENATAL HISTORY For this pregnancy, she started ante natal follow up at St. Mary's hospital at 20/40 gestation.

Ante natal profile was done: Blood group A Rhesus positive, VDRL/HW were Negative Hb - 10.6g/dl. She was treated for malaria once in December 2005.

FAMILY SOCIAL HISTORY She is married and works as a sales lady. Her husband is unemployed and they reside at Anyany. She does not smoke or take alcohol. There is no history of chronic illness in the family.

On examination, she was in fair general condition, not pale, jaundiced, cyanosed or oedematous.

Vital signs: BP - 120/80 mmHg, PR – 80/min, RR – 20/min, T- 36.9 ° C.

P/A: Abdomen was uniformly distended, fundal height was term, longitudinal lie, cephalic, and head was 5/5 up, foetal heart was I 38/min, regular. There was no contraction palpated V/E: There was a normal external genitalia, cervix was 2cm long, posterior, moderate consistency. There was no cord felt and there was no discharge on the examining finger.

An impression of Postdatism with false labour and poor Bishops score was made.

Management The patient was admitted, given intramuscular Tramol 100mg stat and given a prescription to buy Prostin E2 pessaries for cervical ripening On 3/5/06, the first PGE_2 peccary was inserted to the posterior fornix at 10.00a.m. then she was for review 6-8 hours later. At 6.00p.m. she reported some lower abdominal pains for which on examination, the descent was 4/5 up, foetal heart rate was 140 beats/minute, on V/E: the cervix was central, 4cm dilated, and fully effaced. ARM done, clear liquor was obtained and labour augmented with 5U of Oxytocin in 500mls of 5% dextrose and the partogram started. She was also given intramuscular Buscopan 40 mg stat and Tramal 100mg stat. At 12.30am on 4/05/06 she developed urge to bear down, and went on to deliver SVD to a life female infant who had an Apgar score of 9/1, 10/5 and 10/10 with a birth weight of 3.4kg. Placenta was delivered via controlled cord traction. The estimated blood loss was 200mls.

On the first post natal day, she reported heavy lochia loss, however the uterus was well contracted. She was discharged homel on the second post natal day for review at the post natal

clinic after six weeks. MCH/FP

DISCUSSION

The patient presented was 25 year old Para 1+0 admitted with false labour in a post dated pregnancy. She was successfully induced with prostaglandin pessaries and delivered by spontaneous vaginal delivery to a life female infant who scored well.

A post term pregnancy is one that persists for 42 weeks or more from the onset of a menstrual period that was followed by ovulation two weeks later. This is not always easy to determine due to varied duration from the onset of menstrual period to ovulation. Therefore, those who ovulate more than two weeks after onset of menstrual flow may be labeled prolonged pregnancy when they are actually not [1]. Thus, most pregnancies reliably 42 completed weeks beyond the last menses probably are not biologically prolonged, and a few not yet 42 weeks might be post term. These variations in menstrual cycle likely explain at least partially, why approximately 10% of pregnancies reach 42 completed weeks, yet a relatively small proportion of fetuses have evidence of post maturity.

Post term pregnancy varies greatly depending on the criteria used for diagnosis and reported frequency range from 4% to 14% with an average of 10% [2]. With the introduction of ultrasound in early pregnancy, this incidence has been reduced to 6.5% [3]. Elfenesh 1998 in her study at KNH and Pumwani Maternity Hospital found the prevalence of post term pregnancy to be 4.9% [4].

The cause of most post term pregnancies remains unknown but conditions associated with it include anencephaly, fetal adrenal hypoplasia, absence of foetal pituitary gland, placenta sulphatase deficiency and extra uterine pregnancy. All these conditions are associated with low oestrogen levels as opposed to the high levels that characterize normal pregnancy [1,3]. The low oestrogen levels results in inadequate production of membrane phospholipids from which arachidonic acid is cleaved for the synthesis of prostaglandin F2 and E2; these are responsible for the rhythmic uterine contractions and effacement of the cervix that occur in

normal labour

Other etiological factors include improved living standards and hereditary factors. Prolonged pregnancy tends to recur in successive pregnancies in the same woman. The condition tends to run in families 5.

The diagnosis of post term pregnancy is difficult to make especially in cases where the patient has not been seen early in pregnancy. The history of the woman's last menstrual period is the best clinical predictor of the date of confinement [6] Information on menstrual patterns, use of ovulation inducing agents and recent discontinuation of hormonal contraceptives may also be beneficial. [3] Our patient was sure of her LMP, had never used any method of contraceptive, had had regular menses and her quickening dates all pointed to post- dates.

Ultrasound sonography in the first half of pregnancy may give a reliable estimation of the age of the pregnancy. In this patient presented, ultrasound had not been done in early pregnancy.
Management of prolonged pregnancy depends on the certainty of dates. For those not in labour, vaginal delivery is aimed at in all cases unless otherwise contraindicated. Post term pregnancy is one of the commonest indications for induction of labour in many centers [3]. In our setup, induction of labour is practiced at 42 weeks. Surfactant test is done to assess fetal lung maturity. Labour induction is done by use of prostaglandin pessaries followed by amniotomy and oxytocin infusion. For patients with unfavourable cervix, prostaglandin are necessary to ripen the cervix. In women with Bishop Score of 7 points and above amniotomy is done followed by syntocinon infusion.

Other mechanical techniques of ripening the cervix include cervical dilatation with a ballooned Foleys catheter with or without extra-amniotic saline infusion and laminaria tents inserted to the cervix these are outdated in many centers today.

This patient had cervical ripening done with prostaglandin pessaries and had subsequent amniotomy done. However she had failed induction hence emergency caesarean section was done.

The rate of maternal and fetal complications increase with gestational age. The maternal risks usually relate to large fetal size. Fetal macrosomia leads to dystocia or cephalopelvic

disproportion. The rate of caesarean section delivery doubles when the gestation passes 42 weeks compared to gestation at 38 – 40 weeks [7, 8]. Other indications for caesarean section will result from failed induction and fetal distress. The patient presented had failed induction with subsequent caesarian section. The infant did not have features of post-maturity.

Fetal complications are related to placental insufficiency and oligohydramnios. Placental insufficiency is due to placental ageing. Amniotic fluid volume decreases from 37 weeks gestation on-wards. In prolonged pregnancy there is 33% decline in amniotic fluid each week [3, 9]. Oligohydramnios may lead to cord compression, which may lead to fetal distress with meconium production and aspiration.

Neonatal complications include birth trauma from macrosomia and meconium aspiration syndrome. The incidence of macrosomia is 3-7 times more frequent in prolonged pregnancy than in term deliveries [10]. These babies are at greater risk of perinatal morbidity especially neurological sequele.

Careful intrapartum management is important to guarantee continued fetal well being. On delivery of the head, effective suctioning of the pharynx before the delivery of the thorax is required if meconium is identified.

Studies are being done to find ways of reducing the risk of prolonged pregnancies. Fetal fibronectin found in the choriodecidual interface is usually found in cervical vaginal secretions 1 – 2 weeks before delivery. The combination of a negative fibronectin test and unfavourable cervix at 39 weeks gestation may be additive in increasing risk of patient remaining undelivered at 41 weeks. One study found that serial membrane sweeping in these patients resulted in earlier delivery compared to controls ".

REFERENCES

1. F. Gary Cunningham., Norman F. Gant et al.Common complications of pregnancy In: Williams Obstetrics. 20 edition, 2001:7 (28) 730-741

2. Bakketeig L.S., Bergsje P. Post term pregnancy. Magnitude of the problem In: Chatmers I, Enkin M. (Eds): Effective care in pregnancy and childbirth. Oxford University Press. 1991: 765-771.

3. Cardozo L. Prolonged pregnancy in: Turnbill's Obstetrics. 2nd edition, 1996: 633643

4. Elfenesh D. Post date pregnancy and fetal outcome. M.Med Thesis, University of Nairobi, 1998

5. Tuck S.M., Cardozo L.D., Studd J.W.W. Obstetric characteristics in different racial groups. Brit.J. obst/gynaecol, 1998: 90:892

6. Anderson H.F., Johnson T.R.B., Barclay A.C. Gestational Age assessment In: Analysis of individual clinical observations. Obst/gynaecol , 1981: 139-173

7. Martin L., Pernoll. Late pregnancy complications, In: Current Obstetrics and Gynaecology, Diagnosis and Treatment. 9tfi edition, 2001: 14; 326-339

8. Eden R.D., Seifer L.S., Winegar A. Perinatal characteristics of uncomplicated post date pregnancy. Obs/Gynaecol, 1986: 69: 296

9. Tongsong T., Srisomboon J. Amniotic fluid volume as a predictor of fetal distress in post term pregnancy. Int . J. Obs/Gynaecol, 1993: 40: 213

10. Spellacy W.N.G., Miller M.S., Winegar A. Macrosomia: Maternal characteristics and infant complications. Obs /Gynaecol , 1995 : 66:158

11. Gary Cunningham, Norman F. Gant et al. Abnormal labour in: Williams's obstetrics. 21' edition 2001: 5 (20); 469-481

OBSTETRIC CASE NO 3

PRETERM PREMATURE RUPTURE OF MEMBRANES AT 30 WEEKS GESTATION — PRETERM LABOUR WITH LIVE BIRTH

NAME: J.N. AGE: 28 YEARS

IPNO: 0971362 DOA: 09.07.04 **DOD:** 13.07.04

28 years old Primigravida, LMP 10/12/03 EDD 17/09/04 GBD 30/40 admitted with C/O Drainage of liquor x 6 hours

HISTORY OF PRESENTING ILLNESS

J.N was well until 6 hours prior to admission when she had a sudden gush of fluid from the vagina while standing doing her household chores. The fluid was colourless and trickled down the legs to the floor. There was no associated vaginal discharge or bleeding. There were no associated abdominal pains at the time of admission. There was no history of trauma, dysuria or frequency of micturition.

PAST MEDICAL HISTORY

Not significant

OBS/GYN HISTORY

She attained menarche at 15 years; she had a regular menstrual cycle of 30 days with duration of flow of 4 days. She had not used any contraceptive method. She was a primigravida. Her last menstrual period was on 10.12.03 and her expected date of delivery on 17.09.04; she was at 30 weeks gestation by dates and had not started antenatal care.

FAMILY SOCIAL HISTORY

She was a single businesswoman. She did not smoke cigarettes or drink alcohol. Her mother was diabetic.

On examination, she was in good general condition, not pale, afebrile, not jaundiced, and without oedema or lymphadenopathy.

Vital signs: BP 120/70 mmHg, pulse rate 84/min and temperature of 36.8°C. The CNS, CVS and RS essentially normal.PA: The abdomen was distended and moved with

respiration. There were no areas of tenderness. The liver and spleen were not palpable. The uterus corresponded to 30 weeks gestation. The foetus was in longitudinal lie and cephalic presentation and the descent was 5/5. The foetal heart tones were heard and regular at 144 beats per minute.

Speculum exam: She had normal external genitalia. The vaginal walls and the cervix were healthy. There was a pool of clear fluid in the posterior fornix and active drainage of liquor was noted from the cervical os. The cervical os was closed and the umbilical cord was not seen.

DIAGNOSIS Preterm premature rupture of membranes at 30 weeks gestation

Investigations:

Obstetric ultrasound scan showed a single intrauterine pregnancy at 28 weeks gestation with reduced amount of liquor.

Haemogram; Hb 12.3 gm/dl WBC 8.5 x 10^9/1 Platelets 295 x 10 ^{9} /L, Blood group A positive, VDRL negative, HIV test negative. Urine microscopy culture and sensitivity was normal with no growth obtained. Endocervical swab culture grew no organisms.

MANAGEMENT

She was admitted to the admitting antenatal ward for conservative management. She was put on bed rest and oral erythromycin 500mg 8 hourly and metronidazole 400 mg 8 hourly. Intramuscular dexamethasone 12mg twice daily fora total of two doses was also given to enhance foetal lung maturity. An endocervical swab was taken for culture and sensitivity and specimens taken for haemogram and urine microscopy culture and sensitivity. Vital sign were taken every 4 hours. Daily patient examinations were done including abdominal palpation for tenderness and examining the liquor for smell and the colour. No signs of sepsis were noted during the conservative management.

Two days after admision, she went into spontaneous labour and had a spontaneous vertex delivery to a live female infant with an Apgar score of 5 and 8 in 1 and 5 minutes respectively and weighing 1400g. The baby was admitted to the newborn unit due to prematurity. The mother continued with oral erythromycin 500mg 8 hourly and metronidazole 400 mg 8 hourly for five days. She remained stable and was discharged to the mothers' hostel to await discharge of her baby from the newborn unit.

DISCUSSION

Premature rupture of membranes (PROM) is defined as the rupture of membranes with leakage of amniotic fluids more than 8 hours before the onset of labour regardless of the gestation [1, 2]. Preterm premature rupture of membranes (Preterm PROM) refers to the rupture before 37 weeks and six days.

The cause of premature rupture of membranes is almost certainly multifunctional. At the molecular level, the premature rupture of the membranes appears to result from diminished collagen synthesis, altered collagen structure and accelerated collagen degradation, possibly in association with concurrent cellular changes within the foetal membranes[3]. It now seems that rupture of the membranes requires the interaction of physical stresses coupled with lack of resistance of the foetal membranes [5].

Various clinical factors have been associated with premature rupture of membranes:-

1. Infection: there is increasing evidence of the major role of infection in PROM [5]. Women with urinary tract infection, bacterial vaginosis and other genita tract infections are more prone to develop PROM. Infection is thought to act in two main ways:-

   a) Infection produces a decrease in the resistance of the connective tissue matrix directly by bacterial production of proteases and phospholipases or indirectly by triggering maternal and foetal macrophage mediated enzyme induction.
   b) Infection is thought to increase prostaglandin production and hence uterine activity.

   The organisms which have been implicated include E.Coli, Group B Streptococci, Gardnerella Vaginalis, Chlamydia, Staphylococcus aureus and vaginal anaerobes [1].

2. Connective tissue disorder and nutritional deficiencies-Connective tissue disorders are associated with weakened foetal membranes and nutritional deficiencies that predispose women to abnormal collagen structure (such as deficiencies of copper, vitamin C and Zinc) have been implicated in the aetiology of PROM. Cigarette smoking especially due to cadmium in tobacco, which sequestrates

copper, coupled with deficiencies of copper and Vitamin C is also thought to play a role.

3. Genetic predisposition: Patient with a positive familial history and a history of previous preterm PROM are more susceptible to premature rupture of membranes.
4. Lack of support: Membrane stretching and uterine over distension due to multiple gestation and polyhydramnios induces mechanical stretching of foetal membranes resulting in the release of several amniotic factors such as PGE_2 and interleukin-8 which increase the risk of PROM. Cervical incompetence reduces the support for the foetal membranes and predisposes to PROM.

There is also an accompanying reduction in fundal height, prominence of the foetal parts and diminished liquor amount. A sterile speculum examination is mandatory for definitive diagnosis. Visualization of leakage of liquor from the cervical os is diagnostic. The amount of liquor, colour, smell and viscosity as well as the state of the cervix must be noted. Liqour is alkaline and turns nitrazine or litmus paper blue, it ferns if air dried on a glass slide and the foetal cells in it stain with Sudan Red or Nile blue dye . Ultrasonography can also be used to determine the amount of liquor.

After premature rupture of membranes at term, 70% of women will labour within 24 hours and 95% of within 72 hours [3]. After preterm PROM, the latency period from membrane rupture to delivery increases inversely with the advancing gestational age e.g. the main latency period from 20-26 weeks gestation is 12 days, at 32-34 weeks its only 4 days (7.)

Management of patients with PROM is either active or conservative depending on the gestational age of the pregnancy. In cases of PROM at term or near term (36 or more weeks of gestation) the best solution is delivery by immediate induction. Those at 34-36 weeks should also be induced but after waiting for 24 hours to 48 hours to allow better lung maturity after administration of corticosteroids. In cases of rupture of membranes under 34 weeks of gestation the objective is to prolong the gestation if there are no signs of foetal distress or infection.

The purpose of conservative management is to allow the foetus to reach to a stage of maturity

at which it can survive outside the uterus, without endangering the mother. Each week gained significantly improves the chances of survival. In all patients managed conservatively it is necessary to screen continuously for the appearance of infection or foetal distress and to intervene at the earliest signs of those complications

Much controversy has existed on the use of prophylactic antibiotics, corticosteroids and tocolysis. Recent evidence has shown prophylactic antibiotics to be effective in prolonging the period between rupture of membranes and delivery and also to significantly reduce the risk of maternal chorio-amnionitis, neonatal respiratory distress syndrome and neonatal sepsis 8 9, 10

REFERENCES

1. Pernoll M.L. Later pregnancy Complications: Premature rupture of membranes. Current Obstetrics and Gynaecology diagnosis and Treatment. 7th Edition 1991 14:332-334.
2. Oxorne H. Amniotic fluid and Preterm Labour. Oxhorne-Foote Human Labour and Birth. 5 edition, Appleton and Lange 1998 43
3. Strauss P.S. Premature Rupture of Foetal membranes. N.Eng J. Med 338:10: 663-67
4. Otieno J.A. Spontaneous Rupture of membranes: A Retrospective analysis of 80 Cases. MMed Thesis, University of Nairobi 1979.
5. Cararas V. Premature rupture of membranes: Maternal and Perinatal Complications Pace review No.95/08
6. Katherine D.W, Carl P.W. Clinical Obstetrics and Gynaecology. W.P Saunders and Company 1992; 19(2); 241
7. Dale P.0, Tambo T. Bendrold et al. Duration of the Latency Period in preterm premature rupture of membranes. Euro. J. Obstetrics Gynaecology Rep. Boil 1998; 30:257-62
8. Merecer B.M, Mlodovnic M, Thurman GR et al, Antibiotic therapy for reduction of Infant Mortality after preterm PROM: A Randomised Controlled Trial. JAMA 1998, 278:282-95

9. Ohlsson A. Treatment of Preterm PROM: A Meta analysis. A.M J. Obstetric Gynaecology 1989:160; 890-906

10. Lewis D F, Brody K. Preterm PROM; A Randomized trial of steroids after treatment with antibiotics. Obstetrics and Gynaecology 1996, 88-801-5

OBSTETRIC CASE NO 4

PLACENTA PRAEVIA TYPE IV –ELECTIVE C/S - GOOD OUTCOME

Name: E. K. **Age:** 29years

IP.NO: 1102707 DOA: 29/6/06 **DOD:** 12/8/06

29 years old Para 1 + 0 G2 LMP 21/11/05 EDD - 28/8/06 GBD 32/40, had quickening in mid-March so gestation by extrapolation of about 34/40 admitted with

C/O Per vaginal bleeding x 4 hours.

HISTORY OF PRESENTING ILLNESS

The per vaginal bleeding was spontaneous in onset while the patient was on bed at night, had a big clot of blood which came out initially then subsequently has been having slight bleeding. There were no associated low abdominal pains or urinary symptoms. There was also no history of trauma prior to the onset of the bleeding.

OBS/GYNHISTORY She had her menarche at 15 years. Her menstrual cycle lasts 28 days while the flow is usually 4 days and is regular. There was no history of contraceptive use. Ist delivery was in 1995, SVD at term to a live male infant birth weight 3.Okg at PMH, who is still alive and well.

ANTENATAL HISTORY She had attended her ante natal clinic twice at Kawangware Nairobi City Council clinic. Profile done: Hb 11.8gm/dl, VDRL/HIV - not reactive, Blood group not known

EXAMINATION, She was in fair general condition, not pale, jaundiced, cyanosed or oedematous.

Vital signs: BP 113/71mmHg, PR - 86/min, RR 20/min

CNS/CVS/RS: Essentially normal

P/A:Fundal height was 34/40, oblique lie, cephalic presentation. Foetal heart rate was 134/min,

regular.

Speculum exam: There was normal external genitalia, cervix was central, 1 cm long , closed with slight oozing noted from the internal os.

An impression of Ante-partum. Haemorrhage was made. An intravenous line was established, blood was taken for grouping and cross matching plus an urgent PCV and the patient was booked for an urgent obstetric ultrasound scan. She was started on an intravenous drip of normal saline. Meanwhile she was extracted to maintain a vulval pad so as to monitor the extent of her bleeding.

Pelvic U/S scan showed a single intrauterine pregnancy in cephalic presentation at an average gestation of 34 weeks. There was adequate amount of liquor and the foetus appeared grossly normal. There was a low lying placenta covering the whole of the internal os, however there was no retro-placental clot noted.

Impression: Placenta praevia type IV at 34 weeks.

Her PCV was 32 %. Blood group B Rhesus positive.

Management

The mother was explained about the above findings. Her management was going to be conservative: strict bed rest in the hospital until term, haematenic supplements, intramuscular Dexamethasone to assist in lung maturity then undergo elective Caesarean delivery at term or earlier if she experiences profuse per vaginal bleeding. She consented to the above plan of management.

Repeat BP reading 4 hours post admission was 121/72mmHg and the vaginal bleeding had completely resolved, so she was transferred to the antenatal wards for the above management.

Repeat Hb on18/7/06 was 11.6gm/dl

Her stay in the ward remained uneventful. She went for an elective C/S at term on 9/08/06. Intra operatively, found a low lying placenta covering the whole of the internal os, and a live male infant who scored 8/1, 9/5, 10/10, birth weight 2400gms was delivered. Estimated blood loss was 500mls.

Postoperative Care

She was observed continuously in the recovery ward until fully conscious and then hourly

for 6 hours and then 4 hourly thereafter. She was put on intravenous benzyl penicillin G 2 MU 6 hourly, Gentamicin 80 mg 8 hourly, intramuscular Pethidine for analgesia for 24 hours then oral Diclofenac. She did well postoperatively and was discharged home on the fourth postoperative day through the postnatal clinic in six weeks on haematinics and analgesics.

Postnatal Clinic

When she was reviewed after 6 weeks in the postnatal clinic, the wound had healed well and the baby was well and exclusively breastfeeding. She was discharged through the family planning clinic for contraception.

DISCUSSION

The patient presented was a Para 1+0 admitted with placenta praevia at 32 weeks gestation and managed conservatively until 37 weeks of gestation when an elective Caesarean delivery was done at term. She delivered a live male infant weighing 2400g.

Placenta praevia is defined as a condition where the placenta is located partially or wholly in the lower uterine segment. Four degrees of this abnormality have been recognized: [1,2,3]

1. **First degree (low lying):** part of the placenta lies in the lower uterine segment but does not reach the internal os.
2. **Second degree (marginal):** the lower margin of the placenta reaches the internal os, but does not cover it
3. **Third degree (Partial):** the internal os is partially covered by the placenta.
4. **Fourth degree (central or total):** the placenta lies centrally over the internal os.

The significance of the different types lies in the increasing morbidity and mortality to the mother and foetus as the placenta becomes more centrally placed. As the lower segment of the uterus forms in the latter half of pregnancy, the placenta tends to become sheared off. Because the endometrium is less well developed in the lower uterine segment, the placenta is more likely to become attached to the underlying muscle (placenta previa accreta), with consequent problems during the third stage of labour/delivery" [3]

The incidence of placenta previa is 1:200 (0.5% of births) [2, 3, 4] Varying incidences have been

reported in different studies at Kenyatta National Hospital; Kirima reported an incidence of 1:116. Ojwang' an incidence of 0.25%, and Mbithi an incidence of 1 percent. [5, 6]

A number of factors appear to increase the risk of placenta previa. These include advancing maternal age, multiparty, smoking, cocaine use, prior placenta previa, one of more previous caesarean births and prior suction curettage for spontaneous or induced abortion [3,4,8]

The strong association between placenta previa and parity, previous caesarean delivery and suction curettage suggest that endometrial damage is an aetiological factor. Presumably each pregnancy damages the endometrium underlying the implantation site, rendering the area unsuitable for implantation. Subsequent pregnancies are more likely to become implanted in the lower uterine segment by a process of elimination [4, 8]

The hallmark of placenta previa, is the sudden onset of painless vaginal bleeding in the second or third trimester of pregnancy. Bleeding may begin without an obvious inciting cause such as pelvic examination, intercourse or onset of labour. When the placenta is located over the internal os, the formation of the lower uterine segment and the dilatation of the internal os result inevitably in tearing of placental attachments. The bleeding is augmented by the inability of the myometrial fibres of the lower uterine segment to contract and thereby constrict the torn vessels [3,4, 8] Our patient presented with painless vaginal bleeding in the early third trimester.

On abdominal examination, the uterus is soft, with absence of palpable contractions in 75% of cases. Fetal distress is usually not seen unless the haemorrhage is severe. The lie is abnormal in up to 35% of cases but if the vertex is presenting it is usually felt, high above the pelvic brim [2, 8]

Per vaginally, there is fresh bleeding. Digital examination is contraindicated in any case of suspected placenta praevia. Speculum examination may be deffered until evidence that placenta

praevia does not exist is obtained, as extra-uterine causes of ante-partum haemorrhage are usually benign and thus need not be diagnosed urgently [1]. Earlier, the double set up examination was considered as the final diagnostic step in the management of placenta previa. With the increased accuracy of ultrasound the criteria for performing this examination have narrowed, and deserve re-evaluation [3, 8].

