Long gynecological commentary

MORBIDITIES ASSOCIATED WITH RADIOTHERAPY TREATMENT FOR WOMEN WITH HISTOLOGICALLY CONFIRMED CANCER OF THE CERVIX AT KENYATTA NATIONAL HOSPITAL BETWEEN 2000-2004

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ABSTRACT

Background.

Cancer of the cervix is the second most common cancer affecting women world wide, but it is the commonest gynecological cancer in Kenya. An estimated 500,000 cases occur yearly world wide, of which 80% are from Sub- Sahara Africa. Women often present in disease stage III and IV, and among Kenyan women presenting with cancer of the cervix only 7-8% are at stages amenable to surgery. The majority of the women (92%) requiring radiotherapy. There are only 2 units in Kenya offering radiotherapy, both are located in Nairobi. Radiation therapy has attended morbidities which could be early or late. Gastrointestinal, genitourinary, hematological are among systems commonly affected.

Objective:

Assess morbidities associated with radiotherapy treatment for women presenting with histological confirmed cervical cancer in the radiotherapy department at Kenyatta National Hospital.

Population: Women treated for cervical cancer at Kenyatta National Hospital radiotherapy unit year 2000-2004.

Study Design and Methodology. This was a descriptive retrospective data was collected from hospital records at the radiotherapy unit. In a 5 year period between 2000-2004 every 5th file of women recorded to have cancer of the cervix was analyzed. Sociodemographic, clinical, and laboratory data was recorded on structured and pre-tested questionnaires.

Results

There were 1545 records with a diagnosis of cervical cancer, of which a sample of 306 cases were assessed; 10(3.3%), were in stage IB, 124(41.5%) stage II, 142(47.5%) stage III, and 23(7.7%) stage IV. A total of 266(86.9%), were squamous cell carcinoma type with 167(54.9%) poorly differentiated. Mean age was 49 years with a (SD +/-11years), 50% were married, of the 58 with HIV results 16(5.2%) were sero-positive. The time between earliest symptoms and initiation of radiotherapy was mean 11 months SD +/-14 months. All the 306 patients underwent radiotherapy, 258 (84.3%) completed EBRT. The common early toxicities were skin burns, diarrhoea and rectal bleeding which latter decreased. After one year skin fibrosis was observed in 137(45%) of the women, GIT toxicities was observed in 107(35%) of the women. Other morbidities encountered were vaginal stenosis, urinary incontinence, dysuria, and anaemia occurred in less than 20% of the study population. About half the patients 161(52%). came back for review in the radiotherapy department after completing the EBRT Loss to follow-up was, 145(47%) at 6 months, 200(66%) at 12 months, 254(83%) at 24 months and 298(97%) at 60 months. At 2 years only 10% and 5% women at stage III and IV respectively

returned for follow-up, while 27% of the women at stage IIA returned for follow-up at 2 year period. No deaths were recorded in the case files so real survival could not be calculated from this study.

Conclusion

1. Eighty eight percent of the women presented with disease stage II and III.
2. Skin fibrosis and gastrointestinal toxicities were observed in more than 30% of the women after 1 year of follow-up.
3. Patients' follow-up declined with time and was dependent on disease stage.
4. Higher radiation dosage and longer treatment time were associated with more systems being involved.

INTRODUCTION

Cancer of the cervix is one of the most common cancers affecting women worldwide (1, 2). It comes second to breast cancer among cancers affecting women (1, 2, 3). But it is the commonest gynecological cancer in Kenya (4.5). An estimated 500,000 cases occur every year of which 80% are from the developing countries (3, 4)). In the developed world the incidence rates are generally low with age-standardized rates less than 14 per 100,000 women compared to 37.4 per 100,000 women in East Africa (5, 6).

The Kenyan national incidence of cancer of the cervix is unknown, as there is no population based cancer registry (5). It is estimated that the incidence is 37-47 per 100,000 women per year (5). Approximately 350-400 cases of invasive cancer of the cervix (ICC) are treated at KNH yearly (5, 6). Yearly estimate of patients with ICC in Kenya is 3,600. Only a few patients reach KNH (Kenyatta National Hospital). KNH is the only public hospital with a functional radiotherapy unit for treatment of ICC (5, 6). In Kenyatta National hospital (1990) it was reported that 46% of the deaths in the acute gynecological ward was due to the complications of cancer of the cervix (5).

This study sought to evaluate the outcome of patients with advanced carcinoma of the cervix treated with radiotherapy.

LITERATURE REVIEW.

Oncogenesis and associated risk factors.

Sexual behavior is a key risk factor associated with cancer of the cervix (8). The age of first intercourse, number of sexual partners, number of partners of the male partner, histories of previous STDs are factors explored in the sexual history (8 and 9). Human papillomavirus (HPV) type 16, 18, 33, 45, 56 are now known to cause cancer of the cervix (8, 9, 10, 11, 12). At KNH it was demonstrated up to 99% of all cases of cancer of the cervix had the major high risk HPV types 16 and 18 (12). In a case control study HPV DNA was detected in 90-100% of the cases compared to 20% of cervical specimen from suitable epidemiological controls (8).Additionally we now have vaccines to type 16 and 18 HPV, which are 100% efficacious in preventing HPV infection and HPV related CIN2-3 for at least 3.5 years after immunization(13, 14). Role of the male partner being a carrier for the HPV DNA has been confirmed (11). It is recommended that all women who are sexually active or at the age of 20 should undergo a screening Pap smear yearly. Once 3 normal smears have been documented the continued survailance can be lengthened.

Other risk factors include cigarette smoking, immunosuppression, parity and long term oral contraception use (11). More recently HIV has been associated with increased risk of disease and increased disease progression,(Invasive cervical cancer has been included as an AIDS-defining illness)(9,12). Immunocompromised women require more aggressive survailance (6 monthly pap smear) . Poverty, level of education and access to medical care are factors that affect what stage the patient presents at (17).

Diagnosis and staging.

Abnormal vaginal bleeding is the most common symptom of invasive cervical cancer (Postcoital bleeding, Bloodstained leucorrhoea discharge, scanty spotting or frank bleeding) (5,15). Other manifestations of advanced disease include pelvic pain, which may radiate to one hip, urinary or feacal incontinence due to fistula formation. Weakness, weight loss and anemia also characterize the late stages (15, 16). Symptoms commonly arise with overt cancer, pre-invasive disease is usually asymptomatic thus the role of routine pap smears for all who are sexually active. .

Physical findings may range from a normal appearing cervix (grossly) to infiltrative cancer producing enlargement, irregularity and a firm consistency of the cervix and eventually of adjacent parametria. Endophytic or exophytic (cauliflower like) growth pattern on the cervix occurs (15, 16). Further disease progression will involve the vaginal fornices, extensive parametrial spreads makes nodular thickening in the utero-sacral and cardinal ligament with a resultant immobile fixed cervix (15, 16).

Examination under anaesthesia (EUA) is performed, a biopsy specimen is take for histology and in the same sitting staging of the disease is done. Other essential work-ups include a complete blood count renal function tests, liver function tests and abdomino- pelvic ultrasound. Some patients may require an Intra-Venous urogram, chest X-ray or even an abdominal CT scan. Cystoscopy is not done at KNH for staging at KNH, it is possible that some patients are under-staged leading to bias in the long term outcome.

Clinical staging estimates the extent of the disease not only for prognostic purposes but also for treatment planning. This also affords a means of comparing methods of therapy for the various stages of disease world-wide (5,15).

Histological types are: Squamous cell carcinoma, Adenocarcinoma and others (large cell non-keratinizing carcinoma, large cell keratinizing carcinoma, clear cell carcinoma etc)(15).

FIGO staging of cervical cancer. (1998) plus treatment. (15).

stage			Characteristics	treatment
Stage0			Carcinoma in situ	Local excision with close follow-up
Stage I. Lesson confined to cervix.	IA Microscopic cervical lesion	A1	stromal invasion not >3mm deep, horizontally >7mm	Simple hysterectomy
		A2	Stromal invasion < 3mm but >5mm deep not wider than 7mm horizontally	
	IB Clinically visible cervical lesion	B1	Lesions > 4cm	Radical hysterectomy+/- radiotherapy
		B2	Lesions <4cm	
Stage II. Tumors extends beyond uterus not to pelvic side wall or upper1/3 vagina		A	Vaginal involvement without parametral	
		B	Parametral involvement	chemoradiation
Stage III. Tumor extends to pelvic side wall or lower 1/3 of vagina		A	Vaginal involvement no extension to side wall	
		B	Extension to pelvic side wall or hydronephrosis	
Stage IV. Extension beyond true pelvis into mucosa of bladder and rectum.		A	Extension into pelvic organs	
		B	Distant metastasis	Palliative chemoradiation

Treatment, out-come and toxicity.

Treatment options available are surgery (radical hysterectomy), radiotherapy and chemotherapy (16). Patients with early stages of ICC can be managed surgically plus or minus radiotherapy depending on whether the nodes are positive for cancer. Surgery is a preferred treatment in the young women because it allows the preservation of both sexual and ovarian functions (5, 15,16). This becomes important in our setting given that ICC is being diagnosed at an earlier age than the resource rich countries. However surgical treatment is very limited in Kenya because at diagnosis only 10% of the patients are operable (stage IIA and below).Women with stage IA, IB and IIA at KNH are primarily managed surgically, while stage IIB and above are referred for the radiotherapy

treatment. Majority of the patients are treated with radiotherapy (4, 5).World-wide the current standard treatment of advanced cancer of the cervix is chemo-radiation.

In 1990 a study at KNH reviewed treatment outcome of patients with ICC and found to be poor, >90%of the patients presented with stage III and above(4) . Partly because the patients present late with advanced bulky tumor and poor histological grades. In an American study the 5 year survival rates for the various stages were ; stage I = 5 year survival rates of 77 - 84%, stage II = 54 - 67%, stage III = 13 - 40% and stage IV = 5 - 13% (15).

In a study done at KNH radiotherapy department, HIV infection was shown to reduce survival by about 9.2 months (5). Highly active anti-retroviral therapy (HAART) has been shown to cause regression of the premalignant lesions (5, 9).

Radiotherapy requires both external beam and intracavitary irradiation. The intracavitary pelvic dosage delivered is to ensure tumoricidal dosages to the cervix, broad ligaments and the lateral pelvic wall (5, 16). The goal of external irradiation is to sterilize metastatic disease of the pelvic lymph nodes and parametria and to decrease the size of the cervix to allow optimal placement of intracavitary radioactive sources. At KNH brachytherapy machine was not functional during the study period; this is associated with more pelvic failures.