Trans-abdominal sonography is used to locate the placenta. It is highly accurate but not infallible, as there are no markers to locate the internal cervical os precisely. The rate of

false negative results with trans-abdominal sonography has been reported to be as high as 7%. The use of trans-vaginal ultrasonography has substantively improved diagnostic accuracy of placenta praevia [3, 4, 8]

There is evidence that low implantations are much more common in early pregnancy but the majority of these resolve and never become symptomatic. A low placenta may be seen in up to 40% of patient on ultrasound in the second trimester, but in 95% of these cases the placenta has moved away from the os by term. A centrally located placenta in the second trimester, despite the likelihood of subsequent upward migration with uterine enlargement, usually does not display the migration phenomenon. The mechanism of the apparent placental movement is not completely understood. [3,4,8]

Women with placenta praevia maybe considered as follows: Those in whom the foetus is preterm but there is no indication for delivery, those in whom the foetus is reasonably mature, those in labour and those in whom haemorrhage is severe as to mandate delivery despite foetal immaturity. Management with a preterm foetus, but with no active bleedings consists of close observation. In some cases prolonged hospitalization may be ideal. However, the woman can be discharged after bleeding has ceased and the foetus is judged to be healthy. The woman and her family must fully appreciate the problems of placenta praevia and be prepared to transport her to hospital immediately [2, 1, 1 in] our setup however, realistic, careful and somewhat subjective patient selection is required for safe outpatient management: patients who have economic disadvantages, little domestic support, young children at home, no telephone facilities or transportation difficulties and the unreliable blood transfusion facilities

are unlikely to be good candidates for this treatment approach. All patients are therefore managed as inpatients until delivery as was the case for our patient.

Early in pregnancy transfusions to replace blood loss and the use of tocolytic agents to prevent premature labour are indicated. If the patient is between 24 and 34 weeks gestational age a single course of dexamethasone or betamethasone (two doses of 12mg intramuscularly separated by 24 hours) should be given to promote foetal lung maturity [2, 8]

Caesarean section is the method of choice for central or partial placenta praevia. Bleeding from the placental bed may occur as the lower uterine segment is weakly contractile. Specific bleeding points if seen may be sutured but the use of multiple sutures to control a generalized bleed is usually futile. Direct injection of oxytocin, ergometrine or prostaglandin (15-methyl $PGF_{2\alpha}$) maybe successful in arresting the bleeding.

Other methods to arrest bleeding are uterine artery ligation or bilateral internal iliac artery ligation. Packing of the lower uterine segment has been suggested. Hysterectomy may be required for those cases where bleeding is not controlled by the above measures or when placenta accreta or percretta are present [1, 2, 3, 4, 8]

Vaginal delivery is usually reserved for patients with a marginal implantation and a cephalic presentation [2]. Double set-up examination is done in the operation theatre under anaesthesia, keeping everything ready for caesarean section. Palpation of the placenta on the lower segment not only conclusively confirms the clinical diagnosis but also identifies the degree. Low rupture of the membranes is done which helps in the initiation of labour and thereby encourages descent of the head. This in turn presses on the separated placenta and controls the bleeding. Oxytocin drip maybe started. If amniotomy fails to stop bleeding or fails to initiate labour, caesarean section is performed [9].

Rhesus immune globulin should be given to all at risk patients with third trimester bleeding who are rhesus negative and unsensitized [10]

1. Beischer N.A., Mackay E.V, Colditz P.B. Bleeding in Late Pregnancy. Obstetrics and the Newborn, an Illustrated Textbook P Edition. WB Saunders Company Limited, London, 1997:195 – 215. Ojwang' S.B.O. Placenta Previa in Kenyatta National Hospital. Mmed Thesis, University of Nairobi, 1974.

2. Mbithi D.L.K. Gray Scale Ultrasound in the Diagnosis of Placenta Previa at Kenyatta National Hospital. Mmed Thesis University of Nairobi, 1983.

3. Chauhan AR., Krishna U. Placenta Previa. Pregnancy at Risk, Current Concepts. 4th Edition, Jaypee Brothers Medical Publishers; New Delhi, . India, 2001:351-356.

4. Dutta. DC. (Ed). Antepartum Haemorrhage. Textbook of Obstetrics. Sixth Edition, New Central Book Agency, Calcutta, 2004:243-254

OBSTETRIC CASE NO. 5

IPS – SUCCESSFUL VAGINAL BIRTH AFTER CAESAREAN SECTION

NAME: A. W. **AGE** 27 YEARS

ID NO 1118969 **DOA** 27/09/06 **DOD:** 29/09/06

27 year old Para 1+0 G2 **LMP** 15/12/06 **EDD** 22/09/06 **GBD** 40/40, admitted with C/O Low abdominal pains x 12 hrs

HISTORY OF PRESENTING ILLNESS.

The low abdominal pains were intermittent, non radiating, increasing in duration and intensity. There were no urinary symptoms or history of drainage of liquor.

OBS/GYN HISTORY

Menarche was atl4 yrs. Her menstrual cycle lasts for 21 days, while the flow usually takes 5 days and is regular.

FP: Used OCP's for two months mid last year.

She had 1 previous scar due to eclampsia at KNH in 2001 at a gestation of 8 months, outcome was a fresh still birth male infant.

ANTENATAL HISTORY

A. W. had attended ante natal clinic at KNH, she had been booked at 16 weeks. Profile done: Blood group O Rhesus positive, Hb 13.4 gm /dl, VDRL and HIV- Negative. The ante natal period was uneventful.

FAMILY SOCIAL HISTORY:

She is a single lady, works as a receptionist at Jaribu credit traders. Takes alcohol occasionally but she doesn't smoke. Her mother is asthmatic. There no history of twinning in her family.

On **examination,** she was in fair general condition, not jaundiced, cyanosed, pale or oedematous.

Vital signs: BP 134/80 mmHg, PR 109 /min. Temp36.8°C, PR 22/min

enstiel incision scar, fundal height was term, longitudinal lie, 4/5 up. Foetal heart was heard at a rate of 150/min, regular. WE cervix was central, lcm long, 2cm dilated membranes were flat.

phase of labour in IPS was made .The plan was for trial of ıe was to be observed in labour ward and reviewed after 4 hours. The of scar was explained to the patient. Blood for grouping and cross) units of compatible blood was requested for and made available. An shed and 500mls of 5% dextrose infusion commenced. Once she got : was to be started on the partogram, where half hourly pulse, blood ıterine contractions and liquor colour observations were to be made ations and determination of the descent of the presenting part and o be made and charted on the partogram. Trial of scar was to be :veloped persistent pulse rate more than 100/min, foetal or maternal oor progress or obstruction of labour.

head was 3/5 up; foetal heart rate was 150/minute regular) minutes

ilated, artificial rupture of the membranes done, clear liquor

and review her after 3 hours.

: Head 1/5 up, foetal heart rate was 142/minute, regular, cervix was she was noted to have the urge to bear down with every contraction. ıd a live male infant with Apgar score 6/1, 10/5, 10/10 birth weight tramuscular Syntocinon IOU was given after the baby was delivered. by controlled cord traction. It

normal and complete. The lower segment was not explored, as the bleeding heavily. Estimated blood loss was 280mls.

ed for two hours in labour ward and the vital signs remained discharged to the floors. The following day the mother and the baby

were discharged home in good condition and were to be reviewed in the post natal clinic in six weeks time.

DISCUSSION

The patient presented was a 28 year old now Para 2+0 who had previously undergone emergency C/S due to eclampsia with poor cervical Bishop's score. She presented in latent phase of labour and progressed to deliver by spontaneous vertex delivery to a life male infant who scored well.

Vaginal birth after C/S (VBAC) commonly known as "Trial of scar" is the delivery of a baby by the vaginal route in a patient who has previously delivered by C/S. The rate of C/S has been increasing the world over and is causing concern. In KNH, the rate has steadily increased from an average 18% for the 1977-80 period [1,2] with repeat C/S accounting for 51.2% of the patients undergoing C/S [2] to an average of 20% in the 1986-89 period [1,4] By 1998 it stood at 29.1% [5]. Recent studies have shown that 60-80% of women with previous C/S who undergo a trial of scar have a successful vaginal delivery [6]

Certain criteria have to be followed when allowing a patient to undergo a "trial of scar" , [6, 7]-

1. The patient should only have one previous C/S delivery, which should not be a classical C/S and should not have a history of uterine rupture.
2. There should be no adverse medical or obstetric condition like hypertension, diabetes mellitus, breech presentation, bad obstetric history or multiple gestations which preclude vaginal delivery.
3. The original indication for C/S was not necessarily recurring in subsequent pregnancies.
4. The true conjugate as indicated by ELP must be equal to or more than 10.5cm.
5. The post-operative course of the previous C/S must have been uneventful.
6. The estimated foetal weight should not be greater than 3500gm.

Radiological pelvic assessment (ELP) should not be used as an absolute determinant of pelvic adequacy for vaginal delivery; the measurements taken do not assess foetal size or presentation and it does not take into account the intra-partum changes in pelvic and fetal

dimensions. It should not solely be used to repeat C/S unless there is obvious pelvic inadequacy clinically [8]

In order to carry out a successful trial of labour, other than a careful patient selection criteria, appropriate technical support must be available in the hospital. There should be a blood bank staffed 24 hours a day with compatible blood available promptly. Electronic foetal heart rate and intra-uterine pressure monitoring should be available. There should be adequate facilities and personnel to begin an emergency C/S within 30 minutes from the time a decision is made [9]. At KNH all the other facilities are available except the electronic fetal monitoring, but primary nursing and partogram have been found adequate for foetal and maternal monitoring. The first signs of uterine rupture are foetal distress and arrest of labour before onset of abdominal pains. The indications for C/S do not change in a trial of labour and should be resulted to as indicated. Our patient had an uneventful labour and progressed to deliver a healthy infant who scored well.

After the third stage of labour, the lower uterine segment used to be explored to determine the integrity of the previous scar. Some authors have questioned the usefulness of this procedure (as it can lead to the worsening of a minor asymptomatic laceration) and suggest that it should be done on symptomatic patients only [10]. This procedure is no longer routinely done in our department.

Although the rates of uterine rupture and neonatal asphyxia are slightly higher in women who attempted a vaginal delivery after C/S than in women who undergo elective C/S, maternal mortality rate do not reportedly differ between women undergoing a trial of labour and women undergoing an elective repeat C/S [11]. The advantages of trial of labour include reduced maternal morbidity, hospital stay is much shorter, the total cost of delivery is markedly reduced and finally the reduction in the C/S rate and its associated complications.

REFERENCES

1. Anabwani G.M. A Retrospective Study of the Outcome of Caesarean Section in KNH during the period March 1977 to February 1979. MMed Thesis, University of Nairobi, 1980.

2. Karanja J.G., Review of Caesarean Section deliveries at KNH in 1980. MMed Thesis, University of Nairobi 1983

3. Wanjohi E.N.,Risk Factors associated with wound infection after Caesarean Section delivery at KNH. MMed Thesis, University of Nairobi 1989.

4. Muriu F.K. A Prospective Study on Caesarean Section Pattern at KNH. MMed Thesis, University of Nairobi 1991.

5. KNH, Labour ward records.

6. Ralph W.H., David N.B.,Operative Delivery; Vaginal delivery following previous Caesarean Section. Current Obstetric and Gynaecologic diagnosis and treatment. 7t" Edition, Appleton and Lange, USA, 26:564-7, 1991.

7. Walton S.M., The ante-natal and intra-partum management of patients with one previous scar. East Aft. Med. Journ. 55(1) pg. 3, 1978.

8. Munjaja S.P.,A review of the use of X-Ray pelvimetry in Obstetrics. J. Obstet. Gynae East Cent Afr 7:19, 1988.

9. Cunningham F.G., MacDonald P.C., Grant N.F. Caesarean Section and Caesarean Hysterectomy. Williams Obstetrics, 18th edition, Appleton and Lange 1989 chap 22 pg.441.

10.SilbersteinT., Wiznitzer A., Kratz M. et al. Routine revision of uterine scar after Caesarean Section: Has it been necessary? Eur. J. Obstet. Gynaecol. Reprod. Biol. 1998 May; 78(1): 29-39.

11. Obara H., Minakami H., Koike T. et al. Vaginal birth after Caesarean delivery, results in 310 pregnancies. J. Obstet. Gynaecol Res April 1998 24(2):129-34.

OBSTETRICS CASE NO 6

OBSTRUCTED LABOUR-EMERGENCY C/S

NAME: W. K. AGE: 23 YEARS

IP NO: 0905767 DOA: 24/09/06 DOD: 29/09/06

23 year old Para 3+0 G4 LMP 09/01/06 EDD 16/10/06 GBD 37/40, admitted with C/O:-

Low abdominal pains x 4 hours.

HISTORY OF PRESENTING ILLNESS

The low abdominal pains were intermittent, radiating to the back but were not increasing in duration or intensity. There was no history of drainage of liquor, discharge of show or urinary symptoms.

PAST MEDICAL HISTORY

Not Significant

OBS/GYN HISTORY

Menarche was at 15 years. Her menstrual cycle lasts for 28 days, while the flow takes 4 days and is regular. There was no history of contraceptive use.

She is Para 3+0, all the children are alive and well.

ANTENATAL HISTORY

She attended ante natal clinic at a Nairobi City council Clinic.Profile done:-Hb 10.9 gm/dl, VDRL- Negative, HIV-Negative, blood group 0 Rhesus positive.

FAMILY SOCIAL HISTORY

She is a house wife while the husband is a businessman. There is no history of chronic illness in the family.

On examination, The patient was in fair general condition, not pale jaundiced cyanosed or oedematous

Vital signs: BP 120/70 mmHg, Temp 36.9°C, PR 72/min

P/A

Fundal height was term, longitudinal lie, cephalic presentation, head was 5/5 up, foetal heart was heard, rate 132/min and regular.

V/E: Normal external genitalia, the cervix was closed, posterior, 2 cm long, there was white curdy discharge on examining finger. An impression of patient in false labour with Vulvovaginal Candidiasis was made. She was put on IM Tramadal 100 mg stat and transferred to the antenatal ward on Clotrimazole pessaries 100mg nocte x 6/7. On 26/09/06 at 2.30 pm she was noted to be having labour pains and transferred to labour ward, where review at 3.15 pm showed:

P/A Fundal height term, longitudinal lie, head 4/5 up, foetal heart was heard and regular, with moderate uterine contractions 3: 40- 60sec: 10 minutes.

On V E the cervix was 8cm dilated, ARM was done, clear liquor was obtained. There was no caput or moulding noted. She was started on IV Syntocinon 5U in 500 mls of 5% Dextrose.

Review at 5 p.m. showed that the foetal head was 3/5 upon repeat WE she was 9 CM dilated. with caput of +I and moulding of 2+with meconium stained liquor grade I with the occiput still at the level of ischial spines. An impression of obstructed labour was made.

Management

She was informed of the diagnosis and the need for an emergency C/S. An informed written consent was obtained for emergency C/S. Meanwhile she was advised to lie on left lateral position. The IV Syntocinon drip was stopped and the patient started on 5% dextrose infusion. Blood was taken for group and cross matching. She was pre-medicated with Atropine 0.6mg intramuscularly. She was then wheeled to theatre and underwent C/S as described earlier in this book.

Intra-operatively, a live male infant was found in occipito- posterior position, Apgar score was 7/1, 9/5, 10/10 with a birth weight of 4.4 kg. Post operatively she did well. Baby's RBS was 3.4 mmol/L. After review by pediatrician, the baby was allowed to join the mother

Post operative management.

Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mls 4 hourly for the first 24 hours. She was also on parenteral

Crystalline Penicillin 2MU 6 hourly, Gentamycin 80 mg 8 hourly and Pethidine 100mg 8 hourly for 48 hours then oral Amoxil 500mg, Flagyl 400mgs and Brufen 400mgs for the next 3 days.

On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet. Patient was discharged home on the 4^{h} post operative day and booked for review at post natal clinic in two weeks.

DISCUSSION

The patient presented was a Para 3 + 0 G4 at term, who had been admitted in latent phase of labour, later, developed obstructed labour due to persistent occupito-posterior position and had emergency cesarean delivery to a live baby. Labour is considered obstructed when there is no progress in spite of strong uterine contractions [1]. Obstructed labour is rare in developed countries [2], but constitutes a major

Obstruction usually occurs at the inlet but may occur in mid cavity or at the outlet. This can be caused either by failure of the cervix to dilate or failure of the presenting part to descend through the birth canal. Obstructed labour is dangerous condition if not treated and can be fatal to both the mother and foetus [6,7].

Complications of obstructed labour if untreated can be fatal to both mother and foetus. The maternal complications include uterine rupture, puerperal sepsis, fistula formation, osteitis pubis, permanent nerve damage and loss of sensation or muscle deterioration in the feet and legs and ultimately maternal death [8]. A study on maternal morbidity and mortality at Garissa provincial Hospital found that 9% of all he maternal mortality was due to ruptured uterus [9]. Vesico-vaginal fistula (VVF) from obstetric causes are common in developing counties, especially in Africa and Asia [2]. The fetal complications include increased incidence of perinatal mortality and morbidity-including cerebral palsy.

Obstructed labour is the consequence of the following abnormalities which may exist singly or in combination:-Abnormalities of presentation, position or development of the foetus.Abnormalities of maternal bony pelvis (contracted pelvis) Soft tissue abnormalities of the reproductive tract. The maternal causes of obstructed labour include deformity of the bony pelvis, pelvic tumors such as leiomyomas, ovarian tumors, tumors of the rectum, bladder of pelvic bones and abnormalities of the uterus or vagina [1] Foetal causes of obstructed labour include macrosomia, malposition or malpresentation such as brow, shoulder, face, persistent occipito-posterior position or compound presentation and congenital abnormalities of the foetus such as hydrocephalus, foetal ascites or conjoined twins.

In a primigravida complete obstruction leads to a state of uterine exhaustion or secondary hypotonia while in multigravidae obstruction becomes established much sooner and progressive thinning of the lower segment may lead to uterine rupture [1, 8]. When the foetal head in stuck in the pelvis fora long time, portions of bladder, cervix, vagina and rectum are trapped between foetal head and pelvic bones and are subjected to excessive pressure. Circulation of these tissues is impaired, oxygen is inadequate and necrosis occurs leading to formation of a fistula in few days. Puerperal sepsis is a serious danger for the mother and foetus especially in cases of prolonged and obstructed labour.

Occipito-posterior position is an abnormal position of the vertex where it is placed posteriorly over the sacro-iliac joint or directly over the sacrum [10]. In the majority of the cases (90%), anterior rotation of the occiput occurs through 3/8 of a circle and then follows the normal course of labour like in occipito anterior position. However, in 10% of the cases, the occiput fails to rotate especially in cases of deflexion of the head, weak uterine contractions or a big baby (like in our case) leading to arrest of labour with marked compression of the occipito frontal diameter with the elongation of the vault at right angles to it. This type of moulding favours tentorial tear because of extreme elevation of falxi cerebri. If the head is below the ischial spines in an oblique occipito posterior position, then a vacuum extraction or forceps rotation and extraction can be attempted. In our case, the occiput remained at the level of the ischial spines so an emergency C/S was the only option available.

REFERENCES

1. Lewis T.L.T, Chamberlain G.V.P. Abnormal Labour-Obstructed Labour Obstetrics by Ten Teachers, 15th Edition, Book Power 19990; 7:209

2. Philpoh R.H. Obstructed Labour. Clinic in Obster and Gyrecol 1982; 9:625-40

3. Lawson J.B. Obstructed Labour. Obsterics and Gynecology in the Tropics and Developing countries. 1" edition 1967; 172-207

4. Wright E.A. Obstructed Labour. Nigerian medical Practitioner -1988; 18:397400

5. WHO. Maternal Mortality. World Health Organization: Safe Motherhood –WHO 1988:1-3

6. Cunningham F.G, McDonald P.C, Grant N.F. Dystocia due to expulsive forces. Williams Obstetrics. 19th Edition, McGraw-Hill, New York 1993; 19:475

7. De Cherney AH, Pernol M.L. Complications of Labour & Delivery. Current Obstetrics and Gynecology Diagnosis and Treatment. 8th Edition, Appleton and Lange, Norwalk, Connecticut 1998; 25:506

8. Ministry of Health. Obstructed Labour/Rupture of the Uterus-Essential Obstetric Care Manual for Health Service Providers in Kenya. A Safe Mother Hood Initiative 2002; 104-107

9. Jaldesa G.W, Qureshi Z.P, Kigondu CS, Wanjala S.M.H. Maternal Morbidity and Mortality at Garissa Provincial Hospital. J. Obster Gynecol East/Central Africa 1998; 14(2) 68-71.

10. Dutta D.C. Textbook of Obstetrics. 6th Edition, New Central Book Agency Ltd, Ch 35 pg 365-371

OBSTETRIC CASE NO 5

RETAINED PLACENTA- MANUAL REMOVAL

NAME: S. N. **AGE:** 29 YEARS

IP NO: 1006076 DOA: 26/01/05 **DOD:** 02/02/05 29

years old Para 2+0 LMP 29/06/04 GBD 30/40 admitted with C/O

Labour pains x 6 hours.

HISTORY OF PRESENRING ILLNESS

S. N. had spontaneous onset of labour while at home 6 hours earlier. There was history of associated drainage of liquor. There was no history of fever or urinary symptoms antedating the onset of the labour pains.

PAST MEDICAL HISTORY

Not significant

OBS/GYN HISTORY

She had her menarche at 14 years, her menstrual cycle lasts 28 with a flow of 4 days and is regular.

Contraceptive: Has never used any.1" delivery 1989 female infant alive and well, 2nd delivery 1990 female infant alive and well

ANTENATAL HISTORY

She attended ANC at KNH once at 24 weeks. Antenatal profile done: Blood group 0 Rhesus positive, Hb- 9.6 gm/dl, VDRL and HIV Negative

FAMILY SOCIAL HISTORY

She is a housewife, husband is a mason, does not take alcohol or smoke.

On examination, she was in fair general condition, not pale, jaundiced or cyanosed Vital signs: BP 110/63 mmHg PR 94/ min Temp 35.30C

P/A: Fundal height 28/40, no longitudinal lie, cephalic presentation, no foetal heart heard.

V/E: There was normal external genitalia, cervix was 6cm dilated, no membranes felt, draining clear liqour. An impression of IUFD with patient in labour was made. Patient was started on IV Oxytocin SU in 500m/s 5% dextrose. She delivered SVD at 8.50am to a live male infant Apgar score 1/1, 1/5, 0/10 birth weight 1250gm, however she developed a retained placenta.

Management

Blood was drawn for grouping and cross matching. An infusion of 500mls of normal saline with 40U of syntocinon was started but there was no response. On re-evaluation, the bladder was not palpably enlarged. On vaginal examination, the placenta was not separated.

The condition and the need for an emergency operation was explained to her and obtained an informed consent. A theatre list was prepared and IM Atropine 0.6mg given. Patient was taken to theatre, placed in supine position, given GA and then placed in lithotomy position. VVT was performed and the area drapped. A digital vaginal exam revealed the same findings as before. The right hand of the surgeon was inserted in to the vagina and cervix, the inferior border of the placenta was identified, and with left hand supporting the uterine fundus, a cleavage line was established between the placenta and uterine wall. The placenta was then sheared off and removed in one piece. It was carefully inspected and noted to be complete and healthy. The membranes were also removed whole. There was moderate bleeding which was controlled by giving IV Ergometrine 0.5mg stat, continuing with the syntocinon infusion and bimanual compression of the uterus. The estimated blood loss was 500mls.

Post operative care

The patient was maintained on an IV infusion of 500mls of normal saline with 20U of syntocinon for 4 hours. She was started on prophylactic antibiotics – Amoxyl 500mg and Metronidazole 400mg 8 hourly. She was also put Iron and folic acid supplementation and Ibuprofen 400mg 8 hourly. There was no need to transfuse her as she had not lost a lot of blood. Her vital signs remained stable, uterus remained well contracted and non tender. She was discharged home on the next day for follow up in the post natal clinic.

DISCUSSION

The patient presented was a 21 year old primigravida, who presented in established labour, progressed well to deliver a life male infant who scored well, but developed a retained placenta.

Physiologically the uterus should contract soon after delivery of the baby. The third stage of labour commences with the delivery of the infant and ends with the delivery of the placenta. The placenta normally separates from the uterine wall and is spontaneously expelled. There is no definitive length of time with in which this should take place, however obstetric tradition has set some arbitrary limit on the third stage duration in an attempt to define an abnormally retained placenta and thus reduce blood loss due to excessively prolonged placental separation.[1],

Typically the placenta separates and is delivered with in 5 minutes in 90% of cases and with in 15 minutes in 97% of cases. A placenta is said to be retained when spontaneous expulsion does not occur with in 30 minutes after delivery of the baby.[1], [2,3] The incidence of post partum haemorrhage, blood transfusion and dilatation and curettage increases progressively after 30 minutes of retained placenta.[4] In our patient the placenta had not been delivered 5 hours later and so a diagnosis of retained placenta was made.

After the birth of the baby, uterine contractions have been shown to continue often causing little discomfort to the patient but causing a rhythmical rise in intrauterine pressure. This rise in pressure coupled with shortening of the muscle fibres reduces the surface area of the uterine surface to which the relatively incompressible placenta is attached, thus aiding the separation of the placenta either from the margins (Duncan method) or from the central position (Schuze method) with the formation of a clot in the retro-placental space that then shears off the placenta from the uterine wall. [1,2,3,5]

Signs of placental separation include lengthening of the umbilical cord and a gush of blood through the vagina, a firmly contracting and rising uterine flindus and change of the uterine shape from discoid to globular. The placenta may then be delivered by the

Brandt-Andrew or Pastore manouvre or can be left alone to deliver physiologically. In our unit active management is preferred and routinely practiced. No signs of separation were noted in our patient.

The incidence of retained placenta varies. In a recent study at KNH, Onyango reported an incidence of 0.76% [6] A variety of factors may favour the occurrence of retained placenta. These include:

- Improper management of the third stage of labour especially attempts to pull on the cord before complete separation of the placenta, kneading of the fundus and Crede's manouvre where the uterus is used as a piston to expel the placenta.
- Preterm delivery.
- Age and multiparity.
- Insufficient uterine contractions due to atony or inertia.
- Placental entrapment by spasm or a contracted cervical ring.
- Congenital abnormalities of the uterus such as bicornuate uterus.
- Leiomyoma of the uterus.
- Abnormal implantation sites of the placenta e.g. succenturiate lobe.
- Previous surgery on the uterus e.g. Caesarean section, myomectomy.
- Over zealous uterine curettage and previous manual removal of the placenta.
- Endometritis of any kind.
- Placenta accreta, increta and percreta.

In view of the fact that initial delivery of the placenta occurred elsewhere it is difficult to state categorically that there was no predisposing factor in our patient.

Retained placenta is a cause of post partum haemorrhage, puerperal infection and even maternal death. Rupture of the uterus and inversion may follow a too vigorous attempt at extraction of the placenta.

Retained placenta is associated with fivefold increase in postpartum haemorrhage. Often in the absence of haemorrhage, a delay of 30 minutes with administration of oxytocic drugs to hasten placental detachment and the subsequent expulsion is permissible but in the presence of haemorrhage manual removal must be undertaken without delay. Foc

partial adherent will in most cases be handled successfully since there is some line of cleavage to insinuate the fingers through. The reported incidence of invasive placenta varies from 1 in

540 to 1 in 93,000 pregnancies.[7] Three types of invasive placentation have been described: Placenta accreta, in which the chorionic villi invade the decidua but not the myometrium, placenta increta in which the villi invade the myometrium and placenta percreta in which the villi penetrate through the myometrium reaching the serosa and often rupture into peritoneal cavity. Invasive placenta has been shown to be associated with poorly developed deciduas. [1,3,8]

Antecedent risk factors associated with invasive placenta include uterine scar usually from caesarean section, uterine curettage and placenta praevia. [3, 7, 8] The diagnosis of placenta increta and percreta is retrospective and only found when laparatomy and hysterectomy are performed. In extremely adherent placentas, hysterectomy may have to be resorted to especially if there is profuse bleeding. In some cases in which the patient is young and wants to retain her productive capacity and is not bleeding profusely, the placenta may be left in situ, the patient covered with antibiotics and observed closely with serial ultrasound, total and differential white blood cell count and P-HCG titers; alternatively methotrexate therapy maybe used which acts by reducing vascularity and enhancing the process of necrosis of the placenta. [2,5,9]

Our patient had successful manual removal of the placenta, was covered with prophylactic antibiotics and put on haematenics. Complications of manual removal of placenta include postpartum haemorrhage especially with morbidly adherent placenta, uterine rupture, inversion and infections. [3, 10] When part of the placenta is morbidly adherent and is not removed, the bleeding may be minimal or the patient may continue to bleed. Patients with minimal bleeding should be observed and warned of the possibility of secondary postpartum haemorrhage which usually occur 10-14 days post delivery. If bleeding continues and patient is desirous of having more children, conservative management with oxytocin or prostaglandins should be tried while internal iliac ligation or hysterectomy are the last resort.

REFERENCES:

1. Polo A, Chang L, Herrera W. et al. Complications of labour and delivery. Current Obstetrics & Gynaecologic diagnosis & treatment. 9th edition. Mc Graw- Hill, New York 2003; 25: 466-476.

2. Cunningham F. G, Norman F. G, Kenneth J. V. Post partum. haemorrhage. Williams Obstetrics, 21st edition. Churchill-Livingstone, New York 2002; 25:635-652.

3. Breezly J. M. Complications of the third stl* e of labour. Dewhurst's textbook of Obstetrics and Gynaecology for postgraduates. 5 edition. Black science, Massachusetts 1995 ; 25: 368-376.

4. Combs C. A, Laros R. K. Polonged Third Stage of Labour: Morbidity and Risk Factors. Obstet. Gynaecol. 1991; 77(6):863.

5. Oxorn H. The retained placenta and placenta accrete. Oxorne-Foote, Labour and birth 5th edition, 1988; 35:555-565.

6. Onyango S. J. 0, Factors influencing retained placenta at KNH, MMed Thesis, University of Nairobi, 2000.

7. Mark D.,Hamstein, Jonathan M.N., et al. Placenta percreta associated with second trimester pregnancy termination. Am. J. Obstet. Gynecol. 1984; 50: 102.

8. Frank Loeffler. Postpartum haemorrhage and abnormalities of the third stage of labour. Turnbull's Obstetrics. 2nd edition, Churchill-Livingstone, Massachusetts 1995; 4:729

9. Donald I. Retention of the placenta without bleeding. Practical Obstetric problems 5th edition 1989; 790-792.

10. Zuspan F.P., Quilligan E.J. Retained placenta, placental cotyledons and membranes. Douglas Stromme's Operative Obstetric. 5th edition 1988; 587-590.

OBSTETRIC CASE NO 8

CARDIAC DISEASE IN PREGNANCY NYHA GRADE II : LIVE BIRTH

Name: R. W. **Age:** 20 years

IP.NO.- 0738306 D.O.A: 29/3/06 **DOD:** 9/04/06

20 year old Para 0+0 G1 LMP 13/07/05 EDD 20/04/06 admitted to the ante natal ward from clinic 18 because she was 37/40 gestation in a known cardiac patient. She did not have any complains

HISTORY OF PRESENTING ILLNESS

She was a known cardiac patient since she was 2 weeks old with an underlying VSD and developed difficult in breathing and easy fatiguability on normal activity. She had been admitted several times for the management of above complaints. The repair of the VSD had been done May 2002 here at KNH.

PAST MEDICAL HISTORY

She had many childhood admissions due to the cardiac disease and was followed in the cardiac clinic. VSD was repaired in May 2002 after which inderal, lasix and digoxin were stopped. There was no history of any other chronic illness, drug or food allergy and /or throat or chest infections.

OBS/GYN HISTORY

She was Para 0+0. She attained her menarche at 12 years. Prior to the current pregnancy, her periods were regular coming every 28 days and lasting 4 days. It was an average flow with no associated dysmenorhoea. She had no history of contraceptive use.

ANTENATAL HISTORY

She began attending ante natal clinic at KNH at 20 weeks gestation. Her ante natal clinic profile were done; Blood group - 0 positive, VDRL negative, HB - 13.5g^{O}/o, height - 5.25ft

Started on Ranferon. Given 2 T.T. injection. Had 8 - visits which were uneventful.

FAMILY SOCIAL HISTORY

She was a secretary with Information Technology Company. Her husband was a Clinical Officer working at Pumwani Maternity Hospital. She did not take alcohol or smoke cigarettes. There was no known family history of heart disease, hypertension, tuberculosis, diabetes or any other chronic illness.

On examination, she was in fair general condition, she was comfortably propped -up in bed. She had mild bilateral pitting pedal edema. She had no Jaundice or lymphadenopathy. She did not have finger clubbing, cyanosis or splinter haemorhages. Vital signs: Temp 36.5°c, PR - 80/min, B.P. - 130/80 RR - 20/mm

CVS

The pulse was regular and of good volume, it was non collapsing and there was no radio-femoral delay. The jugular venous pressure was not elevated. Had a healed thoracotomy scar: Precodium was slightly active. Apex beat was at 6^{h} intercostal space along the anterior axillary line. Precordial thrills were present. A pan systolic murmur loudest at the left sternal border radiating to the back was heard, first and second heart sounds were also heard.

R/S:

She was not in respiratory distress. She had normal vesicular breath sounds over all lungs fields. She had no crepitations or rhonchi.

PA:

The abdomen was uniformly distended and moved with respiration. The fundal height was corresponding to 38 weeks gestation. The fetus was in cephalic presentation, and longitudinal lie. The foetal heart rate was 144 beats/min and regular.

Foetal head was 5 fifths above the pelvic brim. The liver and spleen were not palpable. There was no tenderness on the right hypochondric region. Pelvic exam was not indicated and therefore was not done

Impression Cardiac disease in pregnancy at 37/40 was made, NYHA Grade II.

Investigations:

1) Haemogram3 1 /4/06

Hb - 11.7g% MCV 82.3fl. MCH:24.2 WBC 8.59X 10^{9}/ L Platelets 201 x $10^{9/1,2}$

U/E: Na+ - 136mm/L K+ - 4.6mm/L Urea 2.9mmol/L Creat - 91 gmol/L

Urinalysis - Normal finding on microscopy, culture - no growth obtained.

ECG : Showed normal sinus rhythm, rate 86/min

Impression: Cardiac Disease Grade II at 37 weeks.

She was admitted to the ward for bed rest to await spontaneous onset of labour. Six days later while in the ante natal ward she was noted to be contracting and was then transferred to labour ward.

Management of labour

Review in Labour Ward showed that she was in early active phase of labour, 3cm dilated and the fetal head was 4/5 up with regular fetal heart rate and good contractions. She was put in left lateral position and propped up. An IV line was put, oxygen by mask was given and prophylactic antibiotic of crystalline penicillin and gentamycin started. An emergency tray containing aminophyline, digoxin, frusemide, sodium bicarbonate and calcium gluconate was prepared and placed nearby. A vacuum extractor was also kept in readiness for use during second stage.

She was found to be progressing well in subsequent reviews and within the *next* 6 hours delivered via assisted vacuum delivery a life female infant birth weight 2580 gms who scored 7/1, 9/5, and 10/10. The placenta was delivered by controlled cord traction. An IV drip with 20U syntocinon was run after delivery and IV frusemide 80mg given.

Post delivery care

She was transferred to the acute room and kept in propped up position. Vital signs were observed $^{1}/_{2}$ hourly and she continued on IV antibiotics and haematinics. She was reviewed by a cardiologist and found to be stable. She was transferred to the postnatal ward after 48 hours. She did not develop any complications post nataly and so was discharged from the ward on the fourth post natal day, to be reviewed in the post natal clinic after 6 weeks and to continue with the routine follow up in the cardiac clinic at KNH. She was advised on using barrier method (condom), or Progesterone only pill so as to have adequate rest of her system before the next delivery.

DISCUSSION

The case presented is a 20 year old primigravida, known cardiac disease patient who had undergone closure of a ventriculo-septal defect in May 2002. She was admitted at term so as to

be able to undergo the process of labour under close supervision. She delivered via assisted vacuum delivery to a live female infant, birth weight 2580gm who scored well. She was discharged home with a healthy baby on the 4th post natal day.

Cardiac disease complicates 1-2% of all pregnancies and is the most important non-obstetric cause of maternal death Sequeira and Ojiambo in 1969 at Kenyatta National Hospital found an incidence of 0.5% with 95% of cases being of rheumatic heart disease origin. 35% of the RHD cases had mural stenosis 3.

In a later study Ngotho reported an incidence of 0.99% 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 predominates.

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 childbearing but also carry a pregnancy to term successfully 5 - R.W. had a VSD which is a congenital anomaly. 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.

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. The grades are:

Grade I. No symptoms limiting ordinary physical activity. Grade II. Slight limitation with mild to moderate activity but no symptoms at rest. Grade III. Marked limitation with less than ordinary activity; Dyspnea or pain on minimal activity Grade IV. Symptoms at rest or with minimal activity and symptoms of frank congestive cardiac failure.

The management calls for team approach involving obstetrician, cardiologist and anaesthesiologist [1, 2, 6, 7] Our patient had been stable after the closure of the VSD and was classified as grade II.

Grade I & II patients are managed as outpatient after initial 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. Grades III and IV patients are usually confined in the wards until delivery [1,2,6,7]

Restriction of maternal physical activity tends to avoid cardiovascular compromise and improves uteroplacental perfusion?. 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.Our patient had an Hb of 11.7gldL and was started on haematinics - Ranferon one tablet 12 hourly.

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 hemodynamic changes that could precipitate cardiac failure and in case of failed induction caesarean section carries an added risk of pneumonia, infective endocarditis and pulmonary edema and embolism [8]. However, caesarean section should still be performed if there is an obstetric indication [5].

Relief of pain and apprehension without undue depression of the infant or mother is especially important during labour and delivery. Epidural anaesthesia and narcotic analgesics are preferable. 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 III and IV patients Digoxin and furesemide 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 fiusemide is kept, ready for use if need arises. Vaginal delivery should be aimed at, and shortening of the 2nd stage by use of elective vacuum extraction. Active management of third stage with ergometrine should be avoided and syntocinon used early if bleeding is excessive otherwise uterine massage minus drugs is encouraged.

A bolus of frusemide 40-100mg, is given late in the first stage to offset the anticipated cardiac output increase from the placental bed. Close obstetric and medical surveillance must continue particularly during the first 24-48 hours and infection guarded against especially infective endocarditis. Early ambulation is necessary to prevent deep venous thrombosis and the attendant risk of pulmonary embolism. A period of 10 to 14 days postnatal observation is recommended[8].

Before discharge contraception should be discussed with the patient. Barrier methods and progesterone only pill is advisable for those who desire another child or do not opt

for tubal ligation [6]. When the family size is complete tubal ligation is the optimal choice. Alternatively vasectomy can be offered to the spouse if desired. Our patient opted for progesterone only pills and barrier methods..

REFERENCES.

1.Cunningham F.G., Norman F. G., Kenneth J.V. Cardiovascular Diseases. Williams Obstetrics 21" Ed. Mc-Grave Hill 2002: 1181-1203

2. De Cherney A.H. and Pernoll. Cardiac, Haemotologic, Pulmonary, Renal and Urinary Tract; Disorders in Pregnancy. Current Obstetric and Gynaecologic Diagnosis and Treatment. 8th Ed. Appleton and Lange 1994; 428-448

3 Sequeira, R. F. and Ojiambo H. P. The Heart in Pregnancy. East Afri. Med. J. 46(1); 18, 1969

4. Ngotho D. K. Cardiac Disease at Kenyatta National Hospital. M.Med Thesis U.O.N. 1982

5. Schoon M.G., Bann R. H., Wolmarams L. Heart Disease during Pregnancy S. Afri. Md. J. Feb 1997; 87 Suppl. IPC 1922

OBSTERTIC CASE NO 9

CERVICAL INCOMPETENCE- McDONALD STITCH INSERTION LIVE BABY.

NAME: H. N. AGE: 30 YEARS

IP.NO: 0861414 D.O.A :14.04.2004 **D.O.D :** 18.04.2004

30 year old Para 2+2 gravida 5 LMP 14/01/2004 EDD21/10/04 GBD14/40, she did not have any complaints at the time of admission.

HISTORY OF PRESENTING ILLNESS

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

PAST MEDICAL HISTORY

Not significant.

OBS/GYN HISTORY

Her menarche was at 15 years and the menses lasted 4-5 days with a regular cycle of 26 to 28 days. She had not used any form of contraception.

She was a pars 2+2 with one living child. In 1997 she had an abortion at 5 months no evacuation was done.. In 1999 she had a spontaneous vertex delivery at 8 months to a female live baby who is still alive and well. In 2002 she had spontaneous vertex delivery at 8 months to a live male baby who passed away at 2 months due to pneumonia. In 2003 she had a spontaneous abortion at 5 months and evacuation was done. Both fetal losses started with drainage of liquor followed by abdominal pain and later expulsion of the respective fetuses. She had no history of trauma or febrile illness associated with the abortions.

ANTENATAL HISTORY

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

FAMILY SOCIAL HISTORY

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

On examination, she was a young woman in good general condition with no pallor, jaundice cyanosis, edema or lymphadenopathy.

Vital signs: BP 115/70 mmHg, Temperature was 36.8C, pulse rate of 76 beats per minute and respiratory rate of 19 per minute.

CNS, CVS and RS were normal.

P/A

She had slight lower abdominal distension. There were no areas of tenderness. The uterine fundus corresponded to 14 weeks gestation. There were no other palpable masses. Speculum exam: She had normal external genitalia with normal vaginal walls. The cervix was patulous and short but with no visible defect.

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

An impression of a patient with foetal wastage secondary to cervical incompetence was made and she was planned for insertion of MacDonald stitch.

Investigations:

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

Urinalysis for microscopy culture and sensitivity was reported to be normal. Hospital in case she developed lower abdominal pain, drainage of liquor or vaginal bleeding. She was to be reviewed in the antenatal clinic in two weeks. The stitch was to be removed at 37 completed weeks.

Re-admission.

She continued with antenatal clinic uneventfully during her six subsequent visits. The stitch was removed in labor ward on 27.09.2004. She came back in labor on the 10. 10.2004 at 39 weeks. Labor progressed well and delivered by spontaneous vertex delivery to a live female infant weighing 3200 grams and scored 7 in one minute and 10 in ten minutes. She and her baby did well and were discharged the following day to be seen in the post-natal clinic in six weeks.

Follow-up

She was lost to follow-up.

DISCUSSION.

The patient presented was a 30 year old Para 2+2G 5 with consecutive foetal wastage secondary to cervical incompetence. She had McDonald stitch inserted at 14 weeks gestation and delivered a live baby at 39 weeks gestation.

In normal pregnancy the cervix remains closed and retains the products of conception within the uterus and during the third trimester, the cervix softens in preparation for parturition. Cervical incompetence can be due to congenital or acquired factors. Some women experience cervical effacement and dilatation with every pregnancy; others have one or more uncomplicated births at term before presenting with the typical manifestations of cervical insufficiency. With cervical insufficiency, there is second trimester or early third trimester foetal loss characterized by painless cervical dilatation with prolapse and ballooning of membranes into the vagina followed by rupture of membranes and expulsion of an immature foetus.[1] The patient presented had two consecutive second trimester pregnancy losses before a McDonald stitch was inserted. The incidence of cervical incompetence ranges from 0.005 —1 per 100 pregnancies.2

At KNH, Njagi reported an incidence of 1 in 90 deliveries.[3] The risk of preterm delivery rises by four times after one preterm delivery and about 10 % of preterm deliveries are caused by true cervical incompetence.[4] The classic presentation of cervical insufficiency is cervical dilatation and effacement in the second trimester with foetal membranes visible at or beyond the external os in the absence of contractions. It may be asymptomatic or associated with

one or more of the following: vaginal fullness or pressure, vaginal spotting or bleeding, an increased volume of watery, mucoid or brown vaginal discharge or vague discomfort in the lower abdomen or back.

Cervical incompetence should be suspected in a patient with recurrent second trimester pregnancy losses. Other more subtle markers of reduced cervical resistance include soft cervical consistency on digital examination, a history of short labours, advanced dilatation before the onset of labour, and progressively earlier deliveries with each successive pregnancy. Funneling of foetal membranes into or completely through the endocervical canal (i.e. hour glassing) or shortening of cervical length and dilatation of

internal cervical os are indicators of cervical incompetence on the ultrasound examination. [5,6]

The aetiology of abnormal cervical function can be divided into two major categories: congenital abnormality and trauma although factors such as uterine over distension and biochemical abnormalities also play a role. Developmental causes of reduced cervical competence include: congenitally short cervix, Mullerian duct abnormalities and in utero exposure to diethylstilboestrol.

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

Traumatic causes of cervical incompetence may be physiological or iatrogenic. Among the iatrogenic causes include:-

- Cervical laceration following spontaneous vaginal delivery
- Prolonged second stage of labour
- Instrumental vaginal delivery
- Cervical injury at the time of Caesarean delivery
- Surgical procedures involving the cervix e.g. mechanical dilatation, cone biopsy and cone biopsy as done in management of CIN [8, 9].

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

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

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

The treatment of cervical incompetence is surgical, consisting of reinforcement of the weak cervix by some type of purse string suture. It is best performed after the first trimester but

before cervical dilatation of 2-3 cm is reached'. The best time for insertion is at 14 weeks so that early abortion secondary to causes like congenital and genetic abnormalities will be complete by then. Njagi found out that McDonald stitch gave the best results if it was done between 13 – 19 weeks of gestation3.

Ultrasound should be done to exclude fetal congenital anomalies and to confirm a living foetus before cerclage'. If substantial dilatation of the cervix has occurred, or bulging of the membranes has occurred, then the likelihood of a successful cerclage is lessened. An attempt can be made to replace the protruding membranes with a balloon which is then deflated and removed[2]. The patient presented was done cerclage at 14 weeks gestation.

Three types of operation are commonly used during pregnancy including McDonald, Shirodikar and modified Shirodikar. There is less trauma and blood loss with both McDonald and modified Shirodikar than with the original Shirodikar – which is preserved for previously failed McDonald procedure and structural cervical abnormalities'[2]. McDonalds stitch can be removed and so can the modified Shirodikar, unlike the conventional Shirodikar, which was a permanent stick requiring Caesarean section for delivery. Trans-abdominal cerclage may be appropriate in rare circumstances. These include traumatic cervical lacerations, congenital shortening of cervix, advanced cervical dilatation and previous failed vaginal cerclage [2, 11]. Disadvantages include need to perform two operations (one for suture replacement and another for Caesarean section delivery), and risk of injury to uterine vessels and ureter2.

Contraindications to cervical cerclage include:-

- Rupture of membranes
- Uterine bleeding
- Uterine contractions
- Chorio-amnionitis
- Cervical dilatation greater than 4 cm
- Poly-hydramnios

- Fetal anomaly" 2

Complications of cerclage especially when performed after 20 weeks gestation are high. The complications include:-

- Haemorrhage
- Rupture of membranes
- Infection (chorio-amnionitis and septicaemia)
- Induction of preterm labour
- Cervical dystocia
- Cervical laceration or uterine rupture at time of delivery
- Vesico-vaginal fistula formation
- Foetal deaths' 2.

- McDonald stitch is removed at 36 completed weeks of gestation, or if there is vaginal bleeding, drainage of liquor or premature labour sets in. This patient came at 38 weeks in labour and the stitch was removed.

- The success rates with both McDonald and modified Shirodkar techniques approach 85-90%[1]. Njagi found the success rate leading to term pregnancy to be 55% and 64.2% in foetal survival[3]. Most case series quote a viable delivery rate of 70 – 90 % after cerclage, compared to 10-30% prior to the proCedUre12.

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

- Conservative management requires patients to have bed rest at home and weekly ultrasounds are done from 15 weeks of gestation and if shortening at or below the critical length of 15 mm is noted, emergency cerclage is done. All cerclages are to be removed if there is evidence of preterm rupture of membranes, preterm labour unresponsive to tocolytics, or attainment of 36 completed weeks of gestation.

- Therapeutic bed rest is the most commonly employed non-surgical approach to the management of incompetent cervix, although its benefit has never been proved in randomized clinical trials[13]. Other suggested modalities include intramuscular hydroxyprogesterone plus a program of bed rest [14], and use of several types of vaginal pessaries and inflatable balloons in an attempt to change the axis of the cervical canal, thereby altering the gravitational force of intra-uterine contents on the cervix. There is generally no indication for tocolytic therapy unless preterm labour is also present. The lady presented here was done cervical cerclage and delivered of a live male infant.

REFERENCES

1. Cunningham F. G, MacDonald P.C, Grant N.F. Reproductive successes and failure. Williams Obstetrics. 20th edition McGrow-Hill, New York 1997; 26:588590.
2. Grimes D. A. Cervical incompetence. Telinde's Operative Gynaecology. 8th edition Lippincort-Raven Publishers, Philadelphia 1997: 481-483.
3. Njagi P.E.N. The management of cervical incompetence by cervical cerclage in KNH. MMed Thesis University of Nairobi 1979.
4. Kump M, Goldkrand J.W. Cervical incompetence: Elective, emergent or urgent cerclage? AM. J Obstet/Gynecol 180:366 (1999)
5. Marder S. J., Jackson M., Sonographic examination of incompetent cervix during

pregnancy. Semin Roentgenol 1999; 34:35.

6. Cousins L., Cervical incompetence 1980: A time for reappraisal. Clin. Obstet. Gynecol 1980; 23:4675.

7. Iams J. D.,Goldenberg R.L., Meis P.J. et al. The length of the cervix and the risk of spontaneous premature delivery. National Institute of Child Health and Human Development Maternal Fetal Medicine Unit Network. N Eng J Med 1996; 334:567

8. Golan A. Langer R., Wexler S. et al. Cervical cerclage – its role in the pregnant anomalous uterus. Int J Fertility 1990; 35:164.

9. Grunberger W. Riss P., Cervical incompetence after previous cervical dilatation and curettage. Wien Med Wochenschr 1979; 129:390.