Chemoradiation

Combination of radiotherapy and chemotherapy (chemoradiation) has demonstrated an improvement in the outcome in cancer of the cervix treatment. The rationale of combining chemotherapy and radiation is to eradicate systemic microscopic metastases which are not treated by local radiation. Women with stage IIB and above on pelvic radiotherapy alone, 35 %– 90% showed disease progress (failure to control disease). (17, 18, 19, 20).

Combined chemotherapy and radiotherapy have better outcome than radiotherapy alone. More than four studies reviews demonstrated increased survival with combined chemotherapy and DXT (19, 20, 21 and 22). An estimated 80% of women in stage IB to IVA had a four year progress free after chemoradiation compared to that of 30% in the DXT only group (19, 20). There was no significant increase in acute and late toxicity in the concurrent therapy group compared to DXT only group (21, 22, 23).

Adjuvant chemotherapy is given before radiotherapy. Whether weekly cisplatin or other platinum based regimes are the optimal choice remains to be determined. Cisplatin alone has less toxicity compared to combination chemotherapy (18, 21, 23). Cisplatin is thought to augment the effect of radiation by inhibiting the repair of radio-sensitized- induced sub lethal damage and by

sensitizing hypoxic cells to radiation. Because of the cytotoxic effect the drug reduces the bulk of the tumor, which leads to re-oxygenation of the tumor with entry into the radio-sensitive phase (19, 20, 21). Other chemotherapeutic agents used for treatment of cancer of the cervix are Paclitaxel, fluorouracil and Ifosfamide (17).

Toxicities

Factors affecting morbidity from radiotherapy include, dose of radiation, fractionation, quality of intracavitary insertions, type of applicators used, proportion of external beam to Brachytherapy dose delivered, combination therapy and duration of treatment(22, 24). Host factors affecting the radiotherapy for ICC include: Patient's age, tumor bulk, other co-morbidity, (i.e. diabetes mellitus and HIV)(16). Anemia is thought to make the tumor radio-resistant (27, 28). Pelvic failure is when there is recurrent tumor 2 years after radiotherapy. (17).

Radiotherapy induces acute toxicity (within 3 months from treatment initiation) and late morbidity from radiotherapy. Acute toxicity includes skin reaction such as erythema or even severe burns, cystitis, and proctitis (26, 27, 28). Brachytherapy has a dose dependent effect on the gastrointestinal of (proctitis). Leucopenia, thrombocytopenia, nausea and vomiting, are effects that are augmented by multiple cytotoxics. Hematological toxicities are often reversible (25, 26). Diarrhea can be relieved by giving the patients Loperamide or Atropine sulphate (lomotil). Enemas with low dose steroid will relieve proctitis. Dysuria, frequency and nocturia are often a result of cystourethritis. (25). Anti-spasmodics are helpful in relieving the symptoms. Skin erythema and burn effects are relieved by proper perineal hygiene and application of topical lotion (25, 26).

Late toxicities (after 3 months since initiation radiotherapy) include chronic diarrhea due to damage of the rectum with (QLE- consequential late effect), small bowel obstruction, malabsorptions, dyspereunia from vaginal adhesions and fibrosis (25, 26). Severe rectal and bladder injury could lead to recto-vaginal fistula and Hemorrhagic cystitis warranting a colostomy and urinary diversion respectively (25, 26, 27).

STUDY JUSTIFICATION

Cancer of the cervix is one of the commonest cancers affecting women in our country. In a year, an average of 350-400 patients are seen in Kenyatta national Hospital with advanced cancer of the cervix in the radiotherapy department. Until early 2006 radiotherapy has been the only modality of treatment of advanced carcinoma of the cervix in our set up.

Radiotherapy as a treatment has an attendant morbidity and mortality as well as the advanced cancer on its own. Rogo et al (4) in their study sixteen years ago in reviewing cancer of the cervix in

general, found that 46% of deaths in acute gynaecology ward were due to complications of cancer of the cervix. No specific study has been done to describe the outcome of patients with advanced carcinoma of the cervix treated with radiotherapy in our set up.

This study sought to evaluate the outcome of patients with advanced carcinoma of the cervix treated with radiotherapy in Kenyatta National Hospital .Quantifying the toxicities and the quality of life among patients who have undergone treatment , will aid in objectively counseling the patients at the initiation of therapy. Target areas for improvement were to be identified.

OBJECTIVE

Primary objectives.

Describe clinical outcome, toxicities and follow-up for women with histological diagnosis of cervical carcinoma post radiotherapy at Kenyatta National Hospital.

. Specific objectives

1. Describe socio-demographic characteristics of women diagnosed with ICC at KNH.
2. Describe histological types and impact on treatment outcome
3. Describe the morbidities associated with radiotherapy
4. To look at the follow-up of women after radiotherapy.

METHODOLOGY

Study design.

This was a retrospective descriptive study.

Methodology

306 out of 1545 files of cancer of the cervix seen over a 5 year period covering 2000 to 2004 were picked from a sampling frame where every fifth number was taken. The sample size was determined using Fischer et al formula of 1986 to determine the minimum sample size as calculated below. The files of the patients were then retrieved from the radiotherapy records department of Kenyatta national Hospital and the data collected using a data collection sheet.

Data collection.

The investigator collected data from all the case files that met inclusion criteria.

Data collection sheet containing social demographic and medical data were filled. The cases are those patients who had been diagnosed with ICC. The diagnosis was based on the histological evaluation. The clinical findings at examination under anaesthesia, at surgery, during and after radiotherapy +/- chemotherapy were filled in.

Sample size determination.

The sample size was calculated using Fistcher et al formula of 1986.

$$N = \frac{Z^2 Pq}{d^2}$$

Where

N= the desired sample size

z- The standard normal deviate usually at 1.96 which correspond to 95% confidence.

p- General complication rates of radiotherapy is usually 20%

Q =1 – p

d – Degree of accuracy with which p is determined set at 0.05

$$\frac{1.96 \times 1.96 \times 0.2 \times (1-0.2)}{0.05 \times 0.05}$$

Therefore n=195

Sampling procedure.

Proportionate sampling was done after deciding on the sample size using the formula above.

A sampling frame was made of all the cancer of the cervix patients treated in the unit from 2000 January to 2004 December (total of1545). The sampling frame was arranged according to when they presented.

Samples selected systematically to the number that presented each year.

Selection was systematic and every other 5th file was selected. A total of 305 files were picked.

Data analysis.

The completed data sheets were verified and then coded for computer analysis .The analysis was done using the SPSS/PC computer package 11.0 version and analyzed by frequency table cross tabulation. For overall survival all the time was taken from time of registration to the time when last seen in the radiotherapy unit. All patients who were lost to follow-up were considered diseased and dead regardless of their status. Different demographic tumor related and treatment related factors were evaluated for out-come.

Inclusion criteria.

All the patients must have had a histological confirmed diagnosis of cancer of the cervix.

ETHICAL CONSIDERATIONS.

The permission to carry out the study was sought from the ethical and research committee of Kenyatta National Hospital.

STUDY LIMITATIONS

1. Patients who have cancer of the cervix and do not end up in radiotherapy department were not captured in this study (ie those who were in the wards).
2. Being retrospective study, there was a lot of missing information.
3. Incomplete documentation of patients follow-up status
4. Lack of consistent ways of grading toxicity in our facility (Common toxicity criteria by RCOG) was not used. .
5. Not all the patients are adequately staged and some tend to migrate to other stages due to the delays of referrals or within the hospital

RADIOTHERAPY TREATMENT.

All the patients reported in this study received EBRT using cobalt 60 (siemens or Thetatrons T ^{280}) machine, via parallel-opposed anterior and posterior fields (AP/PA). The field sizes were adopted depending on the FIGO clinical staging of the disease. Most of the patients received a dose of 40-50 grays to point A. Point A being a reference location 2 cm lateral and 2 cm superior to the cervical os. Fractionation was 1.8-2.0 Gy tumor dose daily, 5 fractions per week within 5 weeks with two days rest from the treatment during the weekend. Negligible number of patients received brachytherapy, the machine was no functional. Once EBRT was initiated patients were seen after completion, unless where toxicity effects lead to discontinuation of therapy. Hence forth they were

seen after 3-6 months in the radiotherapy department. Documentation of acute morbidity (GIT, skin, and GUT) and pelvic tumor control was done.

STUDY SITE.

The study was carried out at KNH, Nairobi, Kenya radiotherapy department. KNH is a teaching and referral hospital with abed capacity of 2000 patients. This is the largest radiotherapy unit in the country. KNH has two telecobalt machines (Siemens and Theraton T^{280}) for teletherapy; Amersham low dose rate caesium-137 remote after loading brachytherapy equipment, two cervixfix apparatus and one stimulator. The brachytherapy machine was not functional during the study period. Most of the patients treated for cancer of he cervix are referred from other hospitals. Patients with terminal disease are treated then referred to the hospice for terminal care.

RESULTS

Table 1: Sociodemographic Characteristics of study population

Characteristics	**Proportion** n/N(%)
Age n=306 mean (SD)	48.6(+/- 11.8)
Parity n=256 mean(SD)	5.9 (+/-2.9)
Marital status n=306	
Married	*151(49.3)*
Single	*17(5.6)*
Divorced/separated	*10(3.3)*
Not recorded	*128(41.8)*

Table 1 shows the sociodemographic characteristic of the of the study population.

The mean age of the study population was 48.6 years with a median of 48 and a standard deviation of 11.8 years. The youngest study subject was 20 years and the oldest was 80 years. Majority 151 (49.3%) of study subjects were married. However, 128 (41.8%) of the study subjects did not have their marital status indicated in records. The mean and SD of their parity was 5.9(+/-2.9). There was lack of documentation of other sociodemographic and reproductive variable in the patients' files.

Table 2: Referring hospitals to the radiotherapy department of KNH

Referred from (n=306)	**N (%)**
District Hospital	*53 (17.3%)*
Provincial Hospital	*45 (14.7%)*
Mission hospital	*9 (2.9%)*
Private Hospital	*145 (47.4%)*
KNH diagnosed	*17 (5.6%)*
National referral Hospital	*6 (2.0%)*
Not in record	*31 (10.1%)*

Table 2 depicts the health facilities that referred the patients to KNH. Referrals from the private hospitals were 145 (47.4%) these were the largest group among the referrals. Almost all the patients were referred by medical officers (96%).

Table 3 subjects clinical, histological characteristics and treatment

Characteristic	**Proportions**
FIGO clinical stage n=299	
Stage I	*10(3.3%)*
Stage II	*124(41.5%)*
Stage III	*142(47.5%)*
Stage IV	*23(7.7%)*
Stage IIB and above	271(90.7%)
Histological cell types n=299	
Squamous cell carcinoma	*266(86.9%)*
Adenocarcinoma	*17(5.6%)*
Others	*14(5.2%)*
Poorly differentiated n=299	167(54.9%)
Earliest hemoglobin g/dl	10.8+/-2.4
HIV Status n=306	
Positive	*16(5.2%)*
Negative	*40(13.1%)*
Unknown	*250(81.7%)*
Duration of symptoms at initiation or EBRT in months	10.8+/-13.7
Radiation dose(EBRT) prescribed in Grays n=258	49.85+/-8.5
Radiation dose(EBRT) received in Grays n=256	45.7+/-15.1
Completed EBRT	258(84.3%)

Table 3 shows the stages which the patients presented at, histological classification and the doses of DXT they received. Mean duration of symptoms was 10.8 months. Majority of the study subjects had FIGO stage III 142(47.5). Stage IIB and above comprised 90.7%. Squamous cell carcinoma comprised more than 266(86.9). Poorly differentiated grades comprised of 167(54.9). Majority of the patients HIV status was unknown. Out of the total 299 successfully analysed for treatment, 258 (86.3%) received the prescribed treatment. The mean haemoglobin level was 10.8 (SD+/- 2.4)

Figure 1: Place of residence

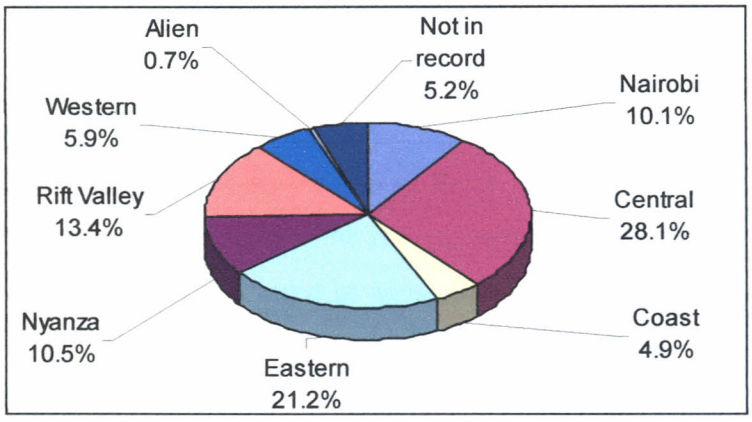

Figure 1: Home province (n=306

As shown in Figure 3, majority of study subjects 86 (28.1%) were residents of Central Province, followed by Eastern 65 (21.2%). The five top districts were Kiambu 20 (6.5%), Nakuru 20 (6.5%), Nyeri 19 (6.2%), Machakos 18 (5.9%) and Thika 13 (4.2%). No resident of North Eastern Province was treated for cancer of cervix in the sample and period covered by the study

Table 4: Proportions of those with morbidity by time (n= 287)

Toxicities n(%)	**3 months**	**6 months**	**12 months**	**24 months**
skin(burns and fibrosis)	135(44%)	143(47%)	137(45%)	123(40%)
gastrointestinal diarrhoea and proctitis)	122(40%)	181(59%)	107(35%)	78(26%)
haematological / transfusion	72(23%)	89(29%)	91(30%)	82(27%)
Genitourinary (dysuria, cystitis)	42(14%)	40(13%)	25(8%)	25(8%)
Rectal bleeding	28(9%)	31(10%)	32(11%)	26(9%)
Vaginal stenosis	17(6%)	20(7%)	23(8%)	24(8%)
evidence of recurrent/ persistent tumour	94(31%)	117(38%)	125(41%)	121(39%)

Table 4 shows the proportion of those with various morbidities over time. The morbidities that were seen in this study as per the records were skin fibrosis /burns, diarrhoea and cystitis / dysuria which at three months occurred in 44%, 40 % and 14% of the patient respectively and at year occurred in 45% , 35% and 8 % of the patients respectively. It is to be noted that these were cumulative figures and not necessarily the morbidities occurring in patient seen at follow up, the figures include patient seen for example from day one to three months or four to six months depending on consideration.

TABLE 6: Number of systems affected.

Number of Systems affected (toxicities)	**n**	**%**
None of the systems	57	(18.6)
One system	66	(21.6)
Two systems	89	(29.1)
Three or more systems	94	(30.7)

Table 6 shows the number of systems individual patients experienced toxicities. Most of the patients had 2 systems or more affected by radiation toxicities, 183(60%). Of the (18%) recorded to have had no toxicity 48 of them did not complete EBRT and so no follow-up data was available. This leaves as with only 9 (2.8%) who were followed up and sure had no toxicity whatsoever.

TABLE 7 Loss to follow-up according to the stage

	Loss to follow-up at 6 month	Loss to follow-up at 12 month	Loss to follow-up at 24 month	Loss to follow up at 60 month
Cancer staging (FIGO)	**n %**	**n %**	**n %**	**n %**
IB (n=10)	2(20)	4(40)	5(50)	8(80)
IIA (n=18)	3(17)	5(28)	8(45)	9(50)
IIB (n=106)	18(17)	26(25)	33(32)	78(74)
IIIA (n=43)	9(21)	11(25)	12(27)	42(97)
IIIB (n=99)	28(24)	33(29)	41(38)	94(95)
IVA (n=21)	5(50)	5(50)	-	-
IVB (n=2)	1(50)	1(50)	-	-
Total (n=306)	**145 (47%)**	**200 (66%)**	**254 (83%)**	**298 (97%)**

Table 7 shows the number of patient who were lost to follow-up with time. At 5 year follow-up almost all the patients in stage III and IV did not return, while 50% of stage IIA returned. Loss to follow-up became worse with the more advanced stage. It is worth noting that 42% of the patients did not return at all after completing radiotherapy.

Table 8. Severity of morbidity (no of systems affected) by age, parity, hb, duration of symptoms at presentation, duration of radiotherapy and dose of radiotherapy.

	No system affected	One system	Two systems	Three and above	P value
Age years (Mean/SD)	47.79 (+/-11.9)	49.23 (+/-12.9)	47.94 (+/-11.7)	49.21 (+/-11.1)	0.807
Parity (Mean/SD)	5.8(+/-2.9)	6.1(+/-2.8)	5.9(+/-3.0)	5.7(+/-2.8)	0.900
Earliest HB (Mean/SD)	10.5(+/-2.4)	10.5(+/-2.6)	11.2(+/-2.3)	10.9(+/-2.2)	0.241
Duration of symptoms at presentation (months) (Mean/SD)	9.49(+/-5.7)	10.4 (+/-14.5)	12.7 (+/-14.6)	10.8 (+/-15.8)	0.684
Duration of radiotherapy (days) (Mean/SD)	27.7(+/-17.3)	31.8 (+/-11.1)	34.9 (+/-12.7)	43.1 (+/-21.7)	0.000
Radiation dose in grays(Mean/SD)	34.7(+/-22.2)	43.8 (+/-12.4)	46.2 (+/-11.5)	52.1 (+/-11.9)	0.000

Table 8. Shows the association between the number of systems affected and the parity, age, HB, duration of symptoms at time oe EBRT initiation, the dosage of radiation received and the duration of the radiotherapy treatment. In the analysis of the association by the analysis of variance, Age (p, 0.807) , parity (p, 0.900), earliest HB(p,0.241) and duration of symptoms before initiation of therapy(p,0.684) had no association of statistical significance with the number of systems affected. However duration and dosage of radiotherapy were significantly associated with the number of systems affected(p,<0.005)

Table 9. Severity of morbidity by histology, grade and seropositivity

	No system affected N (%)	**One system affected N(%)**	**Two systems affected N(%)**	**Three and above N(%)**	**P value**
Histology					0.212
Squamous	45(81.8)	61(92.4)	77(87.5)	83(92.2)	
Adeno+others	10(18.2)	5 (7.6)	11(12.5)	7 (7.8)	
Grade(differentiation)					
well diff	13(23.6)	13(19.6)	21(24.4)	18(20.0)	0.888
mod diff	14(25.5)	16(24.4)	22(26.5)	15(16.7)	
poorly diff	18(32.7)	25(37.9)	31(36.0)	40(44.4)	
others	10(18.2)	12(18.1)	12 (13.1)	17(18.9)	
HIV test					0.974
Positive	4(7)	3(4.5)	5(5.6)	4 (4.3)	
negative	7(12.3)	7(10.6)	13(14.6)	13(13.8)	
unknown	46(80.7)	56(84.9)	71(79.8)	77(81.9)	

Table 9 demonstrates the part played by the histology, grade and HIV serostatus on the severity of morbidity as denoted by the number of systems affected. There was no significant correlation between the histological type (p, 0.212), grade of the cancer (p, .888) and the HIV serostatus (p, 0.974) and the number of systems affected by the radiotherapy treatment.

Table 10. Loss to follow up by province of origin

	Distribution (overall)(%)	N(%)at 6/12	N(%) 12 months	P value	OR 95%(CI)
Nairobi	31(10.1)	10(9.5)	6(12.8)	ref	ref
Central	86(28.1)	32(30.5)	11(23.4)	0.372	0.573(0.169-1.945)
Eastern	65(21.2)	18(17.1)	13(27.7)	0.769	1.204(0.349-4.152)
Rift valley	41(13.4)	17(16.2)	6 (12.8)	0.449	0.588(0.149-2.326)
Nyanza	32(10.5)	12(11.4)	5(10.6)	0.623	0.694(0.162-2.971)
Western	18(5.9)	5(4.8)	3(6.4)	1	1(0.173-5.772)
Coast	15(4.9)	8(7.6)	1(2.1)	0.184	0.208(0.021-2.103)
Uknown	17(5.8)	3(2.9)	2(4.2)	0.323	-
TOTAL	306(100%)	104(100)	47(100)		

Table 10 depicts how the province of origin influenced loss to follow up at 6 months and at one year. With Nairobi as the point of references, there was no demonstrated significance in loss to follow up as concerns the province of origin from any of the provinces, by 6 month 32% of patient had been lost to follow up up from central compared to 27.8% from western province at the same month. The number from central constituted 30.5% of those lost to follow up at 6 months while from western it constituted just 4.8%. The OR for central was 0.588 while western was 1. Western in comparison to Nairobi is nearer significance in regard to OR but the p value is 1, making it non contributor in regard to influence. This meqans distance may not be the only factor to contribute to loss of follow up, there could be other factors as well.