10. Kagia J. W. Radiological diagnosis of cervical incompetence at KNH in 1979. MMed Thesis, University of Nairobi 1980.

11. Davis G., Bergella, V., Talluci M. Patients with prior failed transvaginal cerclage: A comparison of obstetric outcome with either trans-abdominal or trans-vaginal cerclage. Am J. Obstet Gynecol, 2000; 183:836.

12. Rush R.W., Isaacs S., McPherson K. et al. A randomized controlled trial of cervical cerclage in women at high risk of spontaneous preterm delivery. Br J Obstet Gynaecol 1984; 91:724.

13. Goldenberg R.L, Cliver S. P., Bronstein J et al. Bed rest in pregnancy. Obstet Gynecol 1994; 84:131.
14. Sherman A. Hormonal therapy for the control of the incompetent os of pregnancy. Obstet Gynecol 1966;28:198.

OBSTETRIC CASE NO 10.

TWIN GESTATION –BOTH BREECH DONE EMERGENCY CIS WITH GOOD OUTCOME.

Name: A.H. Age:25years

D.O.A:22/4/06 D.O.D:26/4/06

25 year old Para 3 + 0 LMP 14/7/05 EDD 21/4/06 GBD 40/40 admitted with C/O

Low abdominal pains x 1/7

HISTORY OF PRESENTING ILLNESS

The low abdominal pain was increasing in frequency and intensity, and was radiating to the back. There was no associated history of drainage of liquor or per vaginal discharge / bleeding. There were no urinary symptoms.

PAST MEDICAL HISTORY

Not significant

OBS/GYN HISTORY

Had attained menarche at 14 years, her cycle is 3/28 days, regular Contraceptive history. Had used DMPA after the first delivery for 1 year.

I" delivery was in 1994 in hospital SVD to LMI who is still alive and well. 2nd delivery was in 1996 in hospital SVD to a LFI who is alive and well. 3rd delivery was in 1998 at home SVD to a LMI who is alive and well

ANTENATAL HISTORY

Attended ante natal clinic at St. Mary's Hospital, made 4 visits, the period was uneventful. Profile done :Blood Group 0 Rhesus positive, P24 - non reactive Hb - 10.5gm/dl, VDRL – Negative.

She received two doses of TT.

FAMILY SOCIAL HISTORY,

She is a housewife, husband is a police officer. She does not smoke or take alcohol. There is no history of chronic illness or twins in the family. She is a Standard four graduate and resides in Eastleigh. **On examination,** She was in fair general condition, not pale, aundiced or cyanosed. She was a febrile but had bilateral pedal pitting oedema. Vital signs: BP 116/73mmHg, PR 84/min, RR 24/min

CVS/R/S/CNS — Essentially normal. **P/A** Fundal height term, longitudinal lie, multiple fetal parts felt, both twins breech, FHHR V/E: Normal external genitalia, cervix central, 3 cm dilated, breech presentation, no cord felt, membranes intact.

Obstetric scan done on 18/04/06 had shown twin gestation, both breech.

An impression of twin gestation, both breech at term was made

Management

She was informed of the diagnosis and the need for an emergency C/S. An informed written consent was obtained for emergency C/S. Meanwhile she was advised to lie on left lateral position. An intravenous access line was fixed and patiend started on 5% dextrose infusion. Blood was taken for group and cross matching. She was pre-medicated with Atropine 0.6mg intramuscularly. She was then wheeled to theatre and underwent C/S as described earlier in

this book.

Intra-operatively she was found to have dichorionic diamniotic twins; first was in breech presentation, life female infant Apgar score 8/1, 9/5, 10/10 birth weight 2500grns, the second one was also a life female infant in breech presentation, Apgar score 7/1, 8/5 and 10/10. Post operatively she did well. After review by pediatrician, the babies were allowed to join the mother

Post operative management.

Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mls 4 hourly for the first 24 hours. She was also on parenteral Crystalline Penicillin 2MU 6 hourly, Gentamycin 80 mg 8 hourly and Pethidine 100mg 8 hourly for 48 hours then oral Amoxil 500mg, Flagyl 400mgs and Brufen 400mgs for the next 3 days.On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet. Patient was discharged home on the 4^{h} post operative day (26/05/06) and booked for review at post natal clinic in two weeks.

DISCUSSION

A. H. was a 25 years old Para 3+0 with diagnosed twins who underwent emergency Caesarean delivery to two life female diamniotic dichorionic infants who had good Apgar scores. She had been diagnosed with twin pregnancy earlier on during her routine ante natal follow up in a private hospital and only showed up in our unit in early labour.

Twin pregnancies are usually associated with higher maternal and foetal morbidity and mortality than in singleton pregnancies. There are two types of twins namely monozygotic and dizygotic. In monozygotic twins a single fertilized ovum splits into two distinct individuals after a variable number of divisions. These twins are almost always genetically identical and therefore of the same sex. Monozygotic twins are referred to as identical twins. Dizygotic twins or fraternal twins arise when two separate ova are fertilized. These individuals are genetically distinct as any other children born

same couple Monozygotic twinning is a random phenomenon. The frequency is fairly constant throughout the world at approximately 4 per 10,000 births. The incidence of dizygotic

twinning varies widely and is affected by several identifiable factors. The frequency of dizygotic twins is low in Asia, intermediate in whites and high in blacks. Japanese and Chinese have the lowest incidence of dizygotic twins and Nigerians have the highest incidences of 50 per 1000 live births. [1, 2]. The incidence of dizygotic twins is affected by maternal age, parity and ovulation induction. Also dizygotic twins occur in taller and heavier women. Dizygotic twinning also tends to have a familial tendency. Dizygotic twinning is associated with multiple ovulations. The different rates of dizygotic twinning are probably due to variations in pituitary gonadotrophin production. Infertile patients treated with menopausal urinary gonadotrophins or Clomiphene citrate are well known to have a dose dependent increase in multiple birth when compared with women who conceive without these agents [1, 2]. The incidence of twin and higher order multiple births has increased significantly over the past 15 years primarily because of the availability. Our patient had dizygotic twins, both female. and increased use of ovulation inducing drugs and newly developed assisted reproductive technologies such as in vitro fertilization [2,4]

Locally, Oyieke in 1978 found an incidence of 1:58.8 deliveries at Kenyatta National Hospital [6], Mutungi in 1990 found an incidence of 1:46 deliveries at Kenyatta National and Pumwani Maternity Hospital [7] Our patient was a 25 year Para 3+0 and had not taken ovulation-induction drugs. However there was history of twinning in the family, i.e. maternal auntie has twins.

Twin pregnancy is associated with an increased number of complications both maternal and fetal which are of clinical importance. These complications include hydramnios, pre-eclampsia, anaemia, postpartum hemorrhage, malpresentations, high perinatal morbidity and mortality, fetal abnormalities, premature rupture of membranes and high risk of

The diagnosis of multiple gestations (twin pregnancy) is based on clinical suspicion. Multiple gestation is suspected whenever the fundal height is larger than expected for gestational age by menstrual dates, also whenever multiple fetal parts are palpated. Multiple gestations should also be suspected whenever hydramnios or unexplained maternal anemia develops, auscultation of more than one fetal heart and the pregnancy which has occurred following ovulation induction or in vitro fertilization. Ultrasonography is usually used to confirm the diagnosis. Ultrasound can diagnose twins as early as 6 weeks by identification of separate gestational sacs. Fetal cardiac activity

can be demonstrated 7 to 8 weeks [4]. In some cases the diagnosis is missed altogether antenatally and only discovered at delivery. Mutungi reported that the correct diagnosis was made in only 40.8 per cent of patients ante-natally while 44.1 per cent were diagnosed either in labor or after the delivery of the first twin. The diagnosis was missed completely in 54.9% of the patients. . Based on clinical findings the differential diagnoses of multiple pregnancies include distended bladder, hydramnios, wrong dates, hydatidiform mole, uterine myomas and fetal macrosomia. Other tests, which can increase in suspicion of multiple gestations but are not confirmatory, include elevated levels of PHCG, estriol, pregnanediol and maternal serum alpha-feto protein. Nowadays the incidence of undiagnosed twins has reduced because ultrasound has become part of the routine antenatal follow-up.

Since hospital admission disrupts normal family life and is expensive it is recommended that patients should only be hospitalized for the same indications that would be used to admit women with singletons. In our set up patients with twin pregnancy are not routinely admitted for bed rest. The patient was managed in this manner, although the twins were undiagnosed till second stage of labour.

Prophylactic tocolytics and cervical cerclage placement have not shown any significant reductions in preterm delivery rate and as such are only recommended to be used when indicated.

Our patient was not given tocolytics ante natally neither did she have cervical cerclage placement since there was no indication. In the unfortunate event of premature rupture of membranes, management is conservative just like in singleton pregnancies. However spontaneous labour seems to occur earlier than in singleton pregnancies [4]. Management of vertex-non-vertex category is controversial. Poor Apgar scores of the secondtwin have necessitated caesarean delivery of this category of twin combinations in some centres. Some other studies have not shown any difference in outcome between vaginal delivery and caesarean delivery of the second twin. Because of documented ill effects of vaginal delivery on low birth weight singleton breech infants delivered vaginally and since there is no evidence that being a second twin gives these infants an advantage relative to their singleton counterparts vaginal breech delivery is not recommended for second twins weighing less that 1500g. However if the second twin weighs between 1500 and 3500g and the criteria for vaginal breech delivery are met vaginal breech delivery is an acceptable option Section is also recommended if either twin shows signs of persistcompromise probable twin-twin transfusion syndrome (gross disparity in fetal size) and nearly all triplets or quadruplets. In vaginal delivery the interval between the delivery of the first and second twin is 10-30 minutes. Oyieke (1978) in his series found out that a delivery interval of less than 10 minutes was associated with a perinatal mortality of 4.9% for the second twin but this increased to 54% with the interval was more than 45

Active management of the third stage of labor by administration of intravenous ergometrine, or oxytocin at the delivery of the anterior shoulder of the second twin is recommended to avert post partum hemorrhage.[2]

In cases requiring induction or augmentation with syntocinon, management is individualized. In cases of termination of pregnancy for one reason or another e.g. in preclampsia, oxytocin administration is indicated if the presenting part is well fixed in the pelvis and the cervix is dilated somewhat. However it requires careful monitoring [3]

There is a high incidence of congenital malformations especially of the cardiovascular and central nervous systems among twins. Some congenital abnormalities include conjoined twin, acardiac twin. Intrauterine death of one twin with continuation of the pregnancy is a rare complication of multiple pregnancy. It is associated with increased morbidity and mortality of the surviving twin especially in monochorionic twin pregnancies. Maternal consumption coagulopathy with hypofibrinogenaemia may occasionally develop leading to troublesome bleeding . In the case presented, the first breech twin was a macerated still-birth, while the second twin was alive and scored well.

Multiple gestation is demanding to the obstetrician, the mother, the pediatrician and to society. Special care both medically and socially has to be taken from the time of diagnosis.

REFERENCES.

1. Ian Macgillivray. Twin pregnancy. Scientific foundations of Obstetrics and Gynaecology. 3d Ed Heinmann medical books international edition, 1997; 248253.

2. Usha Chitkara and Richard L. Berkowitz. Multiple Gestations. Obstetrics –Normal and problem pregnancies. 4th Ed. Churchill Livingston 2002: 827-861.

3. Martin L.Pernoll and Ralph C.B. Multiplexregnancy. Current Obstetric and Gynaecologic Diagnosis and Treatment. 9 Ed Appleton and Lange 1999: 357366.

4. Cunningham F.G., Norman F.G., Kenneth J.L. et al. Multifetal pregnancy. Williams Obstetrics 21' Ed. McGraveHill 2001; 765-804.

5. Lewis T.L.T., Chamberlain G.V.P. Multiple pregnancy. Obstetrics by Ten Teachers. 15' Ed ELBS 1990: 136-141.

6. Oyieke J.B.O. A 2V2year review of some aspects of twin delivery at Kenyatta National Hospital. M.Med Thesis UoN 1978.

7. Mutungi A.K. A prospective study of twins at Kenyatta. National Hospital - Antenatal care and delivery. M.Med Thesis U.O.N. 1990.

8. Hawrylyshyn P.A., Barkin M, Berristein A., Papsin F.R. Twin pregnancy – a continuing perinatal challenge. Obstet Gynaecol, 1992; 59:463

9. Maclennan A.H., Green R.C., O'Shea R et al. Routine hospital admission in twin pregnancy between 26-30 weeks gestation. Lancet 1990; 335:267

10. Saunders M.C., Dick J.S., Brown I.M. The effect of hospital admission for bed rest on the duration of twin pregnancy: A randomized trial. Lancet 1985; 2:793

11. Katz M, Robertson P.A., Creay R.K. Cardiovascular complications with terbutaline treatment for preterm labor. Am J. Obstet Gynecol, 1991: 139: 605

12. Khunda S. Locked twins. Obstet Gynaecol 1972: 39; 453

13. Duenhooelter J.H., Wells C.E., Reisch J.S. A paired controlled study of vaginal and abdominal delivery of the low birth weight breech fetus. Obstet Gynecol 1979; 54: 310

14. Goldenberg R.L., Nelson K.G. The premature breech. Am. J. Obstet Gynaecol, 1987: 127:240.

OBSTETRIC CASE NO 11

HIV IN PREGNANCY – ELECTIVE CAESAREAN SECTION – LIVE BABY Name:

Name;J. M. **Age:** 24 years

IP.NO: 1063180 DOA: 3/5/06 **DOD:** 8/5/06

24 years old Para 0 + 2 G 3 at GBD 38/40, LMP 9/8/06, EDD 16/5/06 admitted for elective C/S.

HISTORY OF PRESENTING ILLNESS

Patient had been found to be HIV positive during the routine ante natal follow up with a CD4 count of 400. She had been counseled then started on AZT 300mg BD since the 34th week of pregnancy. She had opted for an elective C/S at term. She was going to feed the baby on formula feeds. She did not have any complaints at the time of admission.

PAST MEDICAL HISTORY. Not significant

OBS/GYN HISTORY

She had attained menarche at the age of 15 years. Her cycles were 3/28 regular, and of normal flow, there was no history of dysmenorrhoea. She had not used any method of family planning.

Para 0+2, LMP 9/8/05, EDD - 16/5/06

1st abortion - 2004 March 3/12 - MVA not done

2nd abortion in September 2004 at 2/12 MVA done.

ANTENATAL HISTORY Started at KNH at GBD of 19/40, had made 9 visits. Profile done: Blood group 0 Rhesus positive, VDRL – Negative, P24 - Reactive, Hb -13.2g% (PCV - 37.8g%), AZT started at 34/40, Height 4.45ft

FAMILY SOCIAL HISTORY

She is single, a business lady, Form IV graduate. She does not smoking or take alcohol. There is no history of chronic illness in the family. She stays at Ruai with her mother.

The baby was put on Nevirapine syrup 5.2mg stat and was to continue with AZT syrup 10.4mg twice daily for the next ten days.

On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet. She was also taught how to reconstitute the feeds for her baby. Post-operatively patient did well and was discharged home on 4^{t} post operative day.

Follow up

She was reviewed in the post-natal clinic after 6 weeks, and was found to have recovered completely. She was referred to the family planning clinic and patient support centre for further counseling, repeat CD_4 counts, viral load and possibly HAART.

DISCUSSION

The patient presented was a 24 year old Para 0+2 G3 at 38 weeks gestation with HIV disease and bronchospasms, who underwent elective C/S for PMTCT. She gave birth to a life female infant who weighed 2600gms with a good Apgar score and was given Nevirapine .

Acquired Immunodeficiency Syndrome (AIDS) was first described in 1981, and a viral etiologic agent demonstrated in 1985. The HIV viruses 1 and 2 have since been known to be the cause of AIDS. HIV infection continues to increase rapidly in the developing world, especially in Africa and Asia[3], and is now the leading cause of death among urban women aged 20-40 years[2]. In East Africa, the seroprevalence of HIV infection in screened pregnant women is reported to be as high as 20-32%[1]. A higher value of up to 41% has been reported in South Africa. Antenatal screening for HIV is presently being advocated for all pregnant women after informed consent, as this provides the opportunity to reduce risk of transmission and offer prophylactic treatment . Our patient benefited from voluntary counselling and testing (V.C.T) antenatal screening, and was put on prophylaxis. At KNH overall acceptability of screening was reported to be 99.4% [4]. ELISA test is the commonly used method of screening for HIV and this was used in our patient. Other diagnostic tests include Western Blot, viral cultures and PCR test. These are important in assessing the severity of the disease and the viral load quantification, both important factors in transmission of the disease to the fetus. CD4 counts and viral loads are used for surveillance. Our patient however had a CD4 count of 400.

Pregnancy may increase the progression to symptomatic infection by accelerating the depletion of helper T lymphocytes and the resulting immunodeficiency [15]. Increased rates of abortion and prematurity have been reported in infected women [1,7] No increase in the congenital abnormalities has been noted in the HIV infected women in the general population [1]. Our patient progressed to term pregnancy and the baby had no anomalies.

The risk of MTCT ranges from 15-30%, with the lowest rate in Europe and the highest is in Africa [15].

Transmission depends on many factors [1, 3, 14]:

i) viral load
ii) biologic /genetic variation of HIV
iii) presence of neutralizing antibody
iv) presence of chorioamnionitis or other STD's
v) mode of delivery
vi) breast-feeding.

Vertical transmission can occur in utero, during labor or delivery and from breastfeeding. It is thought that probably about 70% of transmission occurs in late pregnancy and labor [14]

Breastfeeding carries an additional risk of transmission, with rates of 15-20% in Europe, 15-30% in USA and 25-35% in Africa [1,3,8].

Reduction of. vertical transmission is of primary concern in infected women and is inclusive of [9,11,12,14,15]

I) treatment of other STD's
II) anti-retroviral therapy (e.g AZT) for the mother during pregnancy/delivery and for the infant after birth
III) reduction in peri-partum exposure e.g delivery by C/S for HIV positive women. The European Collaborative study suggested a 50% reduction with this policy.
IV) Avoidance of intra-partum invasive procedures e.g fetal scalp electrode or blood sampling
V) Vaginal cleansing with 0.25% chlorhexidine during labor, and repeated 4 hourly with infant washing after birth
VI) Avoidance of breastfeeding wherever possible- in developing countries, the risk of not breastfeeding may be the greater
VII) Passive immunotherapy for the baby and/or mother is being evaluated, and active immunization is undergoing preliminary studies in the USA.

VIII) Our client was given nevirapine prior to elective caesarian section, and the baby was also given the same as well after birth. All these combined; significantly reduce the chances of MTCT. Studies have shown that C/S delivery combined with the three part AZT prophylaxis reduces transmission rates by about 85% comp aired with other modes of delivery [11,12]

IX) Prevention and treatment of HIV in women and infants is priority public health concern. Policies to strengthen access to prenatal care, voluntary counselling and testing for HIV infection and provision of anti-retroviral therapy for HIV infected pregnant women should be emphasized.

REFERENCES

1. Johnstone F.P. HIV and pregnancy; Br J Obstet Gynecol, 2001; 103(12): 1184

2. Wilkinson D. Continued explosive rise in HIV prevalence among Pregnant women in rural South Africa. AIDS, 1999, 13:740

3. McIntyre J. Transmission of HIV from mother to child: Strategies for prevention. Postgraduate doctor, Africa 2002; 19(1):4-6

4. Kiarie J.N. The acceptability of perinatal HIV screening; MMed Thesis, UON 1996; 16:158

5. Catoux M. Acceptability of voluntary HIV testing by pregnant women in developing countries; an international survey. AIDS, 1998, 12: 2489

6. Minkoff H. Pregnancy resulting in infants with AIDS or AIDS-related complex Obstet Gynecol, 1997,69:287

7. Lopita I.The prevalence of infection with HIV in patients being treated for spontaneous abortions at KNH. Med Thesis, UON 1992: 16;146.

8. Southern S.O. Milk borne transmission of HIV; J Hum Virol, 1998: 1; 328.

9. Connor E. Paedatrics AIDS Clinical Trials Group Protocol 076 Study Group. Reduction of maternal-infant Transmission of HIV 1 with AZT treatment. N Eng J Med 1994 331:1173-1180

10. Shaffer N. Short course AZT for perinatal HIV-1 transmission In Bangkok Thailand. An

RCT; Lancet, 2000; 353: 773.

11. The International Perinatal HIV Group. Mode of delivery and vertical transmission of HIV-1: A meta-analysis from 15 prospective studies. N Eng J Med, 1999, 340: 977

12. European Collaborative Study. Caesarian Section and risk of vertical transmission of HIV-1 Infection.2002; 68-79

13. John G.C., Nduati R.W., et al. Genital shedding of HIV-1 DNA during pregnancy: Association with immunosupression, abnormal cervical or vaginal discharge, and severe vitamin A deficiency. J Infect Dis, 1997, 175:57

14. Gordon M.S. HIV in pregnancy : Aids to Obstetrics and Gynaecology, 5h ed.Churchill Livingstone. 2001: 12:694

15. Anderson J. A guide to the clinical care of women with HIV. 2002 preliminary edition. HIV and reproduction. 2002; 7:21-48

OBSTETRIC CASE NO 12

PRE-ECLAMPSIA AT TERM - INDUCTION OF LABOUR WITH GOOD OUTCOME

NAME: D. A. **AGE:** 28years

IP NO: 1011105 **DOA:** 10/8/06 **DOD:** 19/8/06

28 year old Para 3 + 0 G4, LMP 24/11/05 EDD 1/9/06 GBD 37/40, admitted as a referral from a private clinic (Immaculate clinic, Kasarani) with

C/O

Low abdominal pains x 1/7

Per vaginal discharge x 2/7

HISTORY OF PRESENTING ILLNESS

The low abdominal pains were not intermittent and were non progressive. The vaginal discharge was white in colour, curdy in appearance and associated with vulval itchiness. There were no associated urinary symptoms or history of fever, headache or joint pains. At the time of referral she had an elevated blood pressure of 160/115 mmHg. There was no associated history of blurring of vision, dizziness or epigastric pains. She gave history of having had increasing blood pressure in all her previous pregnancies which usually settled after delivery. During her previous pregnancy, she had been having an elevated blood pressure since a gestation of 20 weeks for which had been controlled on Aldomet and Phenobarbitone but resolved completely after delivery.

OBS/GYN HISTORY

She attained menarche at 14 years, her menstrual cycle lasts 28 days and has a flow of 3 days.
Para 3 + 0 G4

1998 - SVD at term, Male infant birth weight 3.5 kg, alive and well. 1999 - SVD at term. Female infant birth weight 2.5kg, alive and well. 2004 – SVD at term, Female infant birth weight 2.4kg - died after one week as she had been borne with foetal distress/IUGR.

She is married and works as a tailor. Her husband is a mechanic. She does not take alcohol or smoke. There is no history of chronic illness in the family.

On examination, she was in fair general condition, not pale, cyanosed, jaundiced or oedematous.

Vital signs: BP 1501105 mmHg, PR 82/min, RR 20/min

CNS/CVS/RS: Essentially normal

PA: Fundal height was term, longitudinal lie, cephalic presentation, head 5/5 up. Foetal heart was 132/min, regular.

V/E: She had normal external genitalia, cervix was 2 cm long, posterior and closed. There was a white curdy discharge on the examining finger. Urinalysis showed a proteinuria of 1+

An impression of Pre- eclampsia with vulvo-vaginal candidiasis at term was made.

She was admitted, put on bed rest, clotrimazole pessaries one nocte for six consecutive days, Aldomet 500mgs and phenobarbitone 30 mgs 8 hourly. Blood was taken for full haemogram, UEC, LFTS, Uric acid and do an obstetric ultrasound scan She was to undergo daily urinalysis while in the ward. She was also put on a foetal kick chart.

11/8/06. She had a BP of 140/100 mmHg and a trace of protein in her urine. The foetal heart rate was normal.

Obstetric ultrasound scan showed a single viable infra uterine pregnancy at 35 weeks gestation in cephalic presentation, with adequate liquor amount. The placenta was posterior and not low lying.

The patient was started on intramuscular Dexamethasone 12mg 12 hourly for two doses for lung maturation. She was also given a prescription to buy Prostin E2 pessaries for induction of labour.

Full haemogram: 12/08/06 Hb - 10.7g%, "C 7.2 x 10^{9}/L Platelets 214 x 10 9/L.

Urea 3.8mmol/I (1.7 - 8.3), Creatinine- 84mm/1 (60-120)

The prostin pessaries were now available, so one was inserted in to the posterior fornix at loam with the second one being inserted at 4pm She had spontaneous rupture of the membranes while in the ward at 10.30p.m.Review showed that the foetal heart was 142/ min, head was 4/5 up. On V/E, there was normal external genitalia, cervix was central, 2-3 cm dilated, partially effaced, membranes absent, draining clear liquor. There was no cord felt. An impression of established active labour was made, she was put on intramuscular Buscopan 40mg stat and Tramal 100mg stat, Oxytocin drip 2.5U in 500mls 5% and started on the partogram. She progressed on well and was noted to have the urge to bear down at 1.15am on 19/08/06. At 130am she delivered SVD a live male infant who had an Apgar score of 7/1, 10/5, 10/10 birth weight of 2700gm.

Review on 20/8/06 while in the post natal ward found that she had a BP ofl26/80mmHg, uterus was well contracted with minimal lochia loss and no calf tenderness. She was discharged home on Aldomet 500mgs and Phenobarbitone 30 mgs 8 hourly . She was to be reviewed in the post natal clinic after two weeks.

DISCUSSION

Pre eclampsia can be classified into mild or severe. Severe pre eclampsia is characterised by blood pressure above 160 systolic or I I OmmHg diastolic, recorded on two occasions at least 6 hours apart, proteinuria exceeding 3g in 24 hours or 3-4+ protein on unstick testing and oliguria (<400mls in 24 hours). Others are cerebral or visual disturbances, epigastric pain, pulmonary edema or cyanosis, impaired liver functions and thrombocytopenia [3].The patient presented had high blood pressure spike of 150/105 MmHg, and proteinuria of I+. She did not present with other symptoms.

Management of patient is aimed at prevention of eclampsia, delivery within a reasonable gestation, recognition and treatment of associated complications and minimise maternal and fetal morbidity. Pre-pregnant counseling is rarely undertaken since it's a disease of primigravidas. Pre-pregnant diabetes and hypertension control should be undertaken where possible[2]. Low dose aspirin, calcium and other dietary supplements like zinc and magnesium appear to be effective

in the prevention of pre-eclampsia It has been postulated that calcium deficiency predisposes to pre-eclampsia . Provision of vitamin E may reduce the onset of pre-eclampsia. It's an antioxidant, thus may help prevent the formation of free radicals, which could initiate endothelial or other forms of tissue damage.[9]

Patient who presents at term or near term with pre-eclampsia should be delivered, but if remote from term, hospital admission should be undertaken [2]. Our patient was at 37 weeks gestation and was thus admitted to expedite delivery.. Fluid and sodium restriction have not been shown to improve pregnancy outcome [2]. Aspirin administration after diagnosis of pre-eclampsia has not been shown to be beneficial ". Anti-hypertensives should be given to reduce maternal risks but they do not affect progression of the disease. The patient should have serial 24-hour urine for proteinuria and creatinine clearance. Platelet count, liver function tests and serum fibrinogen, fetal surveillance b fetal kick chart, fetal heart rate and serial ultra-sounds should be undertaken. Where preterm delivery is necessitated steroids to enhance fetal surfactant production should be administered [1,2,1]

Severe pre-eclampsia is associated with many maternal complications among them is abruptio placenta, thombocytopenia, HELLP syndrome, eclampsia, renal failure, liver function abnormalities and rarely hepatic capsule rupture may occur. Pulmonary oedema may occur in 2% of pre eclampsia [1,2,3].

Fetal complications may also result, preterm delivery, uteroplacental insufficiency, abruptio placenta, unexplained fetal death, intrauterine growth retardation, and reduced amniotic fluid.

REFERENCES

1. Mabie B., Sibai B.M. Hypertensive Status of pregnancy. Current Obstetric and Gynaecology Diagnosis and Treatment. 9th Edition Appleton and Lange: 1998:340

2. Cunningham F.G., McDonald P.C., Gant P.C., Leveno K.J. Hypertensive disorders in Pregnancy . Williams Obstetricts 21St Edition. Appleton and Lange: 2001:794.

3. Redman C.W.G. Hypertension in Pregnancy. Turnbulls Obstetrics. 3d Edition Churchill Livingstone: 1999: 503-523

4. Mati J.K.G. Studies on Pregnancy Hypertension in Kenya; Some aspects of Epidemiology, Prognosis, Differential Diagnosis and Aetiology. MMed thesis UON, 1975

5. Kaburu J.G. Outcome of pregnancy in patients with Hypertensive Disease. MMed Thesis UON, 1992

6. Belizam J.M., Villar J. The Relationship between Calcium Intake and Oedema, Proteinuria and Hypertension. A.J. Clin Nutri 1980; 33; 2202-10.

7. Belizam J.M., Villar J. Calcium Supplementation to prevent Hypertensive disorders of pregnancy. New Engl. J.med 1991; 325: 1399-1405

8. Itanth J.C., Goldenberg R., Parker C, et al. Low Dose Aspirin Therapy to Prevent Pre-eclampsia. A.J. Obstet Gynaecol 1993; 168: 1083-1093

9. Habel C., Robert J.,Taylor R. Lipid Peroxidation in Pregnancy; new Perspectives on Pre-eclampsia A.J. Obstet gynaecol 1989; 161: 1025-1034

OBSTETRIC CASE 13

GESTATIONAL DIABETES MELLITUS -ELECTIVE PRETERM DELIVERY BY CESAREAN SECTION

Name:	F.W.	IP No:	0709225
Age:	31 yrs	DOA:	16/6/04
Parity:	2 +0 G 3	DOD:	17/7/04

PRESENTING COMPLAINTS

F.W. was admitted via the antenatal clinic because of elevated random blood sugar (11.6 mmol/L) after clinical suspicion of diabetes mellitus because of glycosuria (3+) and Bad Obstetric History (BOH).

OBSTETRICS AND GYNAECOLOGY HISTORY

- F.W was now Para 2 + 0 G3, L.M.P. of 08/11/03, so the E.D.D was 13/08/04, and the Gestation by dates 33+ weeks, with no living child. She had one previous scar of 2002. She also had chronic hypertension diagnosed in the year 2001.
- Her first delivery was induced at about 8 months because of intra-uterine fetal death in the year 2000. The outcome was a fresh stillbirth weighing 1900gms. No cause was identified for the fetal demise. Her 2"° delivery in 2002 was by caesarean section at about 32 weeks because of reduced fetal activity. The resulting life male infant, weighing 1.5 kg, died within the day of delivery because of respiratory distress. She had both deliveries at the KNH.
- She started antenatal clinic early, in the current pregnancy, at bout 12 weeks gestation. Her antenatal profile was as follows;
    - i) HB 12.8gidl – Normal RBC indices.
    - ii) Blood group 0 Rhesus positive
    - iii) VDRL and ELISA both non reactive
    - iv) Renal function tests: Normal urea, electrolytes and creatinine.
    - v) Her initial random blood sugar was 7.2mmol/L.

- Her antenatal period had been unremarkable. However, on the day of admission a urine dipstick showed plus 3(3+) glucose and a random blood sugar requested was elevated 11.6 mmol/L. She was therefore admitted for investigations and management.
- Her menarche was at 15 years, menses were regular, not painful, lasting between 3-5 days, and occurring at an interval of 22-28 days. She had never used any contraceptives methods.

PAST MEDICAL HISTORY

She was diagnosed to have chronic hypertension in 2001. She was well controlled on Aldomet 500mg three times daily. She had never been hospitalized for non- pregnancy related illness in the past.

FAMILY AND SOCIAL HISTORY

She stayed with her husband in upper hill Nairobi. She was a laboratory technician, working with the ministry of health. She did not smoke cigarettes or drink alcohol. There was no family history of diabetes mellitus or twinning. Her father was on treatment for hypertension. Her brothers and sisters were all alive and well.

EXAMINATION

She was in good general condition, well nourished, not obese. She was not pale, did not have jaundice, edema, or lymphadenopathy.

She weighted 72 Kgs. Blood pressure 140/90 mmhg pulse 80/min, respiratory rate 18/min and temperature of 36.6°c.

SYSTEMIC EXAMINATION

The respiratory and cardiovascular systems were examined and found to be normal.

The central Nervous system was also normal.

ABDOMINAL EXAMINATION

The fundal height was 32 weeks, longitudinal lie, and cephalic presentation. The fetal heart was 138 beats per minute and regular. The head was 5/5. She had a midline sub-umbilical scar.

WORKING DIAGNOSIS

A working diagnosis of Gestational Diabetes mellitus was entertained and the following investigations geared towards confirming the diagnosis and planning management were ordered: Fasting blood sugar, oral glucose tolerance test (OGTT), Glycosylated hemoglobin and an obstetric scan. Results were as follows: -

i) Fasting blood sugar 7.8 mmol/L (above normal)

ii) 3 hour 100g glucose tolerance test

- At 1 hour = 11.2 mmol/L (above normal)
- At 2 hrs = 8.5 mml/L
- At 3 hrs = 7.8 mmol/L

iii) Obstetric scan – showed a single intra-uterine pregnancy with a Biophysical profile score of 8 in 8. The fetus corresponded to 33 weeks by BPD, FL and AC. The placenta was fundal posterior and estimated fetal weight 2.2 kgs. No fetal anomalies were detected. Umbilical artery blood flow was also normal.

iv) HBA 1c 6%.

Based on the above results a diagnosis of Gestational Diabetes mellitus at 33 weeks was confirmed.

Treatment plan

F.W was started on subcutaneous regular insulin 8 i.0 8 hourly. Serial blood sugars were done early in the morning (fasting, 6.00 am), at 3.00 p.m. and 9.00 p.m. daily. The insulin was adjusted according to the blood sugar levels, and good control of between 3.8 – 5.8 mmol/I achieved with 12 i.0 of S.C. insulin 8 hourly. The dietician also gave her dietary advice.

Fetal well-being was monitored by means of a fetal kick chart and weekly Biophysical profiles planned. She was however unable to do weekly scans because of the cost. The fetal kick chart was satisfactory as was the blood sugar control. Because of previous unexplained intra-uterine death, at 35 weeks a

decision to enhance fetal lung maturity with Dexamethasone and delivery at 36 weeks was made. No surfactant test was done.

At 36 weeks, she underwent an elective caesarean section, and delivered a life female infant 2550grams, with APGAR scores of 7 in one minute, 8 at 5 minutes and 10 in 10 minutes. The baby had no congenital malformations, and did not develop any complications in the early neonatal period. The mother did well postoperatively with no evidence of puerperal sepsis. After delivery blood sugars fell dramatically to below 5.Ommol/l, and she was discharged on the 5th postoperative day for follow up in the post-natal clinic in 6 weeks.

FOLLOW UP

At 6 weeks, a fasting blood sugar was found to be 4.6 mmol/L and thus normal. She opted for barrier methods of contraception for the time being, so that she can achieve her desired family size. Both mother and baby being well, they were discharged from the clinic.

DISCUSSION

Presented is a 31-year-old Para 2 + 0 G3 with no living child with mild chronic hypertension and gestational diabetes mellitus. She was managed with insulin and delivered preterm by elective caesarean section with a good maternal and neonatal outcome.

Diabetes mellitus is a clinical syndrome characterized by lack of, or insensitivity to insulin (1). It is the most common medical complication of pregnancy in the United States. Patients can be separated into those who were known to have diabetes prior to pregnancy (overt or pre-gestational), and those diagnosed during pregnancy (Gestational). Gestational diabetes mellitus, accounts for close to 90% of found all cases of diabetes in pregnancy, and has been found to complicate 4% of all pregnancies, in the United States. Pre-existing diabetes affects approximately 1-3 pregnancies per 1000 births (2).

The modified white's classification is still widely used to classify pre-gestational diabetes occurring in pregnancy. This classification emphasizes that end-organ derangements especially involving the eyes, kidneys and heart have significant effects on pregnancy outcome. The following table depicts this classification.

Whites classification of diabetes in pregnancy description

Class	Description
Cass A	Chemical diabetes diagnosed before pregnancy; managed by diet alone; any age of onset or duration.
Class B	Insulin treatment necessary before pregnancy; onset after age 20 years; duration less than 10 years.
Class C	Onset before age 10-19 years; or duration of 10-19 years.
Class D	Onset before age 10 years; or duration of 20 or more years; or chronic hypertension; or background retinopathy.
Class F	Renal disease
Class H	Coronary artery disease
Class R	Proliferative retinopathy.
Class T	Renal Transplant

However, for most clinical purposes diabetic women in pregnancy may be divided into 3 classes,

Class I Non insulin requiring glucose intolerance responsive to dietary .

Class II Insulin requiring glucose intolerance without associated vasculopathy.

Class III Insulin requiring glucose intolerance with associated vasculopathy.

Our patient had gestational diabetes mellitus without apparent vasculopathy. She therefore fell into class I of the general classification of diabetes in pregnancy, the white's classification being non applicable to her condition.

Pregnancy is a diabetogenic condition. This is because of insulin antagonism by the action of human placental lactogen (HPL), oestrogen and progesterone. Placental insulinase may also contribute, by accelerating insulin degradation. Other insulin antagonists like cortisone are also elevated in pregnancy. As a result of these changes, pregnancy may unmask previously undiagnosed diabetes mellitus. In addition some complications of pregnancy like nausea and

vomiting and increased incidence of urinary tract infections make control difficult. Indeed the pregnancy-associated switch in fuels from glucose to lipids predisposes pregnant diabetics to Diabetic Ketoacidosis (DKA).

Diabetes in pregnancy is associated with significant maternal, fetal and neonatal risks. The risks

to the mother and foetus are related with the degree of control and the presence of cardiovascular and renal complications. The presence of other conditions like hypertensive disease further complicates the outlook. Our patient was well controlled when the diagnosis was made and had no evidence of end-organ derangements. Her hypertension was mild and well controlled. Among the maternal risks is the difficult associated with blood sugar control, hence likelihood of DKA and hypoglycaemic episodes, increased incidence of operative delivery due to macrosomia; increased incidence of infection, especially urinary tract infections; increased incidence of preterm labour; increased incidence of preterm labour; increased risk of hypertensive disease in pregnancy and exaggerated pressure symptoms of pregnancy due to macrosomia and hydramnios. Fetal risks include: congenital anomalies; macrosomia leading to birth trauma and late unexplained fetal demise. Neonatal morbidities include respiratory distress syndrome neonatal hypoglycemia, hypocalcemia, polycythemia, hyperbilirubinemia and future developmental delay. Our patient and her baby did not develop any of these complications.

Gestational diabetes mellitus (GDM), i.e. diabetes which first appears and that which is 1st recognized in pregnancy is diagnosed based on the original work of 0' Sullivan and Mahan and modified by carpenter and Coustan (3). Risk assessment is undertaken in the first visit and those thought to be at high risk have a glucose tolerance test (OGTT) as soon as possible. For those not at high risk, a screening 50g oral glucose load without regard to meals or time of the day may be undertaken. If the results are unequivocal, then a 100g oral 3 hours OGTT is done in the USA. The WHO recommends a 75 g oral glucose load. Plasma glucose levels suggested in 1982 (carpenter and Coustan) were a fasting level > 95 mg/dl; 1 Hour values > 180mg/dl-, 2 hour values > 155mg/dl and 3 hour values > 140mg/dl.

REFERENCES.

1. F. Gary Cunningham, Norman F. Gant,. Diabetes In: Williams Obstetrics, chapter 51, Pg 1359-1381. 21st edition. McGraw - Hill companies, 2001
2. Carla Janzen, Jeffrey S. Greenspoon, & Sue Palmer. Diabetes in pregnancy. In: Current Obstetric & Gynecologic Diagnosis & Treatment. Chapter 18, Pg 326337. 9th edition. McGraw Hill companies, 2003.
3. Gerald N. Burrow, Thomas P. Duffy: Medical Complications in pregnancy. 5th (Eds). 1999. W.B. Saunders Company.
4. American College of Obstetricians and Gynaecologists. Diabetes and pregnancy, Technical Bulletin, 1994: No. 200.
5. Lidegard, 0: Oral Contraceptives, Pregnancy and risk Of Thromboembolism. Br. J. Obstet. Gynaecol. 1995; 102-152.

OBSTETRICS CASE 14

UNSENSITIZED RHESUS NEGATIVE MOTHER-NORMAL VAGINAL DELIVERY

NAME:	J. W.	IP NO.: 0973131	WD 1A
AGE:	19 YEARS	DO A 13/7/04	
PARITY	0 + 0 G 1	DOD 15/7/04	

PRESENTING COMPLAINTS

G.W. was admitted to labour ward via the antenatal clinic KNH, for induction of labour because of her Rhesus negative state at term.

HISTORY OF CURRENT PREGNANCY

G.W. was Para 0 + 0 G1 at admission. Her L.M.P. was on 3.10.03, so her expected date of delivery was 10.7.04. The gestation by dates at admission was 40+ weeks. She had started attending antenatal clinic at KNH, in February 2004. She had received 2 doses of tetanus toxoid and her antenatal period had been uneventful. Her antenatal profile was as follows:

Haemoglobin level – 11.5 g/dl

VDRL - non reactive

ELISA - non reactive

Blood group - A rhesus negative

Because of her Rhesus negative state, an indirect combs tests (ICT) done at about 26 weeks was negative. She was not given anti–D human immoglobulin in the antenatal period. A repeat ICT at 36 weeks was again negative. She was advised of the need for protection against sensitisation at delivery and given a prescription for anti–D, 300μg to come with when in labour or at her E.D.D., on 10.7.04. She was however unable to come on 10th because of personal commitments. She came on 13th for induction of labour.

GYNAECOLOGICAL HISTORY

She attained her menarche at 14 years and had regular cycles, with menses lasting 3-6 days and occurring at an interval of between 28-30 days. She had occasional

dysmenorrhea, relieved by analgesics. She had used oral contraceptives before the current pregnant for a period of 3 years. She had never had a sexually transmitted disease. She had never had a pap smear done.

PAST MEDICAL HISTORY

She had never been hospitalised before.

FAMILY AND SOCIAL HISTORY

She was a married business lady who lived with her husband in upper hill. Her husband was a police officer attached to the traffic headquarters. She was the 2nd born in a family of 4 siblings and all her family members were alive and well. She dropped from school in form 2 to get married. There was no family history of chronic ailments. There was no history of twinning in the family.

SYSTEMIC ENQUIRY

Non-revealing

EXAMINATION

She was in good general condition and well nourished. She was not pale and did not have jaundice, oedema or lymphadenopathy. Her vital signs were normal, with a blood pressure of 110170 mm Hg, pulse of 80 per minute, respiratory rate of 18 per minute and temperature of 36.2°C.

ABDOMINAL EXAMINATION

Abdominal was uniformly distended and moving with respiratory. No surgical or therapeutic scars were present. The abdomen was non-tender. The fundal height was term, longitudinal lie with cephalic presentation. The head was 4/5. The fetal heart rate was 140 beats per minute.

SYSTEMIC EXAMINATION

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

PELVIC EXAMINATION

The external genitalia were normal. The cervix was posterior, and closed. It was of moderate consistency and about 1.5-2cm long. There was some candidiasis discharge on the examining finger. The total bishop score was 3, and unfavourable.

DIAGNOSIS

A diagnosis of unsensitized Rhesus negative mother at term was made, with a poor bishop score. A decision to induce labour was made.

MANAGEMENT

A prostaglandin E2 pessary was inserted into the posterior fornix in the afternoon of 13/7/04. She was subsequently advised to rest in bed. About 6 hours later, when due for review, the cervix, was noted to noted to be central, 1-2cm dilated, soft consistency and about 0.5cm long. Artificial rapture of membranes (ARM) was done with controlled liquor drainage. The liquor was clear and no cord was felt on subsequent vaginal examination. She was started on 5 W. of Syntocinon in 500ml of 5% dextrose, initially to run at 10 drops per minute and to be increased by 10 drops every 30 minutes to a maximum of 60 drops per minute or 3 strong contractions lasting > 40 seconds. She was for review every 4 hours or as need arose.

About 4 hours later, she had picked up contractions and was having 2-3 contractions lasting 20-30 seconds in 10 minutes. The cervix was now 4 cm dilated. The fetal heart rate remained regular at 138 beats per minute. Now that she was in active labour a partograph was started and the induction continued. No abnormalities were detected on the next review. About 5 hours later she reported an urge to bear down. On vaginal examination, the cervix was found to be fully dilated. The head was 1/5. Half an hour later, she progressed to second stage and delivered a live female infant, weighing 3.0 kg and with Apgar scores of 9 in one minute and 10 in 5 minutes. At delivery the cord was double clamped, and cord blood taken for ABO blood group and Rhesus group typing: Haemoglobin level; bilirubin level and direct Coombs test. The neonate was taken to the newborn unit for observation.

The baby's blood group was found to be B-rhesus positive, haemoglobin 15.5g/di and bilirubin level 0.6mg/di. The baby was discharged to stay with the mother after 24 hours. The mother received an intramuscular injection of 300 µg (microgram) of anti-D human immunoglobulin on

her 2nd postnatal day and was allowed home a day later for follow-up in the post-natal clinic at 6 weeks. She did not turn up for the post-natal clinic.

DISCUSSION

Presented is a 25 years old unsensitized Rhesus D negative mother who had induction of labour at term and delivered vaginally. The baby was blood group B Rhesus positive, and the mother received 300[tg (microgram) of anti-D 24 hours after delivery. Though her Rhesus D negative state was diagnosed in the antenatal period, no prophylaxis was given.

A foetus receives half of its genetic components from its mother and half how its father, and may therefore have different blood groups than those of its mother. These antigens reside on red blood cells. The red blood cells have over 400 recognised antigenic factors of which the most important are the ABO, Rhesus, Kell, Duffy, Kidd, Lutheran, P., and M.N.S. Landsteiner and Weiner described the Rhesus factors on human erythrocytes in 1940. The Rhesus antigens are inherited independently of all other blood group antigens. The Rhesus factor is a complex antigen consisting of 3 pairs of alleles occupying a specific locus on the chromosome; Cc, Dd, Ee. The d antigen has however never been isolated, and its existence is doubtful. However, only the D antigen is highly immunogenic to assume clinical significance by causing isoimmunization (1). An individual lacking the Rh factor (Rh-negative) may carry a rhesus positive foetus.

There is considerable racial variation in the incidence of Rhesus-negative individuals in a population. The highest reported incidence is among the Basques, at between 3035%. Among the North American Caucasians it is about 15%, while among the American African it is between 7-8%. The incidence among Asian groups is below 1% and virtually non-existent among the mongoloid races (2,3,4). In Nairobi, Kenya among

mothers attending antenatal clinics, a rate of 5% has been reported(s). At the Kenyatta National Hospital (KNH) a rate of 4.1 % was found (6).

Rhesus isoimmunization not only results when a Rhesus negative mother carries a Rhesus

positive pregnancy but also from heterogous blood transfusion. The latter is the most common cause of sensitisation due to the less common antigens. Although the mother could be sensitised if enough erythrocytes from the foetus were to reach her circulation to elicit an immune response, this occurs infrequently. Among the mitigating factors against Rhesus isoimmunization are:

1. Insufficient transplacental passage of antigen and or antibody. 0.25ml of fetal red blood cells represents a critical volume, with severity of sensitisation increasing with greater volumes.
2. Variability of maternal immune response to antigen. About 1/3 of Rhesus negative women do not respond.
3. Protection is also afforded by ABO incompatibility between the mother and fetus. This is because incompatible fetal red blood cells entering the maternal circulation are rapidly destroyed before they can elicit an immune response. It reduces the incidence of sensitisation to about one-tenth of that seen when there is ABO compatibility. The patient presented was blood group "A" Rhesus "D" negative and her baby blood group B Rhesus D positive. There was therefore ABO incompatibility between the mother and her baby.
4. Other factors include variable antigenicity of the antigens and the varying rates of antigens occurrence.

Though fetal maternal haemorrhage goes on throughout pregnancy, the amount of blood involved is often insignificant to lead to sensitisation. Risk factors that favour increased fetal maternal haemorrhage include: amniocentesis, abortion, abdominal trauma, antepartum haemorrhage, caesarean section, and manual removal of the placenta (1,3). The primary immune response to the A antigen manifests over 6 weeks to 6 months. Its usually weak, consists predominantly of IGM antibody, which is too large to cross the placenta. Thus the first pregnancy is not at great risk. A second antigen challenge generates an amnestic response, which is both rapid and almost

exclusively IgG. Bowman observed that the longer the duration between challenges, the greater the increase in both the quantity of antibody and avidity with which it binds to the red blood cell (7). The presence of these features increases the chances of more severe fetal disease.

The overall risk of isoimmunisation for a Rhesus positive ABO-compatible infant with a Rhesus

negative mother is 16% (1,2,3). About 2% of the women will be immunized by the time of delivery, another 7% will have anti-D antibody by 6 months post-partum, and the remaining 7% will be "sensibilized"; that is they do not ordinarily produce antiDantibodies, but will demonstrate D-isoimmunisation when challenged in a subsequent pregnancy by another Rhesus D-positive foetus (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 larger volume (>200ml) the risk is slightly greater than 8%. However, at no volume, does there seem to be 100% immunization risk (1,2).

The development of effective maternal prophylaxis against sensitisation is credited to Finn and associates (1961) in England and Freda and co-workers (1963) in the USA (3). The immunoglobulin prevents Rhesus isoimmunization by competitive inhibition. All the antigenic sites (Loci) 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 (1,8). For the Rhesus negative insensitized mother, administrations of anti-D after delivery prevents up to 95% of Rhesus sensitisation. New cases however continue to occur, mainly due to sensitisation during pregnancy, which renders prophylaxis after delivery useless. Routine antenatal anti- D immunoglobulin therapy is therefore of proven cost benefit and should be given to all non-sensitized Rhesus negative women at 28 weeks (9). A second dose of anti-D, in the third trimester, at 34 weeks is now thought to be highly protective, not only for that pregnancy but also for the next two pregnancies and possibly the third. The failure rate of antenatal prophylaxis has been found to be a mere 0.1% compared to 1-6% postpartum (10).