DISCUSSION

A total of 1545 women were referred for treatment at KNH radiotherapy department from 2000 to 2004 with an annual average of 300-350. Most of the patients were from districts not too far away from Nairobi. Central and Eastern provinces were the highest represented (28.1%, 21.2%). Only 5.9% were from western province and none from north eastern. This could be due to the distance, cost and detachment from social support that has to be considered by the patient and her family. It is estimated that 3600 women die from cancer of the cervix yearly in Kenya (4, 5). This leaves out more half of woman who requires care at this specialized centre.

The average age at diagnosis was 48 years in this study. This is almost similar to the age found in the American population of age 50. A study done 16 years ago (Rogo et al) reported a similar age of presentation, therefore has been no change in our age of presentation as seen in this study (4).

A majority of the referral were made to the unit by doctors. It is not clear why a significant propotion of the patients were referred from private 145(47.4%). One would expect the public district and provincial hospitals to have a bigger propotion but they accounted for 98(31%) of the referrals. This may suggest that a greater number of women with cancer of the cervix are not able to acces care due to financial reason. Those who could pay and see a doctor could have been the only ones who were able to travel to KNH for treatment.

More than 50% of the patients at index presentation were at stage II (41.5%) and stage III (47.5%).This has changed from what done in earlier studies (Rogo et al 1990) at KNH about 80% of the patients presenting at stage III (4). This improvement in patient presenting relatively in earlier disease stages than before could be attributed to the improvement in the health care system. This compares well with an Indian study where stage IIB (33.7%) stage IIIB (21.8%), but differs with the findings of stage IB (26.6%). This could be a population where there is better access to services as far as cancer of the cervix is concerned (28). Squamous cell carcinoma is still the most common histological type of cancer of the cervix at the prevalent rate of (85%); this is not different from the other literature (15).

Patients who had received surgical treatment prior to radiotherapy were 35 (11.6%). Some of these patients belonged to stages that is essentially in-operable .This suggest disease progression may have

ccurred after surgery, or the patient was under-staged. The staging indicated is that done before being ent for radiotherapy treatment regardless of long ago that was to initiation of radiotherapy. .

)ccurrence of acute and late morbidities associated with treatment or disease progression was ignificantly higher than in other studies. The most frequent toxicity involved the gastrointestinal ract, having a peak of occurance at 6 months 143(47%).This is similar world wide with the GIT being affected more often than other systems. Vaginal stenosis was found in 20% at 6 months, this differs with report of a prospective study in the same unit by Kagema et al 2002(33) which found that almost all women had vaginal stenosis post radiotherapy at KNH. This difference could be as a result of clinicians not asking questions pertaining changes in sexual practices and no vaginal exams during the follow-up period. Rectal bleeding, incontinence of urine or stool, cystitis/ Dysuria, urgency and vaginal bleeding occurred in less than 20%. Comparable studies quote figures less than 5 % for most of these morbidities (Indian retrospective study, rectal bleeding 1.2%, vaginal stenosis 1.1%).This can be explained by the difference in treatment type (28). In this study patients only received EBRT while in other studies: EBRT, brachytherapy, and chemotherapy were used (Pederson et al 1994, Sood et al 2002, Wang et al 1998). Diarrhea and colitis was an acute morbidity that decreased after the first 3-6 months. The number of women with postradiational GI toxicities reduced from 122 (42.8%) at 3 months to 78 (27.4%) at 24 months. In a Perdesons study 1994 on 442 cervical cancer patients, 7.7% had early complications (29). Sood et al 2002 study, 4% of 24 patients treated with EBRT and Brachytherapy had grade 3-4 acute toxicity compared to 8.0% of 25 patients who in addition got chemotherapy. In all these studies GI toxicities was the most common early toxicity (30, 31, 32).

Of the women who returned at 3 months, pelvic failure or (residual tumour) was found in 94(32.6%) which was higher than in prospective study done in the same unit. Gichangi et al (2002) reported residual tumor rate to be about 19% at 4-7 months follow up. (5). Lanciano et al (1991) and Perez et al (1995) both reported a 5-year pelvic failure rate of 10-74% depending on the clinical stage .Majority of the patients who were followed up for a period had 2 systems affected by toxicities. This compares well with what Gichangi et al found in KNH. The number of systems affected had a significant statistical association with the radiation dosage and the duration of radiotherapy. This could be explained by the higher radiation exposure of the tissues leading to more damage to the gut , genitourinary and other surrounding tissues(15).

14. Constance Mao et al. The efficacy of Human Papillomavirus-16 Vaccine to prevent cervical intraepithelial neoplasia. Obstret and Gynecol 2006; 107:18-27

15. Christine H H. Premalignant and malignant lesions of the uterine cervix. Currents Obstetrics and Gynecologic, Diagnosis and treatment.9th Edition Author Alan H D and Lauren Nathan. 894-915.

16. Cannistra S A, Niloff J M. Cancer of the uterine. N Engl J Med 1996: 334; 1030-38.

17. Rose P G, Bundy B N, Watkins E B, et al. Concurrent cisplatin based radiotherapy and chemotherapy for locally advanced cervical cancer. N Engl J. Med 1999 340: 1144-53.

18. Keys H M, Bundy B N, Stehman F B, et al. Cisplatin, radiation and adjuvant hysterectomy compared with radiation and adjuvant hysterectomy for bulky stage IB cervical carcinoma. N Engl J Med. 1999, 340: 1154-1161.

19. Whitney W, Sause W, Bundy B N, et al. A randomized comparison of Fluorouracil plus cisplatin verses hydroxyurea as an adjuvant to radiation therapy in stage IIB-IVA carcinoma of the cervix with negative Para-aortic lymph nodes. J. Clin Oncol. 1999; 17:1339-48.

20. Morris M, Eifel PJ, Grigsby PW et al. Pelvic radiation with concurrent chemotherapy compared with pelvic and Para-aortic radiation for high-risk cervical cancer. N Engl J Med 1999; 340: 15:1137 -43

21. Eifel P J, Winter K, Morris M, et al. Pelvic irradiation with concurrent chemotherapy verses pelvic and Para-aortic irradiation with high risk cervical cancer an update of Radiation Therapy Oncology group training. (RTOG). J. Clin Oncol 2004; 22: 874-80.

22. Peters III WA, Liu PY, Barret II RJ, Stock RJ et al. Concurrent chemotherapy and pelvic radiation therapy compared with pelvic radiation therapy alone as adjuvant therapy after radical surgery in high-risk early stage cancer of the cervix. J Clin Oncol 2000; 18: 8: 1606-13.

23. M King C, McConkey T, N Latief et al. Improved survival after concurrent weekly cisplatin and radiotherapy for cervical carcinoma with assessment of Acute and late side effects. J Clin Oncol 2005 18: 38-45.

24. Peres C A, Grigsby D W, Locket M A, et al. Radiotherapy morbidity in cancer of the cervix. Int J Radio Oncol Bio Phys 1999: 44; 858-866.

25. Tan L T. Russell S, Burgess S. Acute toxicity of chemo-radiotherapy for cervical cancer. The Addenbrookes experience. Clin Oncol 2004: 16; 255-260.

26. Kirwan J M, Sydunds P, Green J A et al. A systematic review of acute and late toxicity of concomitant chemoradiation for cervical cancer. Radiother Oncol. 2003: 68; 2117-2600.
27. Trotti A, Byhardt R, Stetz J, Gwede C et al. Common Toxicity criteria. Version 2.0. An improved reference for grading the acute effects of cancer treatment: impact on radiotherapy. Int J Rad Oncol Bio Phys 2000; 47: 13- 47.
28. E. P Sainabishkumar, F D Patel, and S C Sharma. Result of radiotherapy alone in the treatment of carcinoma of the uterine cervix; a retrospective analysis of 1069 patients India. Int J Gynecol Cancer 2005; 15 890-897.
29. Pederson D, Bentzen SM, Overgaard J. Early and late radiotherapeutic morbidity in 442 consecutive patients with locally advanced carcinoma of the uterine cervix. Int J Radiat Oncol Biol Phys 1994; 29: 941-952
30. Wang CJ, Leung SW, Chen HC et al. The correlation of acute toxicity and late injury in radiotherapy for the cervical carcinoma: evidence suggestive of consequential late effect. Int J Radiat Oncol Biol Phys 1998; 40:85-91
31. Simonds P, Sinistero G, Zola P et al. Complication of uterine cervix carcinoma treatment. The problem of a uniform classification. Radiother Oncol 1989; 14: 9-17
32. Perez C A, Grigsby D W, Casto-Vista H, et al. Carcinoma of the uterine cervix, impact of prolongation of overall treatment time and timing of Brachytherapy on outcome of radiational therapy. Int J Rad Oncol Bio Phys1995: 32; 1275-1288.

VDRL: -ve

HIV: -ve

Hb: 9.0 g/dl

2 injections of Tetanus Toxoid were given during her ante-natal visits.

Family and Social History

She is a housewife married to a transport officer. There is no family history of any chronic illness. She doesn't smoke cigarettes or drink alcohol.

Physical examination

C.A was in fair general condition and was not pale or febrile.

Abdominal examination

The abdomen was uniformly distended. The fundal height was term, the lie longitudinal, the presentation cephalic and the descent 5/5.

Vaginal examination

The external genitalia were normal, the vagina warm and moist. The cervix was 1 cm long and the cervical os was closed and posterior. The pelvis was adequate.

Clinical Impression

Post- term pregnacy in a primigravida.

Plan of management

C.A. was admitted for delivery. She was counseled on the diagnosis and the plan of management. Prostaglandin E_{2a} pessary was inserted in the posterior fornix for cervical ripening at 8am. The fetal heart rate was reassuring , the descent was still 5/5, the cervical os was closed central and soft. She was monitored closely and noted to be having contractions 8 hours after the initiation of induction.

At 4pm she was having mild contractions found to be 3 cm dilated with a regular fetal heart. ARM was done (clear liquor obtained) and Oxytocin at 5 IU was started.

At 7 pm she was getting 2 moderate contractions in 10 minutes.

At 9pm she was getting 3 moderate contractions in 10 minutes.

At 10pm she was now getting 3 strong contractions in 10 minutes.

At 11pm she delivered by Spontaneous Vertex Delivery (SVD) a live male infant who weighed 3400g and on the Agar scale scored 8/1, 9/5, and 10/10.