Our patient did not receive antenatal prophylaxis, because post-partum prophylaxis is the standard protocol of managemen at the KNH. This now needs to be reviewed. The standard recommendation for the post-partum prophylaxis is to give anti-D, within 72 hours of delivery, but it has now been shown to be effective in preventing isoimmunisation if given up to 28 days after deliver (3).

The use of the Kleihauer test at potential sensitisation events will indicate whether additional doses of ant-D over and above the standard dose of 300µg (micrograms) are required.

For potential immunization events in the first half of pregnancy with a negative Kleihauer test, a dose of 50 microgram is thought to be adequate. In the 2nd half of pregnancy a dose of 100µg(microgram) is recommended (11).

The management of a pregnancy complicated by isoimmunization is guided by the past obstetric history and maternal antibody titres. A history of a previously affected foetus is especially important. The maternal antibody titres should be quantified. The mother is then 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. Fetal hemolysis as may be shown by amniocentesis may necessitate early delivery.

The Lileys charts are used to evaluate the severity of fetal hemolysis and the intervention criteria for fetal management. Depending on the severity of the disease, amniocentesis is repeated at 1-3 week intervals. **Zone** I generally indicates unaffected or mildly affected foetus, in which ammniocentesis is repeated every 2-3 weeks; the aim being delivery near term, after fetal lung maturity is attained. Zone II, or moderately affected fetuses; require weekly amniocentesis and delivery as soon as pulmonary maturity is reached. The severely affected foetus, in zone III, Needs intervention if death is to be avoided within a few days. Intra-uterine transfusion may be performed using 0-negative washed and irradiated packed cells. At KNH, intrauterine transfusion is not available. As a result of the lack of facilities for intrauterine transfusion, perinatal morbidity and mortality remains high among the isoimmunised women in Kenya. At KNH, a perinatal mortality rate of 600 per 1000 was reported. The practise at KNH is to

do exchange transfusion after delivery, for those babies that are severely affected. Other modalities of treatment that have been tried include plasmapharesis; immunosuppression with high doses of steroids; promethezine and D-positive erythrocyte membranes in enteric coated capsules to induce T-Suppressor cell formation (3,9). For the sensitised mother with repeated pregnancy loss, artificial insemination with Rhesus negative donor sperm remains an option.

REFERENCES

1. Norman B.B., John S. W; Rhesus immunisation in pregnancy. A review, obstet. Gynecol. Surv. 1993; 46 (12): 80
2. Pernoll M.L; Late pregnancy complications. In current obstetrics and Gynaecologic diagnosis and treatment 9th Ed. Appleton and Lange, 2003:295-300.
3. *Cuningham F. G., Norman F.G, Kenneth J.L. et al. Diseases and Injuries of the* foetus and newborn. In; William's obstetric 21st ed. Mc craw Hill, 2001: 1040-1068.
4. *Whitfield C.R. Blood disorders in pregnancy. In, Dewhurst's Textbook of obstetrics and Gynaecology. For post-graduate 5th Ed. Edited by Charles R. Whitefield. Black* well Science 1995. Chapter 16 pg. 228-250.
5. *Mati J.K.G,, Aggarwal V.P, Sanghvi H.C.G., et al. Nairobi Birth Survey; Antenatal* care in Nairobi. J. Obstet and Gynecol East and Cent. Afri. 1983; 2(1): 1
6. *Mulandi T.N.; A 2 year prospective study to show the effectiveness of anti-D* gamma globulin in preventing Rhesus isoimmunization in Rhesus Negative pregnant thesis, U.O.N, 1985.
7. *Bowman J.M., (1989). Maternal alloimmunization and fetal haemolytic diseases. In* Reece EA, Hobbins J.C, Mahoney M.J, Petrie R.H (eds). Medicine of the Fetus *and mother page 1152-1182. Philadelphia, PA J.B. Lippincott.*
8. *Magnus S., Rhesu isoimmunization. New perspective in maternal – Fetal* medicine. Obstet. Gynecol. Surv. 1991, 46(4(): 71.
9. *Bowman J.M, Chown B, Lewis M., Pollack J.M. (1978). Rhesus immunization during pregnancy: Antenatal prevention Canadian Medical Journal, 118:623-629.*
10. Mackenzie **12,** Selinger M., Bowell P.J.; The management of red cell *isoimmunization in the 1990's. In: Progress in obstetrics and Gynaecology vol. 9* Churchill Livingston 1991; 31-54.
11. *Chown B., Duff A.M., James J et al. (1969). Prevention of primary Rhesus* Immunization: First report of the western Canadian trial: Canadian medical Journal 100: 1021-1027.

OBSTETRICS CASE 15

NON REASSURING FOETAL HEART IN EARLY LABOUR - EMERGENCY CAESAREAN SECTION.

NAME: C. S.

IPNO 1108866 AGE: 26 YEARS

DOA 14/09/06 **DOD:** 21/09/06

26 years old Para 0+0 G1 LMP 23/11/05 EDD 30/08/06 GBD 42/40 admitted with Labour pains x 1/7

HISTORY OF PRESENTING ILLNESS

There was positive history of discharge of show but no drainage of liquor and no urinary symptoms.

She gave history of having had regular menses prior to current pregnancy. Her quickening was in early May 2006 so by extrapolation she was about 38 weeks plus..

PAST MEDICAL HISTORY.

Not significant

OBS/GYN HISTORY.

Menarche was at 14 years. Her menstrual cycle lasts for 28 days while the flow takes 4-5 days and is regular.

There was no history of contraceptive use in the past.

ANTENATAL HISTORY.

She attended ante natal clinic at Ongata Rongai Health Centre fora total of 6visits. Period was uneventful. She received 2 doses of anti tetanus toxoid injection.

Profile: Blood group 0 Rhesus positive, VDRL — Negative, Hb 13.0gm/dl, HIV-Negative

FAMILY SOCIAL HISTORY

She is married, a housewife. Husband is a casual labourer.There is no history of chronic illness in the family.

On examination, she was in fair general condition, not pale, jaundiced, cyanosed or oedematous

Vital signs: BP 129/68 mmHg PR 80 min Temp 36.7°c

P/A

Fundal height was term, longitudinal lie, cephalic; head 5/5 up, foetal heart rate was 132/ minute, regular.

V/E: Normal external genitalia, cervix posterior, 1.5 cm long, soft, closed white curdy discharge on examining finger.

An impression of false labour secondary to Vulvo-vaginal Candidiasis was made. Patient was admitted to ante natal ward on Clotrimazole persarries after having been given IM Pethidine 100 mg stat.

While in the ward, patient had spontaneous ruptures of membranes at 1.08 am on 15/09/06, then was transferred to Labour ward.

Review in L/W: P/A Fundal height was term, longitudinal lie, head was cephalic 4/5 up, foetal heart rate wasit 180 /minute, irregular.

V/E Normal external genitalia, cervix was 4cm dilated, there was meconium stained liqour grade 3 which was draining.

An impression of non reassuring foetal heart (NRFH) in early labour was made.

Management:

She was informed of the diagnosis and the need for an emergency C/S. An informed written consent was obtained for emergency C/S. Meanwhile she was advised to lie on left lateral position and given oxygen by mask. An intravenous line was established and 5% dextrose infused. Blood was taken for group and cross matching. She was pre-medicated with Atropine 0.6mg intramuscularly. She was then wheeled to theatre and underwent C/S as described earlier in this book.

Intra operatively, a LFI with loose cord round the chest xl was extracted, Apgar score 6/1, 7/5, 8/10, birth weight was 3450 gm admitted to NBU due to asphyxia. The patient was reversed

from general anaesthesia uneventfully.

Post operative care. Post operatively she was on intravenous fluids 500mls 5% dextrose alternating with normal saline 500mls 4 hourly for the first 24 hours. She was also on parenteral Crystalline Penicillin 2MU 6 hourly, Gentamycin 80 mg 8 hourly and Pethidine 100mg 8 hourly for 48 hours then oral Amoxil 500mg, Flagyl 400mgs and Brufen 400mgs for the next 3 days.

On the first post operative day the bowel sounds were established, so she was started on oral sips and early ambulation. By the second post operative day she was on light diet. Post-operatively patient did well and was discharged home on 7^{h} post operative day. (The baby was discharged from NBU on the sixth post natal day).

DISCUSSION

The patient presented was a primigravida who was seen with a non reassuring foetal heart (foetal distress) in early labour. She delivered by emergency Caesarean section to a live male infant.

Foetal distress is defined as a complex of signs indicating a critical response to stress characterized by metabolic derangements, notably hypoxia and acidosis that affects the functions of vital organs, mainly the brain to point of temporary or permanent injury or death [1]. Foetal distress is usually diagnosed during labour but can occur at any time during pregnancy. This is because during labour foetal monitoring is usually routine. It maybe divided into two phases – acute and chronic. Chronic distress implies an interval of sub-lethal foetal deprivation that affects growth and development that may be caused by a reduction in placental perfusion, by a placental abnormality or by deficient foetal metabolism [1], while acute foetal distress is a shorter reflection of the distress in labour when the stress of uterine contractions is added to a chronic lack of transfer of nutrients and/or oxygen 2.

The incidence of foetal distress varies widely mainly because of a lack of agreement on what should constitute it and the specificity of the clinical signs and tests performed. In Nairobi, its

incidence was found to be 5.2% for singleton pregnancies and contributed 10.4% of the total Caesarean deliveries [3]. In America, acute foetal distress was diagnosed in 20% of all obstetric patients [1]. This wide variation is attributed to the sophisticated electronic monitoring available in the USA.

The human foetus can basically become distressed in three common ways:

- Insufficiency of uterine blood flow as in hypertonic uterine contractions
- Insufficient umbilical blood flow
- Decrease in maternal arterial oxygen content as in severe anaemia or cardiac disease 4,5,6,

Our patient most likely developed foetal distress resulting from insufficient umbilical blood flow as the cord was relatively tight around the foetal chest intra-operatively.

Foetal heart rate monitoring as a means of assessing foetal well being has been the subject of some controversy. Clinical and laboratory studies demonstrated that foetal hypoxia reliably produces changes in FHR patterns; however these changes in FHR may also occur in the absence of foetal distress. The reason it is still important to detect a compromised foetus is because current estimates indicate that about 20% of all stillbirths, 20-40% of cases of severe mental retardation arise from intra-partum events leading to asphyxia 7.

FHR monitoring can be effected using either auscultatory monitoring or electronic monitoring. The electronic monitors can either be external or internal. The auscultatory method is the common widely used method of monitoring FHR in labour worldwide. It is subject to considerable human error and must be used with considerable care. The foetal heart rate tones should ideally be recorded for 30 seconds immediately after a uterine contraction at least every 30 minutes during the first stage, every 15 minutes during the

7

second stage and every 10 minutes in the delivery room . Ideally if an abnormal FHR pattern is detected, direct FHR monitoring should be instituted. In our set up, the auscultatory method is the principal method used with each patient assigned a primary nurse.

Diagnosis of foetal distress is based on certain clinical findings including tachycardia, bradycardia, passage of meconium into the amniotic fluid and late heart decelerations. Unrecognized foetal distress can result in foetal jeopardy and therefore skillful foetal monitoring (both ante-partum and intra-partum) is imperative if this is to be prevented. Ante-partum foetal surveillance helps identify the potential for foetal compromise. Recording of foetal movements in high risk pregnancies has been widely used to predict early foetal compromise. In case of cessation of foetal movements, foetal heart sounds have been observed to disappear within the next 24 hours. In fact Sadovsky and Polishuk found loss of foetal movements to be more reliable than urinary oestriol in predicting impending foetal demise [6] Other methods of ascertaining ante-partum foetal well beng include performing contraction stress test, non-stress test and more recently biophysical profile.

The patient presented here had irregular foetal heart rate, mainly tachycardia, and spontaneous rupture of the membranes revealed meconium stained liquor. These signs have a low predictive value for foetal distress when considered alone, but taken together their value rises considerably. Yeomans et al in 1988 showed that labour complicated with meconium staining of amniotic fluid was more likely to be associated with neonatal acidosis [8] However, meconium staining alone predicts for foetal distress in only 3.5% but when taken together with foetal heart rate abnormalities the predictive value raises to about 35%. Therefore a foetus with heart rate abnormalities and meconium staining of liquor calls for more aggressive investigations like scalp blood PH analysis if the patient is in labour. Foetal distress certainly exists if tachycardia, late FHR deceleration and lack of FHR short term variability occur and are confirmed as an ensemble characteristic of the uterine contraction and FHR patterns. If severe variable deceleration persists for 30 minutes or more, or if any degree of late deceleration persists despite attempted therapy, foetal distress is present. Concomitantly, the foetal scalp PH will probably be 7.20 or less and meconium will be passed in to the liquor

Management of foetal distress or possible distress involves several steps:-

- The mother's position should be changed; this may relieve pressure on the umbilical cord and also uterine pressure on the inferior vena cava therefore

improving uterine flow with increased $^{0}_{2}$ supply and CO2 removal.

- Maternal hypotension should be corrected, the change in position will probably correct supine hypotension but additional measures include elevation of the legs and rapid administration of fluids. This helps restore arterial pressure and increase blood flow in the intervillous space
- Steps taken to decrease uterine activity by stopping the administration of oxytocin, decreased uterine activity permits better placental perfusion and reduces the stress of violent contraction.

- Administration of 02 at the rate of 6-8 L/min by face mask increases the maternal-foetal $^{0}_{2}$ concentration gradient and will increase maternal-foetal 02 transfer.

- If maternal acidosis is the cause of foetal distress, the acid base balance should be corrected by giving sodium bicarbonate and infra-venous hypertonic glucose (usually 50%) may be administered when there is maternal deprivation acidosis or hypoglycaemia '- If the signs of foetal distress persist for 30 minutes, or if there is foetal distress despite conservative treatment, immediate delivery is mandatory. The mode of delivery should be dictated by obstetric judgement by considering the presentation, station, position, cervical dilatation and presumed foetal state. Our patient was 4 cm dilated and was therefore delivered by emergency Caesarean Section. The infant was mildly depressed with an Apgar score of 6 at 1 minute, but the score of 7 at 5 minutes indicated a chance of good long term outcome.
- Fetal distress has met with disfavor in recent years, and the American College of Obstetricians and Gynecologists now urges physicians to employ the more descriptive "non-reassuring fetal heart rate tracing." ACOG acknowledges, however, that the phrase fetal distress has been so commonly applied to abnormal intrapartum FHR patterns that it will be difficult to get obstetricians to abandon the term completely 9

REFERENCES.

1. Herrera E., Pernoll M.L.,Complications of Labour and Delivery. Current obstetrics and gynaecological diagnosis and treatment. 7h edition, Appleton and Lange, ch. 24 pg.493; 1991

2. Chamberlain G.U.P., Ritchie J. W. K., The foetus, placenta and amniotic fluid: Foetal surveillance. Dewhursts' textbook of obstetrics and gynaecology for postgraduates. 4th edition, Blackwell scientific publications 1986 ch.10 pg.461

3. Mati J. K. G. , Sanghvi H. C. G., et al. The Nairobi Survey I. J. Obstet. Gynae. East Cent. Aft., 1:132; 1982.

4. Itskovitz J., La Gama E. F.,The effect of reducing umbilical cord flow on foetal oxygenation. A.M. J. Obstet. Gynaecol. 147:813-18; 1983

5. Parer J. T.,The effect of maternal hypoxia to foetal oxygenation and the umbilical circulation. Eur. J. of Obs. & Gynae. Repr. Biol. 10 :125-36; 1980

7. Sadovsky E., Polishuk W.Z. Foetal movements in utero: Nature, assessment, prognostic value, time of delivery. Obstet. Gynaecol. 50:49; 1977

8. Yeomans E., Gilstrap E., Leveno K.J. et al. Meconium stained amniotic fluid and neonatal acid base status. Obstet. Gynaecol 73: 175; 1989

American College of Obstetricians and Gynecologists:

RESEARCH DISSERTATION:

REASONS FOR FAILED INDUCTION OF LABOUR WITH PGE_2 AND $PGF_{2\ ALPHA}$ FROM 28 WEEKS GESTATION AMONG SUBJECTS WHO ARE INDUCED AT KENYATTA NATIONAL HOSPITAL MATERNITY UNIT.

ABSTRACT

Background: Induction of labour involves the artificial initiation of uterine contractions by pharmacological or mechanical means before spontaneous onset of labour due to various indications with the aim of achieving a normal delivery and favorable pregnancy outcomes. The goal of induction of labour is to prevent the potential risks to the fetus and /or the mother with prolonged intrauterine existence by achieving a normal delivery as soon as possible. Currently at most maternity units induction of labour is commonly done using prostaglandins (mainly PGE_2 and PGF_2 alpha with or without oxytocin and amniotomy. The success rate or failure rate of induction of labour is determined by the mode of delivery and the outcome of pregnancy. A failed induction of labour usually refers to failure to progress to active phase of labour or when a mother that was being induced ends up being delivered by caesarean section. Reasons that contribute to failure of induction of labour have not been clearly defined. This is despite the fact that the rate of induction has been shown be to increasing in most maternity units over the years. Understanding these factors is critical to efforts to increase success rate associated with induction of labour especially if the factors can be corrected. This study sought to determine modifiable and un-modifiable factors that contribute to failure of induction of labour among mothers undergoing labour induction with PGE_2 or PGF_2 alpha from twenty eight weeks gestation.

Study objectives: Was to determine the reasons for failed induction of labour among mothers undergoing induction of labour with PGE_2 or PGF_2 alpha from twenty eight weeks gestation.

Study population: Four hundred and eighty mothers were induced with PGE_2 or PGF_2 alpha from twenty eight weeks.

Study setting: Kenyatta National Hospital maternity unit.

Study design: The study was a retrospective descriptive study.

Study methodology: Mothers who had undergone induction of labour during the study period with PGE_2 or PGF_2 alpha from twenty eight weeks were recruited into the study using the maternity register and hospital records. To determine the induction rate the total number of deliveries during the study period was obtained. Using a questionnaire the socio demographic characteristics, intrapartum course, maternal and fetal outcomes were entered in the questionnaire. Analysis of the data obtained was done using the Statistical Package for Social Sciences (SPSS) version 16.0 and personal computer data editor programmer. Data validation was done before analysis.

Results: A poor Bishop' score< 7, prolonged duration of labour, incorrect administration of prostaglandins and mothers with infants weighing ≥ 3500 grammes had a significant association with failed induction of labour.($p< 0.05$). The overall induction rate was 8.8% while the caesarean section rate was 23.8%.

Conclusions: Standard operating procedure needs to be developed in the department in order to standardize the management of mothers undergoing induction of labour in the unit.

Recommendations: A proper maternal evaluation needs to be done before commencing mothers on induction of labour. A prospective comparative study which is more analytical and comprehensive should be done to assess the factors that may lead to failed induction of labour. Standard operating procedures should be developed on mothers undergoing induction of labour in order to streamline the services.

INTRODUCTION

Induction of labour refers to the artificial initiation of uterine activity to effect labour before the spontaneous onset of uterine contractions usually above twenty-eight weeks gestation. Induction of labour is indicated for a variety of maternal and fetal conditions, when benefits either to the mother or fetus outweigh those of continuing the pregnancy [1] The goal of induction of labour is to minimize potential risks to the fetus and the mother.[1, 2] Initially most obstetricians and midwives were reluctant to interfere with the natural course of pregnancy by artificially hastening the onset of labour. This was because the methods used were considered dangerous.[1,2]

Historically intravenous oxytocin infusions have been the agents of choice for induction of labour. However oxytocin infusion in pregnant women with unripe cervix has been associated with a high failure rate. This is because the state of cervix before induction of labour has tremendous impact on pregnancy outcomes.[2] In modern obstetrics the most commonly used methods for cervical ripening and induction of labour are use of prostaglandin compounds particularly E_1, E_2, F_2 alpha, amniotomy and oxytocin.[3] These methods can be used either singly or in combination.

Prostaglandins are a series of closely related twenty carbons unsaturated fatty acids containing a cyclopentane ring. They are derived from essential fatty acids and arachidonic acid and are synthesized in most organs of the body. Prostaglandins are divided into various groups according to the configuration of the cyclopentane ring. The number of double bonds on the side chain is depicted by a subscript below the letter depicting the series.[4] The most commonly used prostaglandins for cervical ripening and induction of labour are prostaglandins E_2 (PGE_2) and Prostaglandin F_{2alpha} (PGF_{2alpha}) preparations which contain dinoprostone and prostaglandin E_1 (PGE_1) or misoprostol, a synthetic PGE_1 analogue. PGE_1 is used in some maternity units for induction of labour and has been shown to be more potent than PGE_2 and PGF_{2alpha} for induction of labour.

LITERATURE REVIEW

The most commonly used prostaglandins for cervical ripening and induction of labour are prostaglandins E_2 (PGE_2) and Prostaglandin F_{2alpha} ($PGF_{2\alpha}$) preparations which contain dinoprostone. Before commencing induction of labour usually the state of the cervix is assessed. To achieve this a scoring system was developed in which the dilatation, length, consistency and position of the cervix is assessed. The distance of the fetal head from the Ischia spine of the pelvis (station of the presenting part) is also assessed. The findings of the parameters are then interpreted by using a scoring system from 0 to 13 known as the Bishop's score. A Bishop's score of seven or less is considered unfavorable.[5]

Bishop's method of cervical scoring for induction of labour

SCORE	0	1	2	3
Cervical dilatation(cm)	0	1-2	3-4	5+
Length of cervix(cm)	3	2	1	0
Station	-3	-2	- 1, 0	+1, +2
Consistency	Firm	Medium	Soft	
Position	Posterior	Central	Anterior	

Administration of prostaglandins

PGE_2 vaginal tablets are given at a dose of three milligrams, six to eight hourly to a maximum dose of six milligrams. At the Kenyatta National Hospital maternity unit the same regimen is recommended. In case of ruptured membranes, intravenous oxytocin is recommended as an alternative initiating agent. If oxytocin is used after PGE_2, six hours should elapse after the last dose of PGE_2 is inserted to reduce the risk of hyper stimulation of the uterus. Oxytocin is recommended as an intravenous infusion of five units in five hundred milliliters of normal saline and titrated from ten drops per minute to sixty drops per minute to achieve good uterine contractions. This can be escalated further to ten units depending on the progress of labour.[6] An

oxytocin pump can also be used which is commenced at 3 milliliters / hour and escalated at 3 milliliters every half hour until 18 milliliters maximum.[6,7]

Prostaglandin F_2 alpha (PGF_2 alpha) can also be used to induce labour. Intravenous administration of PGF_2 alpha is indicated for term induction of labour and for evacuation of a third trimester foetal death in utero where a 15-micrograms/ milliliters solution of PGF_2 alpha is infused by intravenous route at a rate of 2.5 micrograms/minute for at least thirty minutes. The level is then maintained if an appropriate uterine response is achieved. If no uterine response has been achieved infusion is continued at 2.5 micrograms/minute every hour until a satisfactory uterine response has been reached, but a level of 20 micrograms/minute should not be exceeded.[8,9] PGF_2 alpha can also be administered extra amniotically by using a Foleys catheter which is inserted through the cervical os and inflated. A diluted solution of PGF_2 alpha (i.e. two milliliters of PGF_2 alpha & eighteen milliliters injection) is infused through the catheter every hourly until cervical ripening is achieved when the catheter falls off. This is specially done in cases of mid trimester pregnancies with intrauterine foetal death.[9]

Artificial rupture of membranes (ARM)

Artificial rupture of amniotic membranes (amniotomy) through the cervix has been documented as a method of induction for over two hundred years. A purpose designed plastic hook or Kocher's forceps is used. Amniotomy is commonly used in combination with oxytocin infusion for induction of labour. In women with a ripe cervix and a high Bishop's score amniotomy has been reported to be 88% successful in inducing labour.[10] Nevertheless amniotomy is best performed only when the cervix is favorable. The presumed mechanism of action is the release of endogenous prostaglandins, which in turn results in cervical changes leading to labour. Before amniotomy pelvic evaluation of the cervix should be done. The presenting part should be well applied to the cervix to minimize the risk of cord prolapse. Various instruments can be used to rupture the membranes. These include the Kocher's forceps, a long clamp, Allis clamp or an Amnihook. The instruments are passed through the fingers and the membranes are hooked or scratched to rupture them. After rupture of membranes the state of the liquor should be noted.[11, 12]

Other induction methods

Other methods of induction of labour include: -

a) Mechanical methods (dilators); these methods are effective in ripening the cervix and include hygroscopic dilators, osmotic dilators (laminaria digatata), the 24 French Foley balloon and the double balloon. They cause mechanical stimulation of the cervix that may result in endogenous release of prostaglandins.[13, 14]

b) Castor oil, bath and enema; these were used as a method of inducing labour. It has been suggested that they can cause reflex hyperactivity of the uterus which can lead to labour.[14,15]

c) Relaxin has been of interest as a cervical ripening agent as it may lead to uterine stimulation activity. Its clinical use has not been determined.[15, 16]

d) Hyaluronidase may enhance cervical ripening when injected to the cervix, but the method is rarely used.[15]

Indications of induction

Indications of induction of labour are several. Before labour induction, maternal and foetal risk benefit analysis should be performed. The indications include; [16]

- Hypertensive disease in pregnancy
- Renal diseases in pregnancy
- Premature rupture of membranes (PROM)
- Postdate pregnancy
- Abruptio placenta
- Rhesus Isoimmunization
- Foetal growth restriction
- Diabetes, renal disease
- Placental insufficiency
- Reduced foetal movements(maternal perception)

Logistic factors can also be considered to induce a patient. These include psychosocial indications, history of rapid labour or risk of rapid labour and foetal anomalies requiring specialized neonatal care. Absolute and relative contra indications of induction of labour have

been outlined. The absolute contraindications include cephalopelvic disproportion, placenta praevia(type 2 posterior, 3 and 4), vasa praevia, previous uterine scar(s) and Malpresentation.[14] The relative contradictions include breech presentation, polyhydromnios, multiple gestations, grand multiparity and prematurity.[14]

Incidences of failed induction

The rate of induction of labor varies between various obstetrics units and has been shown to be increasing over the years. Overall induction rate in the city of Nairobi was given as 5.7 % (Mati et al 1983).[17] In Kenyatta National Hospital in 1984 the induction rate was to be found to be 5.6%.[18] At the Aga Khan Hospital in Nairobi, Kenya, a study was done on the review of risk factors associated with induction of labour in 1999. 120 women on induction of labour (cases) versus 240 women (controls) who had achieved spontaneous labour were reviewed and the rate of induction of labour was 14%.[19] In 2002, a prospective descriptive cross sectional study done at the Kenyatta National Hospital to determine the indication for induction of labour and pregnancy outcome, a total of 185 women undergoing induction of labour were assessed. During the study period a total of 1455 deliveries were conducted giving an induction rate of 12.7%. [20] On maternal outcomes the caesarean section rate was 22.1% while 75.1% delivered by spontaneous vertex delivery. These studies showed that the rate of induction has been increasing upwards. With such trend it is expected that the failure rate of induction of labour is expected to rise especially if the indication of labour induction is not considered properly. With induction of labour on the increase especially without clearly identifiable indications of inductions the consequences for the mother in terms of failed induction leading to caesarean delivery is a very relevant issue to be considered.

Predictors of failed induction

Various studies have been done on indications of labour inductions and predictors of failed induction of labour with the aim of improving the success rate. In one prospective hospital based study done in 2008 at Kathmandu Medical University, the indications of labour inductions and the predictors of failed inductions were analyzed. The incidence of labour induction was

19.7%.[21] The caesarean section (CS) rate was 36.4%. Of these 36.4%, the reasons for C/S were failed induction (74.07%) and foetal distress (25.03%). The predominant indication for induction of labour was post term pregnancy (51.28%) followed by PROM (17.3%), isolated oligohydromnios (8.97%), hypertension (8.33%) and maternal perception of reduced foetal movements (7.69%). Failed induction of labour was increased in primigravidas (41.2%) compared to multigravidas (23.7%) [21]. Failure rate of induction of labour was 53.8% when maternal age was greater than thirty years and 28.2% in those less than thirty years.[21] 24.1% had failed induction of labour when Bishop's score was greater than 5 and 40.8% when it was less than 5. This finding on the Bishop's score was complimented by another study which showed that induction of labour in the presence of unripe cervix (>3) results in a longer and higher incidence of caesarean section and fetal asphyxia.[21, 22] On gestation, mothers between 38 to 41weeks gestation had failed induction in 31% while in those > 41 weeks, failed induction was found in 28% of the mothers. The best outcome was seen when foetal weight was 2500 to 2900grammes (22.8%) while 72.7% had failed induction when birth rate was greater than 3500grammes.[21] The conclusion of the research was that despite proven benefit of induction of labour in selected cases one must keep in mind its impact on increased rates of operative delivery. Strategies for developing practical guidelines may help to prevent unwarranted cases and help to decrease the current high operative deliveries.[21]

Other studies have investigated the duration of latent phase of labour and its influence on the mode of delivery amongst induced mothers. In a retrospective analysis of 978 nulliparous mothers it was demonstrated that as the length of latent phase increased the likelihood of birth by caesarean section increased significantly ($p< 0.0001$).[23] A longer latent phase greater than 18 hours was associated with greater rate of caesarean section especially in over 18 hours.[22,23]

From these studies it is reasonable to conclude that the indications of induction of labour have to be convincing and modifiable and factors such as Bishop's score have to be considered before commencing induction of labour. This was shown in a study that was done in Australia in 2008 over a 15 month period where 1057 consecutive inductions were performed. The induction rate of labour was 10%. The CS rate was 16.5%.[22] On indications for CS 74(7.0%) women were done due to failed induction of labour. 58(5.5%) women were done due to foetal distress.19 (1.8%)

women due to cephalopelvic disproportion + malposition. An analysis of indications for IOL revealed that number of inductions had debatable obstetric indications. It was therefore concluded that tailoring induction of labour to cervical score and indications of IOL night reduce the CS rate of failed IOL. [22]

Induction of labour is associated with several risks and complications with adverse maternal / foetal outcomes. The cause of failed induction may be due to foetal distress as a result of increased uterine activity or cephalopelvic disproportion that is undiagnosed before induction. Increased uterine activity (hyper stimulation) can occur especially by prostaglandins and oxytocin use. This can lead to uteroplacental hypo perfusion and foetal hypoxia.[1, 19, 24] In fact foetal asphyxia and placental abruption are more common as indication for CS in induced mothers than in those undergoing spontaneous labour. Systemic maternal side effects of prostaglandins and oxytocin also include nausea, vomiting, diarrhea, pyrexia and hypersensitivity/anaphylactic reactions. Bronchospasm can occur therefore making induction of labour with prostaglandins among asthmatic patients risky.[24] Use of oxytocin can lead to water intoxication. This is because of its structural and functional similarity to pituitary antidiuretic hormone. When administered in large doses over a twenty-four hour period it can result in water intoxication that can lead to hypernatremia, confusion, convulsion, coma, congestive heart failure and death. A long induction delivery period can also lead to sepsis. Other complications of oxytocin infusion include abruptio placenta, precipitate delivery and neonatal hyperbilirubinemia.[12, 24, 25] The potential risks of amniotomy include umbilical cord compression, cord prolapse, chorioamnionitis and rupture of vasa praevia. Stripling of the amniotic membrane may result in bleeding from a low lying or undiagnosed placenta praevia.[1, 17, 19, 20]

.LE / STUDY JUSTIFICATION:

of labour is a common and important obstetric procedure which aims to minimize isks to the fetus and / or the mother that would result with continuing the pregnancy. not all mothers that undergo induction of labour end up with a successful delivery. At /atta National Hospital maternity units, approximately 22.1% mothers undergoing ı of labour are ultimately delivered by Caesarean section.[20] Reasons that contribute to f induction at the hospital have not been clearly defined. This is despite the fact that the induction has been shown to be increasing from 5.6%[18] in 1984 to 12.7% in 2002.[20] anding these reasons is critical to efforts to increase success rate associated with on of labour especially if the factors can be corrected. This study sought to determine able reasons that contribute to failure of induction of labour among mothers undergoing ion of labour with PGE_2 or PGF_2 alpha from twenty eight weeks gestation. The cteristics of mothers diagnosed with failed induction of labour was also considered,

;EARCH QUESTION

there reasons that may be associated with failed induction of labour among mothers lergoing induction of labour for various indications with PGE_2 and PGF_{2alpha}?

ONCEPTUAL FRAMEWORK

ıarrative

Induction of labour refers to the artificial initiation of uterine activity to achieve labour before the spontaneous onset of uterine contractions usually above twenty-eight weeks gestation. Induction of labour is indicated for a variety of maternal and fetal conditions, when benefits either to the mother or fetus outweigh those of continuing the pregnancy The goal of induction of labour is to minimize potential risks to the fetus and the mother by achiveing a favourable outcome.

The success rate or failure rate of induction of labour is determined by the mode of delivery and the outcome of pregnancy whereby a failed induction of labour usually refers to failure to progress to active phase of labour causing the mother to be delivered by caesarean section.

RATIONALE / STUDY JUSTIFICATION:

Induction of labour is a common and important obstetric procedure which aims to minimize potential risks to the fetus and / or the mother that would result with continuing the pregnancy. However not all mothers that undergo induction of labour end up with a successful delivery. At the Kenyatta National Hospital maternity units, approximately 22.1% mothers undergoing induction of labour are ultimately delivered by Caesarean section.[20] Reasons that contribute to failure of induction at the hospital have not been clearly defined. This is despite the fact that the rate of induction has been shown to be increasing from 5.6%[18] in 1984 to 12.7% in 2002.[20] Understanding these reasons is critical to efforts to increase success rate associated with induction of labour especially if the factors can be corrected. This study sought to determine modifiable reasons that contribute to failure of induction of labour among mothers undergoing induction of labour with PGE_2 or PGF_2 alpha from twenty eight weeks gestation. The characteristics of mothers diagnosed with failed induction of labour was also considered.

RESEARCH QUESTION

Are there reasons that may be associated with failed induction of labour among mothers undergoing induction of labour for various indications with PGE_2 and PGF_{2alpha}?

CONCEPTUAL FRAMEWORK

Narrative

Induction of labour refers to the artificial initiation of uterine activity to achieve labour before the spontaneous onset of uterine contractions usually above twenty-eight weeks gestation. Induction of labour is indicated for a variety of maternal and fetal conditions, when benefits either to the mother or fetus outweigh those of continuing the pregnancy The goal of induction of labour is to minimize potential risks to the fetus and the mother by achiveing a favourable outcome.

The success rate or failure rate of induction of labour is determined by the mode of delivery and the outcome of pregnancy whereby a failed induction of labour usually refers to failure to progress to active phase of labour causing the mother to be delivered by caesarean section.

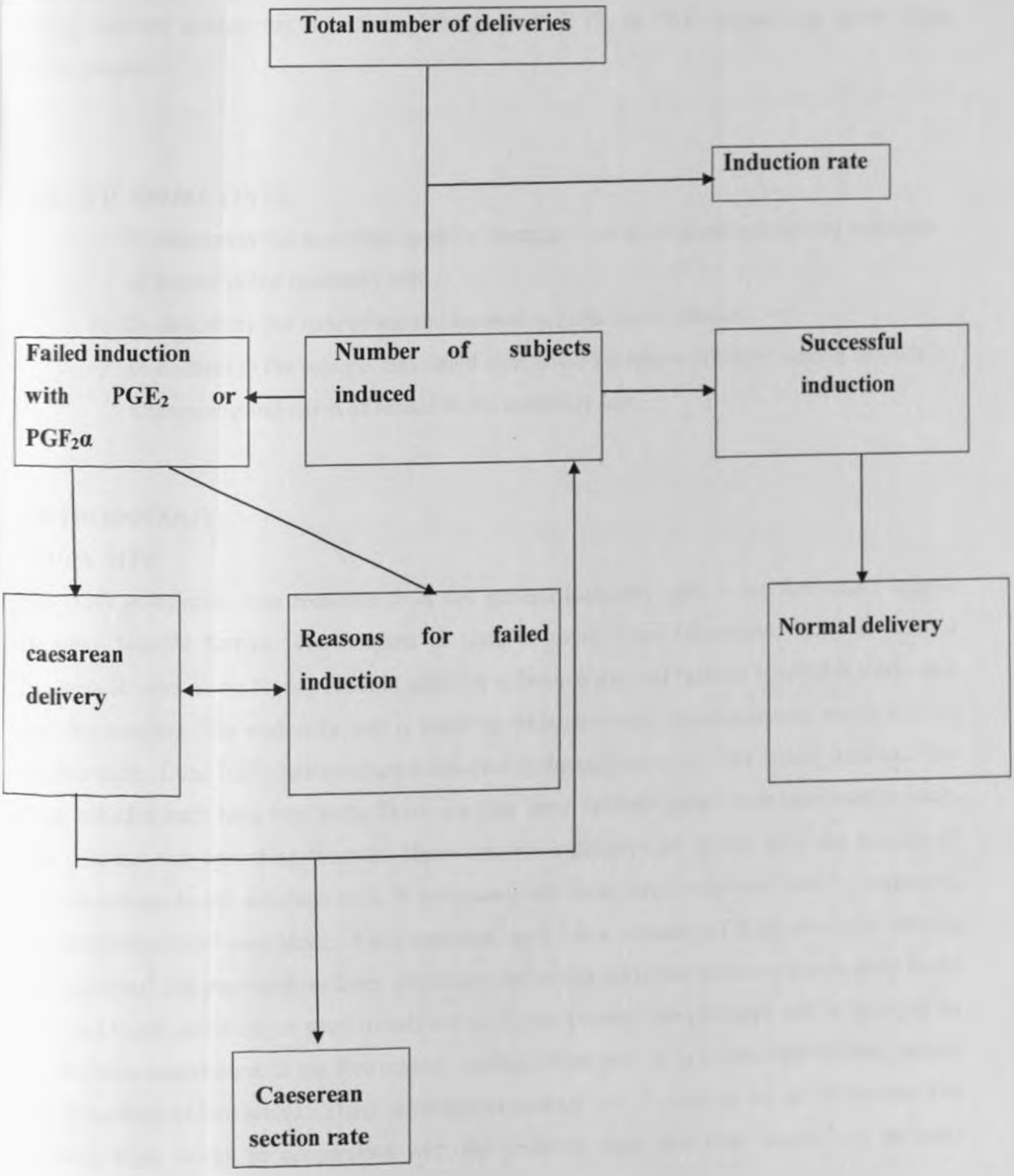
Reasons that contribute to failure of induction of labour have not been clearly defined yet the rate of induction keeps on increasing in most maternity units there by also increasing the failure rate. This was a retrospective descriptive study to determine the reasons for failed induction of labour among mothers undergoing induction of labour with PGE_2 or $PGF_2\alpha$ from twenty eight weeks gestation.

Mothers undergoing induction of labour who were induced with PGE_2 or PGF_2 α.and failed to achieve a normal labour (i.e delivered by caesarean section) were studied inorder to determine if there were reasons associated with failed induction of labour. The variables that were studied and their effect on induction of labour were:

- Age
- Educational level
- Parity
- ANC attendance
- Gestation at induction
- Bishop's score
- Pelvic assessment
- Prostaglandin insertion
- Duration of labour
- Fetal birtweight

The findings of this study would help in improving the management of mothers admitted for induction of labour for various reasons.

Diagramatic conceptual framework



STUDY OBJECTIVES.

BROAD OBJECTIVE

The overall objective was to determine reasons associated with failure of induction of labour among mothers undergoing induction of labour with PGE_2 or PGF_2 alpha from twenty eight weeks gestation.

SPECIFIC OBJECTIVES

a) To determine the sociodemographic characteristics of subjects undergoing induction of labour in the maternity unit.
b) To determine the indications and method of induction of labour.
c) To determine the reasons associated with failed induction of labour among subjects undergoing induction of labour in the maternity unit.

METHODOLOGY:

STUDY SITE

The study population was recruited from the general maternity unit at the Kenyatta National Hospital, Nairobi Kenya. The hospital is situated about three kilometers from the Central Business District along Ngong road. In addition to being a national referral hospital it serves as a teaching hospital. The maternity unit is made up of labour ward, three antenatal wards and the newborn unit. Over 7000 deliveries are conducted in the unit annually. The labour ward has first stage cubicles each with two beds. There are also three delivery suites with two coaches each. The unit has two operating theatres. There are two incubators in labour ward for transfer of preterm infants to the newborn unit. Ward rounds are done daily in labour ward by registrars, senior registrars and consultants. Each antenatal ward has a capacity of thirty-two beds serving both antenatal and postnatal mothers. Registrars and senior registrars review patients daily in the antenatal wards while major ward rounds are held once a week. The newborn unit is managed by the pediatric department. It has five nursery cubicles. One cubicle is isolated for infected babies and those born before arrival. There are thirty incubators and ten cots in the newborn unit. The obstetric team works in cooperation with the pediatric team and both teams hold monthly maternal and perinatal mortality meetings.

The study site was found suitable because it is busy maternity unit with deliveries occurring in the unit per day. The maternity unit at the Kenyatta National Hospital is also a teaching hospital for various cadres of students therefore it is expected that a high quality of services offered at the unit. The maternity unit is also the main referral centre in the country where mothers from various other health facilities with complicated pregnancies are admitted for management.

STUDY DESIGN

The study was a retrospective descriptive study whereby the files of four hundrend and eighty mothers who had undergone induction of labour during the study period were used to determine the reasons for failed induction of labour with PGE_2 or $PGF_{2\alpha}$ from twenty eight weeks gestation at the KNH maternity unit.

STUDY SUBJECTS

Four hundred and eighty mothers who were admitted in the maternity unit during the study period and underwent induction of labour with PGE_2 or $PGF_{2\alpha}$ from twenty eight weeks for various reasons were identified as the study subjects.

SAMPLE SIZE DETERMINATION

The minimum size required in this study was calculated according to the formula: [26]

$$n = \frac{N * Z^2 * p * (1-p)}{d^2 * (N-1) + Z^2 * p * (1-p)}$$

Where n was the minimum sample size required

N was the expected eligible number of deliveries in a year

Z was the standardized score at 95% two tail level to 1.96.

P was the failure of an induction given that it was induced =induction rate at 12.7%*22.1% failure rate of all inductions. The induction rate was found to be 12.7% in a prospective descriptive study at KNH in 2002 while the failure rate was found/ estimated to be 22.1%[20]

d was the level of precision taken at 0.05

Therefore p=0.028067

1-p=0.971933

n=7000

Z^2=1.96

d^2=0.0025

N-1=6999

Calculating the sample size

$$n = \frac{7000 * 1.96^2 * 0.028067 * (1-0.028067)}{0.05^2 * (7000-1) + 1.96^2 * 0.127 * (1 - 0.028067)}$$

n = 400 mothers

Adding 20% non response sample size required = **480**

Therefore the files of **four hundred and eighty** mothers who had undergone induction of labour with PGE_2 or PGF_2 alpha from twenty eight weeks were considered.

DATA COLLECTION

Data was extracted from the hospital files in the records department containing doctors and midwives handwritten notes. The maternity register in labour ward which has a record of normal spontaneous labour and caesarean section was also used. At the maternity unit, approximately five mothers are induced daily for various reasons with PGE_2 and PGF_{2alpha}. To achieve a sample size of 480 mothers, the files of all mothers who had been induced in the last six months i.e. from October 2010 to March 2011 were retrieved during the study period. Since there were no records of induced mothers in the maternity register the files of all the mothers who had been induced was obtained directly from the records department with the assistance of the records clerk during the study period. Only files that fulfilled the inclusion and the exclusion criteria of the study were retrieved for use in the study. Once the files had been identified, the principal researcher and a trained research assistant collected the data by filling in a structured data collection form.

The data collection form was coded to make it easier to enter the information obtained in the computer. The data of the total number of mothers who delivered in the maternity unit during the study period was obtained until a sample size of four hundred and eighty mothers who underwent induction of labour from twenty eight weeks with PGE_2 or PGF_2 alpha either singly or in combination with amniotomy and / or oxytocin was achieved. A study number was entered for each file identified. Any mother who was delivered by caesarean section after undergoing induction of labour was considered as failed induction. Using a formatted and pre-tested questionnaire the principle investigator then extract information from the marked files. The information retrieved included the socio demographic characteristics, previous obstetric history, data on the indication of induction of labour, clinical presentation of mother at induction, the Bishop's score of the cervix, the method of induction of labour, mode of administering the PGE_2 or PGF_2 alpha, progress/duration of labour, mode of delivery and foetal outcomes. Every used file was marked with a sticker to avoid repetition of data. The patients' names were kept confidential by using the study numbers. To determine the rate of induction the total number of deliveries during the study period was considered.

DATA MANAGEMENT

The data wsas checked for completes and correctness then entered into a microcomputer using SPSS (statistical package for social sciences version 16.0) and personal computer data editor programme. Data validation was done before analysis. Errors were cleaned and data coded. Chi square test of significance was used. Analysis of data included descriptive statistics i.e. frequency distribution, means, standard deviations, proportions and cross tabulations. Computation of data was done using the Pearson likelihood ratio and Mantel-Haenszel test of linear associations. Tables were used to represent the results.

ELIGIBILITY CRITERIA

Inclusion criteria

a) Mothers admitted at the Kenyatta National Hospital maternity unit and induced with prostaglandins PGE_2 or PGF_2 alpha.
b) Mothers with gestational age of twenty eight weeks and above.

Exclusion criteria

a) Mothers with gestational age below twenty eight weeks were excluded.

b) Mothers for induction of labour should not have gone into spontaneous labour.

ETHICAL CONSIDERATIONS

Permission to carry out the study was sought from the Kenyatta National Hospital Research and Ethical Committee (KNH/UON-ERC). The study therefore commenced after obtaining the approval of the head of department in records to retrieve the files.

The questionnaires did not bear any patients names and the mothers were only identified by study numbers. The information obtained from the files was kept confidential and was used for research purposes only. On completion and approval of the research study after the data had been verified the information obtained in the questionnaires was destroyed.

STUDY LIMITATIONS

- Since this was a retrospective descriptive study all the information was obtained from the patients files and therefore there was no way of verifying the information given on the patient's file.
- Poor recording of information by the records clerk led to some information being partially recorded or missing.
- Some handwritings in the files were illegible
- Some files had torn or missing pages.
- Identifying files of mothers who had been induced with prostaglandins in the records department was tedious since these mothers are classified as normal or caesarean section deliveries in the maternity register in labour ward.

RESULTS

The principal investigator carried out a retrospective descriptive study among mothers who had undergone induction of labour at the Kenyatta National Hospital maternity unit between October 2010 and March 2011. The files of 480 mothers who had undergone induction of labour during the study period and fulfilled the inclusion criteria were retrieved and found suitable for analysis. During the study period a total of 5472 mothers were delivered in the unit giving an induction rate of 8.8%. Table one shows the socio demographic characteristics of mothers who had undergone induction of labour with either PGE_2 or $PGF_{2\alpha}$ in the maternity unit.

Table one: sociodemographic characteristics of induced subjects

Socio demographic variables (%)	Number
Age(N=480)	
≤20	66(13.8)
21-25	168(35.0)
26-30	142(29.6)
31-35	64(13.3)
36-40	29(6.0)
40-45	10 (2.1)
≥46	1(0.2)
Marital status (N=480)	
Single	76(15.6%)
Married	386(80.4%)
Divorced/separated	6(1.3%)
Widowed	12 (2.5%)
Educational level (N=480)	
None	6(1.2%)
Primary	182(37.9%)
Secondary	192 (40.0)
Tertiary	100 (20.8%)
Parity (N=480)	
1	157(32.7%)
2-3	284(59.2%)
4-5	33(6.9%)
≥5	6(1.3%)

Most of the mothers undergoing induction of labour for various reasons were aged between 21-25years i.e. 35% with 78.4% of the mothers being aged 30 years and below. The mothers who were aged between 30 to 35years were 13.3% while 6.0% were aged 36 to40 years. The mothers

who were aged 41to 45 years were 2.1%. The youngest mother was aged 17 years and the oldest 46years. The mean age of the mothers was 27.2 years and the mode was 25 years.

Majority of the mothers who presented to the unit for induction of labour were married i.e. 80.4% with only 15.6% being single. 1.3% were either divorced or separated while 2.5% were widowed.

Most of the mothers had gone to school up to secondary level i.e. 40.0%. Primary school education was attained by 37.9% of the mothers while 20.8% had attended either college or university. Only 1.2% of the mothers had no formal education at all.

Majority of the mothers, 59.2% who were being induced were carrying their 2^{nd} or 3^{rd} pregnancies. 32.7% of the mothers' undergoind induction of labour in the unit were primigravidas while only 1.3% who were induced had their 5^{th} pregnancy.

The mothers underwent induction of labour due to various reasons. Induction of labour was done either with prostaglandin E_2 or Prostaglandin F_2alpha. These were used either in combination with artificial rupture of membranes with or without use of oxytocin. Table two shows the indications of induction of labour and mode of induction.

Table two: **Indications and method of induction of labour**

Indications of induction (n=480)	Number (%)
Preeclampsia/eclampsia	141(29.3%)
Postdates(≥42weeks)	25(5.2%)
PROM	13(2.8%)
Rhesus negative status	30(6.3%)
Reduced fetal movements	37(7.8%)
Fetal demise	26(5.4%)
Recurrent false labour	23(4.8%)
Induction at 40-41weeks+	150(31.2%)
Maternal disease in pregnancy	10(2.0%)
No indication	25(5.1%)
Method of induction (n=480)	
PGE_2 only	28(5.8%)
PGF_{2alpha} only	9(1.9%)
PGE_2+amniotomy	91(19.0%)
PGF_{2alpha}+amniotomy	6(1.3%)
PGE_2+amniotomy+ oxytocin	329(68.5%)
PGF_{2alpha}+amniotomy+ oxytocin	7(1.5%)
PGF_{2alpha}+oxytocin	10(2.1%)

Most of the mothers, 31.2% where induced after having presented to the unit at 40 to 41weeks+ gestation. They were actually not postdates. Only 5.2% of the mothers had attained the gestational age of 42 weeks and above. Pre eclampsia or eclampsia contributed to 29.3% of the induced mothers. Their gestation ranged from a gestational age of 28 to 39 weeks. Mothers with reduced fetal movements were 7.8% while 6.3% of the mothers were induced due to their rhesus negative status. Fetal demise constituted 5.4% of the mothers induced. Some mothers underwent induction of labour without any identifiable cause i.e 5.1%. Recurrent false labour was also an indication of labour in the maternity unit i.e 4.8%. They underwent induction of labour from 38 weeks onwards.