The placenta and membranes were extracted complete. A small perineal tear was repaired.

Post-natal care

After delivery she was transferred to ward GFA a post-natal ward.

On the first postnatal day she was stable and was in good general condition. She was not pale or febrile.

The uterus was well contracted with a fundal height corresponding to a gestation of 14 weeks. The breasts were active and non-tender. The calf muscles were non-tender and the lochia loss was normal and not foul smelling.

She was therefore discharged home. She was advised to come to the post-natal clinic in 6 weeks.

DISCUSSION OF POST TERM PREGNAMCY

Post-=term pregnancy is defined as a pregnancy that is 42 completed weeks (294 days) or more from the first day of the last menstrual cycle. (Synonyms; Post –dates, Post mature, Prolonged pregnancies Post -term)(1, 2).

It is impossible to get the true incidence of post term pregnancies. Published rates range from 14.9% for the Norwegian women with registered menstrual date to 2.2 for women with singleton pregnancies without fetal abnormalities whose dates were based on mid trimester ultrasound (2). Local figures include incidence of 10% in pumwani and one of 4.9% in study done in Kenyatta (3). It thought that the true incidence world wide is dropping due to the increase in elective delivery for various reasons and the effect of premature deliveries. Some of the pregnancies may have wrong dates and so wrong estimation (3, 4).

The causes of post term pregnancies are unknown but there are some associations. Low estrogen level in pregnancy, anencephaly, placental sulphatase deficiency, fetal adrenal hypoplasia, absence of fetal pituitary are associated with post term pregnancies (1,2). Post term pregnancies tend to repeat themselves, in a woman with a previous history of post-term pregnancy there is a relative risk

of 2.2 of this repeating itself in the next pregnancy. Daughters of mothers who went post term had a greater chance of having it also(2).

Confirmation of the dates is essential for proper diagnosis. The last menstrual period is used but it may be incorrect where menstrual cycles have been irregular or the use of oral contraception with effect of withdrawal bleeding. Earliest pregnancy test: it ought to have been within the first 6 weeks from the last menstrual cycle, if 36 weeks have elapsed since that pregnancy test the baby is presumed to be mature(4,5,6). Quickening occurs at 18- 20 weeks in primigravida and 16 – 18 weeks for multiparous, therefore 20 weeks after quickening the baby is presumed mature. If 20 weeks have elapsed since the first audible tones on feto-scope then the baby is mature. Ultrasound dating varies with gestation. Crown ramp length when done 12-30 weeks has an error of +/- 1 week, at 20-30 weeks of +/- 2 weeks, and after 30 weeks +/- 3 weeks. Measurements of biparietal diameters and femoral length in the second trimester are more accurate in predicting fetal maturity than menstrual dates even when these are reliable. When fundal height estimation is done between 18-30 weeks when the bladder is empty, this corresponds to the real fetal maturity(4,5).

The risk of un-explained still births at 41 weeks and beyond is higher than other periods of pregnancy. In modern obstetrics women with diabetes, pre-eclampsia, twins, advanced maternal age, previous perinatal mortality and recurrent APH, are likely to be delivered between 37-41 weeks(5,7). So women allowed to reach 42 weeks represent a low risk group yet the rate of unexplained still births is higher at this gestation. Placental aging and uteroplacental insufficiency may explain the Intrauterine growth restriction (IUGR) observed. Post term babies who are small for age have a higher risk of stillbirth compared to those who are large(7). However most babies continue to gain weight and the challenge becomes birth dystocia, shoulder dystocia and cephalopelvic disproportion, increased rate of obstetric trauma and cesarean section. Other conditions associated with post-term pregnancies are: neonatal encephalopathy, cerebral palsy, Meconium stained amniotic fluid, and fetal distress(7, 8, 9).

The management is controversial, but there is a consensus that given the complications associated with post term pregnancies they should not be allowed to progress beyond 42 weeks(1,2,8). Options include routine induction at term to prevent pregnancies reaching 42 weeks, routine induction at 42 weeks or shortly before then and selective induction at 42 weeks of cases thought to be at risk of adverse outcome. Routine induction of labour at 41 weeks is associated with reduced perinatal mortality with no increase in cesarean section rates regardless of parity, method of induction or

ambient cesarean section rates(7). As far as women views on induction of post term pregnancies only few 5% opted for expectant management after 42 weeks.

Fetal surveillance is key when expectant management group. These includes: ultrasound assessment of amniotic fluid (amniotic fluid index should be more than 8 cm), Cardiotocography (reactive fetal heart rate tracing are reassuring), Fetal movements counting (10 charted counts in 12 hours are acceptable), biophysical profile. Oligohydroamnos occurring in post term pregnancies increases the risk of poor outcome. These leads to increased risk of cord compression, and any release of Meconium produces a thick, viscous fluid leading to Meconium aspiration syndrome (7,8,9).

Once the decision to deliver is made the date for delivery is dependent on individual case. If the cervix is unripe (poor bishop score) then ripening of the cervix is done by use of prostaglandins preparation that are readily available (2). When the cervix is favorable then sweeping of the lower uterine segment and subsequent amniotomy is done and labour was induced with oxytocic agents (1,2). Once induction is done then there is need for close intrapartum monitoring to recognize fetal complications and manage then accordingly on time. Such may be Meconium release, macrosomia, fetal intolerance to labour with abnormal fetal heart rates (7, 8, 9).

REFERENCES

1. Cunningham FG, Norman FG, Leveron et al . Post term pregnancies in William Obstetrics 21st Edition 2001. pg 730-741
2. Crowley P. Prolonged pregnancy. In Turnbulls Obstetrics 3rd Edition 2001 chap 32 pp 521-531.
3. Elfenesh B Post date pregnancy and fetal outcome. Mmed Thesis University of Nairobi 1998.
4. Phelan JP, Myong OA, Eden RE et al. Postdatism clinical Obstet and Gynecology 1989: 32(2)
5. Paul T Walkes. Post date pregnancies E medicine Aug 8 2002
6. Pernol ML. Late pregnancy complication. In Current Obstetrics and Gynecology Diagnosis and treatment. 8th edition 15; 338-339
7. Mannino F: Neonatal complication of post term gestation. J Report med 1988 march :33 (3) 27(1-6)
8. Levaron KJ, Quinn JG Cunningham FJ et al. Prolonged pregnancy obstetric concerns that cause fetal distress. AMJ Obstet and Gynecology 150 : 465, 1984
9. Crowley P. Intervention for preventing or improving outcome of delay at or beyond term. Cochrane Data base syst rev 2000: (2) CD 000170

Obstetric 2

PLACENTA PREVIA TYPE 1V- EMERGENCY C/S PREMATURE BABY.

NAME; R.M. 1PNO. 1102600

AGE; 27YEARS LMP: 29/11/05

DOA 2/06/06 EDD: 6/09/06

DOD 29/07/06 GESTATION 28 WEEKS

PARITY: 1+O

Presenting complain

She presented with history of painless vaginal bleeding 4 hours prior to admission.

History of presenting illness

She was well till the evening of admission when she noticed that she had developed vaginal bleeding. Had changed pad 4 times that were fully soaked. There was no history of trauma or coitus prior to the onset of the bleeding. She had not experienced any other bleeding episode prior to this. There was no history of per vaginal discharge or drainage of liquor.

Past medical history

Nothing of significance was noted on the past medical history.

History of current pregnancy

Her last menstrual period was on the 29th of November 2005 and her expected date of delivery on 6th of August 2006.She had not started antenatal care, all the profiles were done in during her hospitalization.

Past obstetric and gynecologic history

She was a Para 1+0.The first delivery was a vaginal birth to a live female infant at term who was borne on the way to hospital in 2001. It was un-eventful pregnancy with no ante partum or postpartum haemorrhage reported.

She attained menarche at 15 years. Prior to conception her menstrual flow was for 3 days in a regular 28 days cycle. Had been on DMPA ( ) after the last delivery, discontinued after 3 years so as to conceive.

Family social history

She was married, schooled upto 4th form level. Stays in Embakasi with the spouse who is a mechanical technician. She neither smoked cigarettes nor drank alcohol.

PHYSICAL EXAMINATION

She was in good general condition, was not pale and had no oedema. She had a blood pressure of 130/70 mmhg, a pulse of 88 /m, a respiratory rate of 20/m and a temperature of 37.1^0C.

Systemic examination

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

Abdominal examination.

The abdomen was uniformly distended and moving with respiration. The liver and spleen were not palpable. The fundal height corresponded to 28 weeks gestation (which was appropriate for the dates).The uterus was not tender and no obvious contraction was observed. The foetus was in longitudinal lie cephalic presentation and foetal tones were heard and regular at a rate of 148 beats per meet.

Speculum examination.

She had a normal external examination with a lightly soaked sanitary pad. The vulva was blood stained but had no active bleeding from the vagina. There was a small blood clot in the vagina, the vaginal walls and the cervix appeared healthy. The cervix was closed and no bleeding was seen from the cervical os.

DIAGNOSIS

A diagnosis of antepartum haemorrhage at 28 weeks was made.

MANAGEMENT.

An intravenous line was secured using a gauge 16 intravenous canula. A blood sample was taken for grouping and cross-match and 2 units of blood ordered. An urgent obstetric scan was done which revealed a single intrauterine pregnancy at 27 weeks and 5 days. The placenta was posterior

low laying and completely covering the internal os. Fetal somatic activity was normal with a regular fetal heart rate of 158b/m. conclusion of placenta previa type IV.

Patient was transferred to the admitting antenatal wards for conservative management of placenta previa. Counseling was done for the patient to appreciate the need for hospitalization and that it will be a long stay. Patient was put on bed rest and close observation on a pad, to inform the nursing staff ion case of any bleeding. She was put on haematinics and intramuscular dexamethasone to be repeated 2 weekly. Blood for grouping and crosshatch was renewed weekly and ordered 2 units of blood to be ready incase of any bleeding.

Other investigations done.

Complete blood count. (5/6/06)

WBC	5.49
NEUT	3.54
LYMP	1.5
HGB	9.63g/dl
Repeat HGB (20/07/06)	9.94g/dl

VDRL Negative. HIV Negative. Blood Group A Positive.

She remained stable till at 35 weeks when she developed heavy per vaginal bleeding. She was transferred to labour ward where she was started on intravenous normal saline. An emergency theatre list was made then intramuscular 0.6mg of Atropine was administered for premedication just before being taken to theatre. Two units of blood were availed as patient was being wheeled to theatre.

Operative management.

She was noted to have brisk vaginal bleeding. The abdomen was cleaned and draped. General anaesthesia was induced and the abdomen opened through a sub-umbilical lower mid-line incision. A lower uterine segment caesarian section was performed. The uterine incision cut through the upper part of the body of the placenta. A live male dyspneic infant who had an apgar score of 8 in 1 minute and 9 in 5 minutes and a weight of 2300 grams. Received by the pediatrician who admitted

him to New Born Unit. The placenta was found to be covering the lower uterine segment covering the cervical os completely. The placenta was removed with ease (no placenta acreta encountered). The uterus was closed in layers and hemostasis achieved. Abdomen closed in layers after correct swab and instrument count. Vulvo-vaginal toilet was done uterus found to be well contracted and patient successfully reserved hemodynamically stable. The estimated blood loss of 700ml. The baby was shown to the mother then taken

Postoperative care.

She was observed continuously in the recovery room till fully awake the hourly for 6 hours and there after 4 hourly. She was put on intravenous penicillin G 2 muMU, 6 hourly, gentamycin 80 mg 8hourly and intramuscular pethidine for analgesia for 24 hours then oral diclofenac.

Baby remained dyspneic and died after 1 week due to respiratory distress syndrome of the new born.

Mother had uneventful post operative period. Counselling was done on the passing of the infant. She was advised to wear a tight fitting brazier and bromocriptine prescribed. She was discharged home on the 8th post-operative day to be seen in the post natal clinic in 6 weeks.

Post natal clinic

She is yet to be reviewed in the post natal clinic.

DISCUSSION.

The patient presented was a Para 1 +0 admitted with placenta previa type IV at 28 weeks gestation. She was managed conservatively until at 35 weeks when an emergency caesarian section was done following heavy vaginal bleeding. She delivered a live male infant weighing 2300 grams who succumbed in new born unit due to respiratory distress syndrome of the new born.

Placenta previa involves the abnormal implantation of the placenta on the lower uterine segment(1,2). Variants include complete implantation over the cervical os (total placenta previa); a placental edge covering the os (partial PP); or the placenta approaching the boarder of the cervical os (marginal PP); when the placenta implants in the caudad one half to one third of the uterus or 2-3 cm from the cervical os (low lying placenta)(1,2).

Advances in ultra-sonography imaging have enabled better definition of relationships between the placental margin and the internal cervical os, leading to a more exact definition of the types and location of PP(2,3,4). Some authors prefer classifying PP as complete or incomplete because of the inability to precisely classify in the absence of cervical dilatation. Complete being the body of the placenta covering the cervical os at the time of delivery and incomplete comprise of the remainder (marginal, partial according to the traditional classification)(1,2,3). In our case the patient had complete placenta previa. Several studies on placenta previa suggest that complete PP may be an entirely different clinical entity to incomplete PP with worsening maternal and perinatal outcome (2).

The incidence PP is 1: 200 (0.5% of births)(1, 3). Varying incidences have been reported in different studies done 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, 7).

There are factors that seem to increase the risk of PP. these include advancing maternal age, multiparity, smoking, cocaine use, prior PP, one or more caesarean births and prior suction curettage for spontaneous or induced abortion(1,2,3,8). Our patient had none of the risk factors mentioned she only 27 years, had a previous vaginal birth with no history of suction curettage.

The strong association between PP, and multiparity, previous caesarean delivery, maternal age and abortion suggests endometrial damage to be the etiological factor. Presumably each pregnancy damages the endometrium underling the implantation site, rendering the area unsuitable. Subsequent pregnancies are likely to become implanted in the lower uterine segment by a process of elimination(8, 9, 10)

The hallmark of PP is the sudden onset of painless vaginal bleeding in the second and third trimester(3). The formation of the lower uterine segment and cervical dilation that may occur at this time invariably leads tearing of the placental attachment. Bleeding may begin with or without any incitement such as pelvic exam, intercourse or onset of labour. The bleeding is augmented by the inability of the myometrial fibres of the lower uterine segment to contract and thereby constrict the torn blood vessels. The patient presented with painless vaginal bleeding in early third trimester with no obvious inciting factor other than formation of the lower uterine segment (2, 3).

On abdominal exam the uterus is normally soft with absence of palpable contraction in a majority of the cases. (about 20% are seen to have palpable contractions)(2, 3). Fetal distress is usually not seen unless the bleeding was severe. The lie is abnormal in upto 35% of the cases but if the vertex is presenting it is usually felt high above the brim. Our patient had a longitudinal lie, cephalic presentation and the presenting party was high.

Digital exam is absolutely contra-indicated in any case of suspected PP, a sterile speculum exam is done followed by an abdominal ultrasound (unless an obstetric scan had been done earlier documenting the placental location)(2, 3).

The most useful study is the transvaginal ultrasonography which provides 100% accuracy in identifying Placenta previa(4). Trans-abdominal ultrasonography provides 95% accuracy. There is evidence that PP is much more common in early pregnancy but most of then resolve as the pregnancy progresses. A low laying placenta may be seen in upto 40% of patient done ultrasonography in 2nd trimester but 95% of them resolves by term. A centrally placed placenta in 2nd trimester, despite the likelihood of subsequent upward migration with uterine enlargement usually does not display the migrational phenomena (2, 9,10).

In our case there was no chance of significant moderation because we were in the second trimester.

For uncomplicated pregnancy continue with expectant management until an episode of bleeding occurs. Studies have not shown any difference regarding maternal and fetal morbidity with home verses hospital management prior to the 1st bleed(2,8).

If however bleeding or contractions occur the patient must got to the hospital as soon as possible for evaluation and further management(2, 3).

If bleeding persists a double set up examination in preparation for immediate surgery is indicated regardless of the fetal maturity. However if the bleeding is minimal and the fetal status reassuring then expectant management may be considered to allow time for fetal maturation(2, 3). Tocolysis can be considered in cases of minimal bleeding and extreme prematurity to give time to administer antenatal corticosteroids(1,2). Patients have to be counseled for prolonged hospital stay because the second bleed may be worse. However the woman can be discharged home after bleeding has stopped and the foetus is judged to be healthy. The woman and her family must be adequately counseled to fully appreciate the problem of placenta previa and be prepared to transport her to hospital immediately the need arises. In our set up however realistic, careful and somewhat subjective patient selection is required for safe out-patient management. Patients who have

economic disadvantages, little domestic support, young children at home, no telephone facilities or transport difficulties and unreliable blood transfusion services are unlikely to be good candidates for this approach. For these reasons our patient was adequately counseled and managed as an in patient.

Caesarian delivery is the safest mode of delivery(2). Most often a transverse incision is used; however a vertical incision may be considered in an anterior placenta previa and the risk of fetal bleeding(13). If the patient is at an increased risk of having invasive placentation the patient and surgical team need to be well prepared prior to delivery. These invasive placentation carry a higher mortality rate (7% of placenta previa have placenta acreta) as well as a high morbidity rate (blood transfusion, infection and adjacent organ damage)(2, 9, 10).

Bleeding from the placental bed can occur given that the lower uterine segment has poor contractile ability. Specific bleeding points can be sutured but use of multiple sutures to control generalized bleeding is usually futile(2,9,10).

Direct injection of oxytocin, ergometrine or prostaglandins (15 methyl PGF2a) may successfully arrest the bleeding. There is a strong association between complete placenta previa and postpartum haemorrhage especially after c/s.

Other methods of arresting the bleeding include uterine artery ligation, bilateral iliac artery ligation. Hysterectomy may be performed in cases where bleeding is not controlled by the above measures or when placenta acreta or percretta are present(14).

Complications of placenta previa include haemorrhage, preterm delivery, congenital malformation, abnormal fetal presentation, placental abruption and low birth weight babies due to intra-uterine growth restriction.(50% of PP have premature deliveries and a greater incidence of LBW and congenital anomalies.)(10, 11)

Vaginal delivery is usually reserved for patients with a marginal implantation and a cephalic presentation (2). During the double set up examination in theatre, palpation of the placenta on the lower uterine segment not only conclusively confirms the clinical diagnosis but also identifies the degree. Low rupture of membranes is done which helps in the initiation of labour and thereby encourages the descent of the head. This in turn presses on the separated placenta and controls the bleeding. Oxytocin drip is started. If amniotomy fails to stop bleeding or to initiate labour the caesarean section is performed (1,2).

Rhesus immunoglobulin should be administered to all at risk patients with third trimester bleeding who is rhesus negative.

Patients should be counseled adequately on the risk of recurrence in the subsequent pregnancies(10). To inform her next obstetrician of these occurrence.

REFEERENCES

1. Cunningham F.G, Gant N.F, Levono K.J, Gilstrap L.C, Hauth J.c, Wenstrom K.D (Editors). Obstetric Haemorrhage. In Williams Obstetrics 21st Edition , McGraw-Hill Company 20012: 619- 688.
2. Bhide A, Prefumo F, Moore J, et al. Placental edge to the internal os distance in the late third trimester and the mode of delivery in placenta previa. BJOG 2003 Sep: 110(9): 860-4.
3. Claydon C.s, Pernoll M.L. Third trimester vaginal bleeding in Currents Obstetric and Gynecologic Treatment and Diagnosis 9th Edition McGraw-Hill Company, 2003:354-366
4. Comstock CH, Love JJ, Bronsteen RA et al Sonographic detection of placenta previa; in 2nd ands 3rd trimester of pregnancy. Am J Obstet Gynecol. 2004 April; 190(4): 1135-40.
5. Kirima J Characteristic s of patients with antepartum haemorrhage. Mmed Thesis, University of Nairobi, 1981.
6. Ojwang SBO. Placenta previa in Kenyatta National Hospital. Mmed Thesis, University of Nairobi. 1974.
7. Mbithi D.L.K. Gray scale ultrasound in the diagnosis of placenta previa at Kenyatta National Hospital. Mmed Thesis University of Nairobi. 1983
8. Fiaz AS, Anath C.V: Etiology and risk factors for placenta previa; an overview and meta-analysis of observational studies. J Maternal featl Neonatal Med. 2003 March 13(3) 175-90.
9. Gilliam M. Rosengerg D. Davis F; The likely hood of placenta previa with greater number of cesarean deliveries and high parity. Obstet Gynecol. 99;976-80, 2002
10. Dutta DC (Ed) Antepartum Haemorhage. In; Textbook of Obstetrics. Sixth Edition, New Central Book Agency, Calcutta, 2004:243-254
11. Anath C.V Demisee K. Smulian J. et al : Relationship among placenta previa, fetal growth restriction, and preterm delivery. A population based study. Obstet Gynenecol 98:299,2001a
12. Ward CR, Avoiding an incision through the anterior previa at cesearean delivery. Obstet Gynecol 2003 Sep 102(3): 552-4.
13. Hwu Ym, Chen CP, Chen HJ et al. Parrell vbertical compressional sutures; a technique to controlling bleeding from placenta previa and accreta during ceaserean delivery . BJOG 2005 Oct 112(10): 1420-3

OBSTETRIC 3

MULTIPLE PREGNANCIES: TRIPLETS VIRGINAL DELIVERY

NAME: G.K	IPNO.1108006
AGE; 35 YEARS	LMP 25/11/05
DOA: 2/08/06	E.D.D 29/09/05
DOD: 15/08/06	GESTATION 36 WEEKS.