Most of the mothers, who were admitted in the unit i.e 68.5%, underwent induction of labour with a combination of PGE_2, amniotomy and use of oxytocin while 19.0% were induced using

PGE_2 and artificial rupture of membranes. Only 5.8% of the mothers were induced after having received PGE_2 pessaries only. The mothers were induced with PGF_2 alpha and oxytocin were 2.1% while 1.9% was induced using PGF_2 alpha only.

The mothers who underwent induction of labour were either delivered normally, by assisted breech delivery, assisted vacuum delivery or by emergency caesarean section. Table three shows the mode of delivery and indication for caesarean section among the mothers who delivered in the maternity unit. In this study mothers who were delivered by caesarean section after undergoing induction of labour for various reasons were considered as failed induction of labour.

Table three: Mode of delivery and indication for caesarean section

Mode of delivery (N=480)	**Number (%)**
Spontaneous vertex delivery	340(70.8%)
Assisted vacuum delivery	15(3.1%)
Assisted breech delivery	11(2.3%)
Caesarean section	114(23.8%)
Indications of caesarean section(N=114)	
Ante partum hemorrhage	12(10.5%)
Uterine rupture	2(1.8%)
Cord accidents	4(3.6%)
Meconium stained liquor	18(15.8%)
Cephalopelvic disproportion	22(19.3%)
Non reassuring fetal status	12(10.5%)
Failed induction of labour	44(38.6%)

Normal delivery accounted for 70.8% of the deliveries while 3.1% and 2.3% were delivered by assisted vacuum and breech delivery respectively. The mothers who were delivered by emergency caesarean section were 23.8%. These were classified as failed induction of labour collectively. However 44 out of 114(38.6%) of the mothers had already been diagnosed with

failed induction of labour due poor progress of labour in the first stage of labour. 19.3% of the mothers who had been commenced on induction of labour were diagnosed with cephalopelvic disproportion in the first stage of labour. There were two mothers i.e 1.8% who developed ruptured uterus and had to undergo emergency Caeserean section.

The caesarean section rate in this study was 23.8%. By being induced with prostaglandins the intention was to achieve a normal delivery. The researcher tried to determine if there is any significant association between failed induction of labour and various socio demographic characteristics/ maternal assessment before induction of labour that could act as predictors of failed induction of labour.

In order to determine the factors that are associated with failed induction of labour the variables that were significantly associated with success rate of the induced mothers were considered.

Table four shows the socio demographic characteristics and their significance on the outcome of induction of labour.

TABLE Four: Reasons for failed induction of labour by sociodemographic characteristics (N=480)

Variable	SVD	Breech	Vacuum	Caesarean	Total	P Value
Age(years)(N=480)						**0.256**
≤ 20	38	1	2	25	66	
21-25	124	5	7	32	168	
26-30	104	4	5	29	142	
31-35	44	0	1	19	64	
36-40	20	1	0	8	29	
40-45	9	0	0	1	10	
≥46	1	0	0	0	1	
Educational level(N=480)						**0.70**
None	5	1	0	0	6	
Primary	179	3	6	74	262	
Secondary	137	8	9	38	192	
Tertiary	18	0	0	2	20	
Parity (N=480)						**0.214**
Primigravida	103	3	4	44	154	
Multigravida	237	8	11	70	326	

In this study there were no significant association between the age of the mother and failed induction of labour (p =0.256). There were also no significant between the educational level and parity with failed induction of labour (p = 0.7 and 0.214 respectively).

Other reasons leading to failed induction of labour were also studied. The ANC attendance, gestation at induction, Bishop's score, pelvic assessment, method of prostaglandin insertion, duration of labour and the fetal birthweight were studied among the mothers to determine if there were any associated significant relationship between failed induction of labour and these parameters. Table five shows a cross tabulation of various maternal parameters and their association with failed induction of labour.

Table five: Reasons for failed induction of labour

Variable	SVD	Breech	Vacuum	Caesarean	Total	P Value
ANC attendance (N=480)						**0.250**
Yes	320	9	14	99	442	
No	16	1	1	14	32	
Not recorded	4	1	0	1	6	
Gestation at induction(weeks(N=480)						**0.080**
28-34	37	1	0	3	41	
>34-38	120	1	2	32	155	
>38-42	262	9	13	79	284	
Bishop's score (N=480)						**0.000**
<7	173	3	8	74	258	
>7	81	2	0	4	87	
Not recorded	86	6	7	36	135	
***Pelvic assessment**						
Adequate	223	9	12	83	327	
Not recorded	119	2	3	29	153	
Insertion of prostaglandin(N=480)						**0.0003**
Correct	77	0	1	9	87	
Not correct	263	11	14	105	393	
Duration of labour (hours)(N=480)						**0.0001**
No labour	27	0	0	0	27	
< 12 hours	137	3	4	11	155	
12-24 hours	160	7	9	51	227	
>24 hours	24	1	2	21	48	
Not recorded	19	0	0	4	23	
Birth weight (grammes)(N=480)						**0.000**
<2000	46	0	2	6	54	
2000-2499	38	0	1	3	42	
2500- 2999	55	1	1	8	65	
3000- 3499	83	2	2	17	104	
3500-3999	98	8	4	57	167	
>4000	24	0	1	23	48	

Mothers who had a poor Bishop's score of less than7 were more likely to undergo caesarean section compared to mothers with a good bishop's score of >7 (p=0.000). In study 173 mothers with a Bishop's score of less than 7 delivered by SVD while only 81 who had a Bishop's score of greater than 7 delivered by SVD. The method of insertion of prostaglandins was more likely to influence the mode of delivery. Those mothers who had their prostaglandins inserted wrongly (p=0.0003) without following the correct time limit were more likely to be delivered by caesarean section compared to mothers who had their prostaglandin inserted correctly.

Mothers who delivered infants weighing more than 3500grammes were more likely to be delivered by caesarean section compared to mothers whose infants were less than 3500 grammes(p=0.000). Mothers who had a prolonged duration of labour<24 hours were most likely to be delivered by caesarean section compared to mothers who had a shorter duration of labour (p=0.0001). There was no significant association between the mode of delivery and age, parity, ANC attendance and gestation at induction of labour.

In this study the significance of the outcome of induction and pelvic assessment was unreliable since most of the mothers undergoing induction of labour in the unit did not have a proper pelvic assessment.

Discussion

The study was conducted at the KNH maternity unit were 480 mothers were induced due to various obstetric indications. The induction rate was 8.8%. Studies have been done in KNH on induction of labour. Overall induction rate in the city of Nairobi was given as 5.7 % (Mati et al 1983).[17] In Kenyatta National Hospital in 1984 the induction rate was to be found to be 5.6%.[18] In 2002, a prospective descriptive cross sectional study done at the Kenyatta National Hospital to determine the indication for induction of labour and pregnancy outcomes an induction rate of 12.7% was obtained.

This study was focusing on possible reasons that could have lead to failed induction of labour. The results of this study show that most of the mothers undergoing induction of labour for various reasons were of the younger age group ie aged less than 30 years. This could be explained by the fact that most obstetrics complications occur in the younger age group. In this study nearly all the mothers had attained some formal education i.e only 1.2% had no formed mal education at all. With a high level of educational status it can be assumed that the mothers were knowledgeable about their obstetrics status.

Only 1.3% of the mothers had more than five children with 59.2% having between two to three children. This was expected because most of the mothers undergoing induction of labour were aged less than thirty years and therefore it was expected that the parity was not high.

Most of the mothers, 31.2% where induced after having presented to the unit at 40 to 41weeks+ gestation. They were actually not postdates. The practice in most obstetric units is to induce all mothers who present themselves at fourty weeks and above. This was the case in this study and these mothers contributed to the highest number of induced mothers in the unit. The mothers who were truly postdates were only 5.2%.

Pre eclampsia or eclampsia contributed also contributed to a high number of induced mothers i.e 29.3%. The study site is a referral centre were mothers are refered and therefore conditions like hypertension were expected to increase in number. It was also noted that some mothers

underwent induction of labour without any identifiable cause i.e 5.1%. This was most likely to cause an increase the induction rate in the unit.

The caesarean section rate was 23.8%. This is a high number considering the fact that on commencement of induction of labour the purpose was to attempt to achieve a normal delivery. In one prospective hospital based study done in 2008, the indications of labour inductions and the predictors of failed inductions were analyzed. The incidence of labour induction was 19.7% and the caesarean section (CS) rate was 36.4%[21].

Most of the mothers, 92.1% who underwent induction of labour in the unit had attended antenatal clinic. Therefore even though the mothers had been admitted to be induced for various reasons, the problems associated by not attending ante natal clinic were minimized. The mothers who underwent induction of labour had a wide distribution of ages i.e. from 17 to 46 years and were being induced for various indications from 28 to 42 weeks gestation. However 5.1% of the mothers in this study were induced without any clear indication. This was unnecessary because it contributed to increased rates of induction in the unit.

Some studies have shown that unnecessary inductions of labour have lead to increased caesarean section rates in maternity units. This was shown in a study that was done in Australia in 2008 over a 15 month period where 1057 consecutive inductions were performed. The induction rate of labour was 10%. The caesarean section rate was 16.5%.[22] On indications for caesarean section 74(7.0%) women were done due to failed induction of labour. 58(5.5%) women were done due to fetal distress.19 (1.8%) women due to cephalopelvic disproportion and malposition. An analysis of indications for inductions revealed that number of inductions had debatable obstetric indications. It was therefore concluded that tailoring induction of labour to cervical score and indications of inductions might reduce the caesarean section rate of failed induction of labour. [22]

23.7% of the mothers who underwent induction of labour in the maternity unit developed complications. 2.5% of the mothers were diagnosed with a non reassuring fetal status while 2.5% had meconium stained liquor. Induction of labour is associated with several risks and complications with adverse maternal / fetal outcomes. The cause of failed induction may be due

to fetal distress due to increased uterine activity. Increased uterine activity (hyper stimulation) can occur especially by prostaglandins and oxytocin use. This can lead to uteroplacental hypo perfusion and fetal hypoxia.[1, 19, 24] 2.5% of the mothers developed antepartum hemorrhage. Studies done on complications of induction have shown that the most common cause of antepartum hemorrhage in induced mothers is abruption placenta and in most cases fetal asphyxia and placental abruption are more common as indication for caesarean section in induced mothers than in those undergoing spontaneous labour.[19] Other complications include the potential risks of amniotomy done during induction of labour i.e. umbilical cord compression, cord prolapse, chorioamnionitis and rupture of vasa praevia.[19,24] In this study 3.6% of the mothers were diagnosed with cord prolapse.

Out of the 114 mothers that were induced, 19.3% of them were diagnosed with cephalopelvic disproportion. This is a high number considering the fact that it was also found that in 31.9% of the mothers undergoing induction of labour did not have pelvic assessment done on them to determine if their pelvis was adequate. This was shown in a study that was done in Australia in 2008 over a 15 month period where 1057 consecutive inductions were performed. 19 (1.8%) women were done caesarean section due to cephalopelvic disproportion and malposition.[22]

89.2% of mothers who delivered in the unit had live births. 2.9% of the mothers had fresh stillbirths. There are several reasons why the mothers might have developed the fresh stillbirths. Prostaglandins can cause uteroplacental hypo perfusion leading to fetal hypoxia and eventually fetal death. [2]The fetus might also have been compromised before the commencement of induction considering the fact that the mothers were induced due to several indications like preeclampsia.

In this study some of the factors the factors that were found to be significantly associated with failed induction of labour included a poor Bishop's score, prolonged duration of labour, correct method of insertion of prostaglandins and mothers delivering infants with fetal weights more than 3500 grammes. Mothers who had a Bishop's score of less than seven were more likely to be delivered by caesarean section compare to mothers who had a Bishop's score of more than seven. [21] Studies have been done on the Bishop's score. In one prospective hospital based study

done in 2008 at Kathmandu Medical University, the indications of labour inductions and the predictors of failed inductions were analyzed, 24.1% had failed induction of labour when Bishop's score was greater than 5 and 40.8% when it was less than 5. This finding on the Bishop's score was complimented by another study which showed that induction of labour in the presence of unripe cervix (>3) results in a longer and higher incidence of caesarean section and fetal asphyxia.[21, 22] In this study it was of concern because 31.8% of the mothers who underwent induction of labour did not have the Bishop's score recorded.

It was also found that a longer duration of labour was more likely to lead to failed induction of labour especially if the mother was in labour for more than 24 hours. Other studies have investigated the duration of latent phase of labour and its influence on the mode of delivery amongst induced mothers. In a retrospective analysis of 978 nulliparous mothers it was demonstrated that as the length of latent phase increased the likelihood of birth by caesarean section increased significantly ($p< 0.0001$).[23] A longer latent phase greater than 18 hours was associated with greater rate of caesarean section especially in over 18 hours.[23]

Among the mothers who were induced, 393(81.8%) had the their prostaglandins inserted by not following the correct timings. Most of the mothers either had one PGE_2 inserted and was not followed by the second one, or the second one was inserted after more than 12 hours instead of the usual 6 to 8 hours. Some of the mothers who were induced with PGF_2alpha and were being via an extra amniotic catheter also had the incorrect timing followed when infiltrating the catheter with the prostaglandins. This was more likely to lead to increased caesarean section rates. PGE_2 vaginal tablets are given at a dose of three milligrams, six to eight hourly to a maximum dose of six milligrams. PGF_2 alpha can be administered extra amniotically by using a Foleys catheter which is inserted through the cervical os and inflated. A diluted solution of PGF_2 alpha (i.e. two milliliters of PGF_2 alpha & eighteen milliliters injection) is infused through the catheter every hourly until cervical ripening is achieved when the catheter falls off. This is specially done in cases of mid trimester pregnancies with intrauterine fetal death.[9]

On fetal weights mothers who had their infants weighing more than 3500grammes were more likely to be delivered by caesarean section compare to mothers whose infants weighed less. This is complimented by other similar studies that have recorded similar findings. In one study the best outcome was seen when fetal weight was 2500 to 2900grammes were only 22.8% of the mothers underwent caesarean section compared to72.7% who had failed induction when birth rate was greater than 3500grammes.[21]

There were no significant association between the maternal age, gestational age at induction and parity with failed induction of labour. However other studies have found different findings. In one study failed induction of labour was increased in primigravidas (41.2%) compared to multigravida (23.7%) [21]. Failure rate of induction of labour was 53.8% when maternal age was greater than thirty years and 28.2% in those less than thirty years. On gestation, mothers between 38 to 41weeks gestation had failed induction in 31% while in those > 41 weeks, failed induction was found in 28% of the mothers.[21]

Conclusions

- Bishops score, duration of labour after induction, fetal weight and mode of administration of prostaglandins will influence the mode of delivery.
- Most of the mothers undergoing induction of labour in the maternity unit did not get a proper maternal evaluation before commencing the process of induction. The Bishop's score and pelvic assessment were not always done.
- Majority of the mothers had an incorrect administration of prostaglandins because the correct timings were not followed or they received the incorrect dosage.
- Some of the mothers were induced without a clear indication therefore increasing the caesarean section rates in the unit.

Recommendations

- There is need to emphasize that a proper maternal evaluation needs to be done before starting mothers on induction of labour in the unit.

- A prospective comparative study needs to be done in the maternity unit to assess the factors that may lead to failed induction of labour in the unit. This will be more analytical and comprehensive.
- The obstetrics and gynecology department needs to develop guidelines or standard operating procedures on mothers undergoing induction of labour in order to streamline the services.

REFERENCES

1. Lippincott Williams and Wilkins, 2000. Cervical Ripening and Labour Induction; Clinical Guidelines. Volume 43(3) pp 524-536.

2. Dutta DC, 1998, Textbook of Obstetrics: Induction of Labour; 4th Ed. Chapter 34 pp558.

3. Shyken JM and Dectrie RH 1995, Oxytocin to Induce Labour, Clin, Obstet Gynaecol 38:2, pp 232-245.

4. William F. Ganong, 1987, Prostaglandins, Review of Medical Physiology, Prentice Hall International Inc, pp225.

5. Calder AA, 1995. Induction and augmentation of labour, In Turnbull's Obstetrics 2nd edition pp 691.

6. Royal College of Obstetricians and Gynaecologists (RCOG), 2002; Evidence Based Clinical Guideline No.9, Induction of Labour, London.

7. G. Justus Hofmeyr: 2003, Best Practice and Research Clinical Obstetrics and Gynaecology; Induction of Labour with Unfavorable Cervix. Vol. No.17, No.5 pp 777-794.

8. Pharmacia, 2000, Prostin F_2 alpha ™ Sterile Solution Dinoprost. NV/SA,-Pours Belgium.

9. Lukas M and Brickerl, 2002, Intravenous Prostaglandin for Induction of Labour. The Cochrane Library Issue No.4. Oxford: Update Software.

10. Rayborn WF et.al, 1994, A Model for Investigating Microscopic Changes Induced by Prostaglandin E_2 in term cervix. J Matern Fetal Invest, 4: 137-140.

11. UK Amniotomy Group: 1994, A Multicentre Randomized Trial of Amniotomy is Spontaneous Labour at Term: Br J Obstet Gynaecol. 1994, 101: 307-309.

12. Hussein P Kofier E., Rasmossen A Et.al. 1983, Oxytocin and Initiation of Human Parturition: American Journal of obstetrics and Gynaecology, 147:503-507.

13. Atad. J. et.al, 1997, Ripening and Dilation of the Unfavorable Cervix for Induction of Labour by a Double Balloon Device: Experience with 250 Cases. Br.J.Obstet.Gynaecolol; 104; 29-32.

14. Alan H.Cherney and Martin L.Pernoll, 1994, Current Obstetrics and Gynecology: The Course and Conduct of Normal Labour and Delivery; 8th Ed, Chapter 10 pp 224.

15. Kelly AJ, et.al 2002, Castor Oil Birth. Enema for Cervical Priming and Induction of Labour. The Cochrane Library Issue 4: Oxford: Update software.

16. American College Obstetricians and Gynaecologists.1999, Clinical Management and Guidelines for Obstetricians-Gynecologists. Number 10:1-10.

17. Mati JKG et.al The Nairobi Birth Survey 111. Journal of Obstetrics and Gynaecology of EA 2:47. 1983.

18.Kaguta TK, Review of induction of labour at KNH, MMed thesis 1984.

19. Khisa WW, 1999, Review of Risk Factors Associated with Induction of Labour at The Aga Khan Hospital Nairobi. MMed Thesis, University of Nairobi, pg. 194-247.

20. Njagi J M, 2002, Indications and pregnancy outcome of induction at Kenyatta National Hospital. MMed Thesis University of Nairobi.

21. Rayamajhi RT et.al, Incidences and indicators for labour inductions and the study of predictors of failed induction. KUMJ (2009) vol 7, No 1, issue25.21-25.

22. S Arulkumaran et.al, Failed induction of labour. Australian & NZ Journal of Obs /Gyn, Vol 25: issue 3: pg 190-193;2008.

23. Michael Beckmann, Predicting a failed induction, Australian & NZ Journal of Obs /Gyn, Vol 47, issue 5, pg 394-398; 2007.

24. British National Formulary; 2003, Prostaglandins and Oxytotics. Chapter 7, pg 379-380.

25. Chalmers I, Campbell H, Thurnbull AC., 1975, The Use of Oxytocin and the Incidence of Neonatal Jaundice. BMJ. 2: pg 116-118.

26. World Health Organization (WHO), 1986, Sample size determination, Users manual, W.H.O/ H.S.T/ E.S.M/ 86: 1 (Revised).

STUDY QUESTIONNAIRE;

REASONS FOR FAILED INDUCTION OF LABOUR WITH PGE_2 AND $PGF_{2\ ALPHA}$ FROM 28 WEEKS GESTATION AMONG SUBJECTS WHO ARE INDUCED AT KENYATTA NATIONAL HOSPITAL MATERNITY UNIT.

A. IDENTIFICATION:

Date D/M/Y _____/_____/_____

Study number _____ _____ _____

B. SOCIO DEMOGRAPHIC DATA

1. Age (in completed years) _____ _____

2. Marital status

   1. Single ☐
   2. Married
   3. Divorced/separated
   4. Widowed

3. Level of education?

   1. None
   2. Primary ☐
   3. Secondary
   4. College/ University

C. OBSTERIC HISTORY

4. Did the mother attend antenatal clinic?

   1. Yes
   2. No ☐

5. Gestation by dates (using LNMP) or scan (in weeks) _____

6. Parity _____+_____

D. INDUCTION OF LABOUR

7. Gestation at which induction of labour was started (in weeks). _____ _____

8. What was the indication of labour _____

9. What was the Bishop's score before induction?

   1. Not done
   2. Actual Bishop's Score ☐

10. What was the pelvic assessment before initiation of induction?

    1. Adequate
    2. Inadequate
    3. Not done ☐

11. What was the fetal presentation before induction of labour?

    1. Not done
    2. Cephalic
    3. Other presentation ☐

12. What was the method of induction used?

    1. PGE_2 only
    2. PGF_2 alpha only
    3. PGE_2+ Amniotomy ☐
    4. PGF2 alpha+ Amniotomy
    5. PGE_2+ Amniotomy+Oxytocin
    6. PGF_2 alpha +Amniotomy+Oxytocin
    7. Others (Specify)

13. Was the PGE_2 administered correctly i.e. two pessaries six to eight hours apart?

    1. Yes
    2. No ☐

14. Was the PGF_2 alpha administered correctly?

    1. Yes
    2. No ☐

E. MATERNAL OUTCOME OF PREGNANCY

15. Was a partograph administered during the first stage of labour?
    1. Yes
    2. No

16. What was the duration of first stage of labour hours?
    1. Not recorded
    2. Actual duration in hours

17. Was /were there any complication(s) during first stage of labour?
    1. No complication
    2. Uterine hyper stimulation
    3. Hemorrhage
    4. Uterine rupture
    5. Cord accidents
    6. Meconium stained liquor
    7. Others (specify)

18. What was the mode of delivery?
    1. Spontaneous vertex delivery
    2. Breech delivery
    3. Vacuum delivery
    4. Caesarean section

19. If C/S what was the reason?
    1. Not stated
    2. Actual reason

F. FOETAL OUTCOME OF LABOUR

20. What was the state of the fetus delivered?

    1. Live birth
    2. Fresh stillbirth
    3. Macerated stillbirth

21. If live birth what was the foetal Apgar score in one minute?

    1. One-minute
    2. Five minutes
    3. Not recorded

22. What was the foetal birth weight?

    1. Not recorded
    2. Actual weight in grams

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi
Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi.
Email: KNHplan@Ken.Healthnet.org

17th March, 2011

Ref: KNH-ERC/ A/54

Dr. Julius O. Ondigo
Dept. of Obs/Gynae
School of Medicine
University of Nairobi

Dear Ondigo

RESEARCH PROPOSAL: "KNOWLEDGE ATTITUDE AND PRACTICE (KAP) OF CONTRACEPTION AMONG SEXUALLY ACTIVE BREASTFEEDING WOMEN IN THE FIRST YEAR AFTER DELIVERY" (P437/12/2010)

This is to inform you that the KNH/UON-Ethics & Research Committee has reviewed and **approved** your above revised research proposal for the period 17th March 2011 – 16th March 2012.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimens must also be obtained from KNH/UON-Ethics & Research Committee for each batch.

On behalf of the Committee, I wish you a fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of the data base that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF A N GUANTAI
SECRETARY, KNH/UON-ERC

c.c. The Deputy Director CS, KNH
The HOD, Records, KNH
The Chairman, Dept. Obs/Gyane, UON
Supervisors: Dr. James Machoki M'Imunya, UNITID, UON
Dr. John Kinuthia, Dept. of Obs/Gynae, UON

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi
Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi
Email: KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/ A/76

7th April 2011

Dr. Julius Ochieng Ondigo
Dept. of Obs/Gynae
School Medicine
University of Nairobi

Dear Dr. Ondigo,

RESEARCH PROPOSAL: **"FACTORS ASSOCIATED WITH FAILED INDUCTION OF LABOUR WITH PGE OR PGF ALPHA FROM TWENTY EIGHT WEEKS GESTATION AT KNH MATERNITY UNIT" (P30/2/2011)**

This is to inform you that the KNH/UON-Ethics & Research Committee has reviewed and **approved** your above revised research proposal for the period 7th April 2011 – 6th April 2012.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimens must also be obtained from KNH/UON-Ethics & Research Committee for each batch.

On behalf of the Committee, I wish you a fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of the data base that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF A N GUANTAI
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Dr. John Kinuthia, Dept of Obs/Gyane, UON