History of presenting illness

She was admitted with complaints of lower abdominal pain that was not increasing in intensity and frequency. The pain had lasted the past 24hour prior to admission with no accompanying drainage of liquor, no per vaginal bleeding and no urinary complains.

She came with an obstetric ultra-scan done at 19 weeks confirming the triplet gestation.

Past medical history

There was no history of chronic illness or surgical operations.

Obstetric and gynecology history

She was Para 1+0 gravida 2, gestation was 36 weeks as per the above dates.

First delivery in 2001 a caesarean birth (due to fetal distress) to a 3600grams live male infant who is alive and well.

Menarche was at 15 years

Cycle of 28- 35 days and duration of 4 days, irregular

She used combined oral contraceptive pill for 2 years till 2003. She had been followed up infertility since early last year and conceived after a course of clomiphene citrate.

Antenatal clinic

She attended at ANC Kenyatta National Hospital from 31 weeks. She made 4 visits.

Her profile parameters were

Blood group o positive

VDRL negative

HIV negative

Haemoglobin level 13.0g/dl

Family and social history

She is a married secretary. Husband is a mechanic. She does drink alcohol or smoke cigarettes. They live in Kariobangi South.

Strong twinning history in her family she is a twin.

On examinaton

She was in good general condition, not pale, afebrile. The blood pressure was 120/70 mmhg, the pulse 84/min, respiratory rate 20/ minute

Abdominal examination

The abdomen was pendulous, with a sub-umbilical mid-line scar. The first twin was in cephalic presentation. There were multiple fetal parts. There were fetal heart beats heard at two different points that were distinct from the maternal pulse and were not synchronous with each other. Neither obvious contraction nor area of tenderness was noted.

Diagnosis:

Multiple gestation of the higher order, in 1psc at 36 weeks.

MANAGEMENT

She was admitted in the antenatal wards for bed rest and observation as she awaited an elective caesarean section at 37 weeks. The patient was given a high protein and calcium diet plus haematinics.

PROGRESS

She went into labour before the date of the scheduled elective caesarean section. An emergency caesarean section was performed

Intraoperative care

An emergency caesarean section was performed though a repeat midline sub-umbilical incision and a transverse incision in the lower uterine segment. The outcome were

1st: live female infant with an Apgar score of 9/1, 9/5and 9/10 weighed 23000grams.

2nd: live male infant with an Apgar score of 9/1, 9/5, 9/10. Weighed 21000 grams

3rd: live female infant with an Apgar score of 9/I, 9/5, 10/10. Weighed 2100grams.

There were three placentas with one membrane dividing the amniotic sacks. The liquor was clear. The infants were reviewed by the paediatrician, who found them to be stable enough and allowed them to room in with mother..

Post- operative Management

The vital sign were observed half hourly until she was fully conscious, one hourly for 4 hours and then 4-hourly. She was put on intravenous fluids, 1 litre of dextrose alternating with normal saline 8-hourly for 24 hours. Analgesia was through intramuscular pethidine 100 mg 8-hourly for the first 24 hours then oral diclofenac 50 mg 8-hourly. She was also put on intravenous augmentin 1.2g 8-hourly for 24 hours then orally for 5 days.

On the third day post op she developed a burst abdomen which was a adequately repaired in theatre. One of the knots was found to have slip. The rest of the post operative period was uneventful. Breast feeding counseling was done and the football method (for the two) was adopted together with doing EBM (for the one) prior to starting the actual breast feeding.

POST NATAL FOLLOW-UP

She was seen in the post-natal clinic one week after discharge. She was in good general condition, the uterus had involuted well and there was minimal lochia Alba. The calf region was normal and she was counseled on contraception, infant feeding and on immunization of the babies as per the KEPI schedule.

DISCUSSION

Twin foetuses result from fertilization of two separate ova i.e. double ovum, dizygotic or fraternal twins. About a third as often twin arises from a single fertilized ovum that subsequently divides into two similar structures, each with potential to develop into a separate individual.

Maternal morbidity and mortality rates are mush higher in multiple pregnancies than in singleton pregnancies due to preterm labour, haemorrhage urinary tract infection and pregnancy induced hypertension(1,2,3). The Perinatal mortality rate of twins is 3-4 times higher to than those of singleton pregnancies and even much higher in higher-order multiple pregnancy(2,3). The Perinatal mortality is high due to chromosomal abnormalities, prematurity, anomalies, hypoxia and trauma(1, 4, 5).

In a heterozygous population slightly more than 30% are monozygotic and nearly 70% are dizygotic.The incidence of multiple pregnancies is approximately as follows(4,5):-

Twins 1:80

Triplets 1: 80^2== 1:6400

Quadruplets: 1:80^3==512000

In multiple births, male predominate but die early. Both are male in 45% of the cases, and both are female in 30%.The frequency of multiple births varies significantly among different races and ethnic groups(2). For example in white, the incidence is in every 100 pregnancies as in blacks 1 in every 80 pregnancies, in Nigeria mainly found twinning to occur once every 20 births(6). These differences could be due to racial variation in follicle stimulatory hormone levels, which can lead to multiple ovulations.At Kenyatta National Hospital Oyieke found the incidence to be 1:58.8, while Mutungi 1:46 at Pumwani Hospital(7, 8). In countries were assisted conception is common the rate of twins and other higher –order pregnancies has been on the rise. Among the women undergoing ART 25% -30% have twin pregnancies (9).

This has been of public health concern because of the high rates of preterm delivery of these neonates' compromises their survival chances and increases their risk of lifelong disability. The concept of fetal reduction has created a new category or twin pregnancies with an accompanying moral/religious implication (1, 9).

Triplets may arise from various twinning processes(1,3,5)

- Monozygotic where by there is repeated twinning of a single ovum(from super-twinning)
- Dizygotic triplet's individual fertilization of independently expelled ova.
- Dizygotic ovum from twinning of two separate ova and elimination of one of the four.

Monozygotic twinning occurs by chance and so may not have differences in the various races or families. The peak for dizygotic twinning is at 35- 40 years then declining sharply (maximum hormonal stimulation increases the rate of double ovulation).

History of previous dizygotic twinning increases the risk of multiple pregnancies by 10 folds(3,4,5).

Those who are twins or daughter to a twin have age 37 as the peak for twinning and plateau at 45.Other factor that are associated with increased chance of twinning are increased body weight (under nutrition has a negative effect), blood group O and A, soon after cessation of a long term contraceptive(2,4,5,10).

Our patient had multiple risk factors; she is a twin, was 35 years of age and had used clomiphene citrate for ovulation induction. Her being a twin gives her a 10 fold chance of polyovulation because it is hereditary. The use of clomiphene citrate increases the pituitary gonadotrophin levels leading to polyovulation(4,5,10). She was at the peak age of twinning.

Multiple pregnancies affect the woman and put her at risk of some specific conditions. Maternal anaemia often develops because of greater demand for iron by the fetuses (3, 4, 5). Marked uterus distension and increased the same on adjacent viscera and pelvic vasculature are typical of multiple pregnancy. Placenta praevia develops where frequently because of the large size of the placenta(4,5).

Multiple pregnancies are classified as high risk because of the increased incidence of maternal anaemia, urinary tract infection, pre-eclampsia, and uterine inertia(5).

In making a diagnosis, a maternal history of twins, older maternal age, high parity, larger maternal size, and previous history of twins should be taken into account. Clinical examination with accurate measurement of fundal height is essential (4, 5). During the second trimester the uterine size is larger than expected for gestational age determined from maternal data by over 4cm on palpation there are two fetal poles in two different quadrants. Also two fetal hearts distinct from each other by 8 beats and asynchronous with the maternal heart beat (3,4,5).

By ultra sound examination, separate gestational sacs can be identified very early in twin pregnancy(3). The identification of each fetal heart should be made in two perpendicular planes. Two heads or two abdomens should be identified to avoid scanning the same fetus twice and interpreting as twin. 99% of multiple gestations are diagnosed before 26 weeks gestation(1,4).

Spontaneous abortion is more likely with multiple fetuses. The incidence of congenital malformation is appreciably increased in twin and higher order multiple gestations compared with singletons. These are defects resulting from twinning itself, which are considered to be teratogenic event(1,4,5). Also defects resulting from vascular interchange between monochrorionic twins. This can give rise to reverse flow with arcades in one twin or one twin dies intravascular coagulation with embolization to the living twin. Defects that occur as a result of crowding include talipes or congenital hip dislocation(4,5). In our case there were no fetal anomalies noted.
Multifetal gestations are more likely to be characterized by low birth weight than singletons due mostly to restricted fetal growth and preterm delivery. In dizygotic twins size discordance results from unequal placentation from one placenta receiving better blood supply than the other(1).

As the number of fetuses increases the duration of gestation decreases(1,5). About half the twins are delivered at 36 weeks or less, triplets at 33.5 weeks. Our patient delivered at 36 weeks. The upper fetal growth limit for singletons and maternal support capacity occurs at 34 to 35 weeks with combined with weight reaching *4000g* to *5000g*. *Delivery before term is the major reason* for the increased risk of neonatal death and morbidity in twins. Maternal hypertension; fetal growth restriction and placenta abruption were the main indication for preterm delivery of twins (*1,4,5,11*).

The antepartum management aims at prevention of preterm delivery of preterm infants, identify failure to thrive of fetuses, avoid fetal trauma during labour and delivery. Expert neonatal care should be provided(11). To prevent preterm delivery the mother should have bed rest, prophylactic administration of beta mimetic drugs.

Many complications of labour and delivery include preterm labour, uterine dysfunction, abnormal presentations, prolapse of the umbilical cord, premature separation of the placenta and immediate postpartum haemorrhage(11). In multiple pregnancy, all possible combination of fetal position may be encountered i.e. cephalic-cephalic, cephalic-breech, cephalic transverse. Labour is generally shorter in twins. In vaginal delivery, the presenting twin bears the brunt of dilating the cervix and the remaining soft tissue of the birth canal(1,5,11). When the first twin is breech, caesarean delivery is the method of choice.14. For the second twin as soon as the first twin has delivered, the presenting part of the second twin, its size and relation to the birth canal are ascertained. If the fetal head or breech is fixed in the birth canal, moderate fundal pressure is applied and the membranes

are ruptured. Labour is allowed to resume while the fetal heart is monitored. The interval between delivery of the first and second twin is safest in less than 30 minutes(1). However Rayburn et al showed a good outcome even if the interval is longer if continuous fetal monitoring is employed (12). The American college of obstetrics and gynaecology has determined the interval between deliveries of twins is not critical in determining the outcome of the second twin(1).

Labour and delivery entails increased risk in triplets. There is high incidence of malposition, increased incidence of cord prolapse and fetal collision(1). Reduced placental perfusion and haemorrhage from separating placentas. Most likely during delivery. Pregnancies complicated by three or more fetuses and delivered by caesarean section(1,4,5).

Our patient underwent an emergency caesarean section with good outcome.

REFFERENCES

1. American collage of Obstetricians and Gynecologists; Multiple gestation : complicated twins, triplets and higher order pregnancies. Practice Bulletin No 56, October 2004.
2. American collage of Obstetricians and Gynecologists. Special problems of Multiple gestation 1998. 253.
3. Cunningham FG, McDonald PC, Grant FC, Leveno KL, Giltrap LC. Multiple pregnancy in the Williams Obstetric 20th edition .pp
4. Walter WAW. Multiple pregnancy in the Turnbulls Obstetrics 3nd edition. Church hill Livingstone. 2001:229-245
5. Pernol ML, Benson RC. Multiple pregnancy in Currents Obstetric and Gynecology, Diagnosis and treatment. 9th edition Mc Graw hill 315-325.
6. Lynch A et al Assisted reproductive intervensions and multiple birth. Obstet Gynecol 2001;97:195.
7. Whitefields CR. Multiple pregnancy in Dewhursts textbook of obstetrics and gynecology for postgraduate students.4th edition Black well publication 1986 482-495.
8. Oyieke SBO. A two and half year review of some aspects of twin delivery at Kenyatta National hospital. Mmed Thesis University of Nairobi.(1978).
9. Mutungi AK. A prospective study of twins at KNH and Pumwani maternity hospital, Antenal care and delivery. Mmed Thesis University of Nairobi 1990.
10. Abudu OO, Agarin E. Twin pregnancy and maternal mortality in Lagos. J Obstet Gynecol East and Central Africa. 1984: 3:7-11
11. Albert Reece JCH. Multi-fetal pregnancies epidemiology, clinical characteristics and management . In medicine of the foetus and mother. 2nd edition pp 243-62. Lippincot-Raven Publishers Philadelphia.
12. Rayburn WF, Lavin JP Jr, Miodvnik M., et al. Multiple gestation: Time interval between delivery of first and second twin. Obstet Gynecol 63:502, 1984.

Obstetric 4

SEVERE PRE ECLAMPSIA AT 30 WEEKS; LIVE BIRTH

Name: D. M IPNO 1097463
Age: 34 years LMP 12/11/05
DOA 8/06/06 EDD 19/8/06
DOD 17/07/06 GESTATION 30 weeks
PARITY 2+0

Presenting complaint

D. M was admitted through labour ward as a referral from heath centre where her blood pressure was noted to be high. She also complained of headache that had been there for four days prior to admission.

History of presenting complaint

She had been followed up in an antenatal clinic where her blood pressure had been noted to be elevated. For two weeks she had been treated in the health centre. She was put on aldomet 250mg plus phenobarbitone 30mg both 8 hourly with good blood pressure control initially.

Four days prior to admission, she developed a persistent bitemporal headache that was not relieved by paracetamol. Blood pressure readings before referral was 216/123mmhg. There was no history of blurring of vision, loss of consciousness or fits. There was no reduction in urine volume. Foetal movements had remained normal

Past obstetric and gynaecological history

She was a para 3+0. Her first delivery was a spontaneous vertex delivery at term to a live male infant who weighed 3200g in 1992 following a normal antenatal period. In 1995, she had a spontaneous vertex delivery at term to a live female weighing 3300g after an uneventful pregnancy. Her 3rd delivery was in 2002, had an IUFD at term and the blood pressure were elevated. The first two children were alive and well.

Her menarche was at 14years. Her periods lasted 4 days and were regular recurring every 28 days. She used combined oral contraceptive pills for contraception between the pregnancies.

Antenatal care

She had attended antenatal clinic at Nembu medical clinic starting from 18 week of gestation. BP ranged between 120/80 to 140/100 mmHg during the follow up and was put on aldomet 250mg plus phenobarbitone 30mg both 8 hourly. Antenatal profile was as follows: VDRL and HIV were negative; Blood group O positive; Hb 12.7g/dl; urinalysis had proteinuria trace to 1+. She had received 1 injections of Tetanus Toxoid (TT)

Family and social history

She was a married tailor who neither took alcohol nor smoked cigarettes. Her husband was a driver. They leaved in Waithaka. There was no history of twins or chronic illness in the family.

Past medical history

Not significant

Systemic inquiry

No significant findings

PHYSICAL EXAMINATION

General examination:

She was in fair general condition, conscious, not febrile, not pale, no jaundice, but had moderate pitting pedal oedema.

Observations

The BP was 200/111mmHg, Temperature 37^0C, pulse 90BPM regular, Respiratory rate was 18 per minute; urinalysis – protein ++.

Respiratory, cardiovascular, and central nervous systems

Essentially normal

Abdominal examination

The fundal height was 28 weeks and mild epigastric tenderness, presentation was cephalic, and the presenting part was balotable. The foetal heart was heard and regular at 140 bpm.

Vaginal examination

Not indicated

DIAGNOSIS

A diagnosis of a 34 year old para 2+0 with severe PET at 30 wks was made.

MANAGEMENT

1. Admit to the antenatal ward after BP has been stabilized.
2. Bed rest
3. Blood pressure monitoring 4 hourly.
4. Daily urinalysis
5. Urea, Electrolyte and Creatinine, Full haemogram and Liver function tests.
6. Obstetric Ultrasound and biophysical profile.
7. Hydralazine 10mg iv slow push, Aldomet 500mg 8 hourly, Hydralazine 50mg 8 hourly.
8. Foetal kick chart.
9. Dexamethasone 12mg im 12 hourly for a day.

She was admitted to the labour ward of KNH. Hydralazine intravenous 10mg was pushed slowly; a recheck in the BP confirmed acceptable lowering of it. She was then transferred to the antenatal wards on Aldomet 500mg three times a day, oral Hydralazine 25mg daily.

INVESTIGATION RESULTS

(On admission)		**Later(19.06.06)**	
Urea	4.4mmol/l	Urea	4.1mmol/l
Creatinine	79umol/l	Creatinine	63umol/l
T. bilirubin	4mmol/l	T. bilirubin	3.1mmol/l
Alkaline phosphate	135IU/l	Alkaline phospatase	99IU/L
WBC	12 x 10^9/L	WBC	9.18 x10^9/L
Hb	12.9g/dl	Hb	12.2g/dl
Platelet	244 x 10^9/L	Platelet	198x10^9/L

Obstetric ultrasound done (9/06/06)

An Obstetric ultrasound scan was done on the day of admission confirmed an intra-uterine pregnancy with fetal cardiac activity of 143 beats per minute. Placenta war ported to be fundal anterior and not low lying. Amniotic fluid was adequate in volume. There was normal fetal parts and fetal movement. Biparietal diameter and femoral length corresponded to gestation of 28 weeks.

PROGRESS

The blood pressure remained unstable with episodes of high recordings. The BP (range 180/110 – 160/80), urine having a proteinuria of 2+ to 3+. She developed facial puffiness and reduced fetal movements on the 20/06/06(31 + weks). An urgent ultrasound, CBC, LFTS and UEC were done.

Obstetric ultrasound done (20/06/06)

Single intra uterine fetus in cephalic presentation with an average computed gestational age of 29 weeks 1 day. BPD 7.46cm corresponds to 30 weeks and 1day

FL 5.29cm corresponds to 27 weeks and 2 days.

AC 23.4cm corresponds to 27 weeks and 5 days

FHR was 151/min and regular. Estimated fetal weight 1176g, placenta fundal anterior and not low lying. Umbilical artery Resistive Index was 0.788

BBP of 6/8

Nifedipine 20mg twice daily were added on her treatment. Dexamethasone 12mg was administered intramuscularly 12 hourly, two doses. She developed persistent headache, vomiting and severe epigastric pain on 29/06/06. The BP taken then was 196/112. A diagnosis of impending eclampsia was made. She was started on magnesium sulphate and a decision was made to deliver her by emergency caesarean section.

Intraoperative care

An emergency caesarean section was performed though a lower abdominal transverse incision (Pfannestiel) and a transverse incision in the lower uterine segment. The outcome was a live male infant with an Apgar score of 7 in 1 minute 9 in 5 minutes and 10 in 10 minutes with a birth weight of 1250 grams. There was a loop cord round the neck. The placenta was grossly normal. The liquor was clear. The infant was reviewed by the pediatrician and admitted in the NBU because of the prematurity.

Post- operative Management

The vital sign were observed half hourly until she was fully conscious, one hourly for 4 hours and then 4-hourly. She was put on intravenous fluids, 1 litre of dextrose alternating with normal saline 8-hourly for 24 hours. Analgesia was through intramuscular pethidine 100 mg 8-hourly for the first 24 hours then oral diclofenac 50 mg 8-hourly. She was also put on intravenous augmentin 1.2g 8-hourly for 24 hours then orally for 5 days.

The blood pressure was controlled on Methyl dopa 500mg 8 hourly and Nifedipine 20mg 12 hourly. Her baby did well in the NBU and was discharged to join her 15/08/06 at 2000g. They were discharged home through the postnatal clinic in two weeks.

Follow up

On review 2 weeks after discharge, the BP was 120/70. She had no complaint, the breasts were not engorged, uterus was not palpable and there was no lochia loss. The Methyl dopa was further tapered to 250mg twice daily.

She was reviewed two weeks later and the BP was 100/60. The Methyl dopa was stopped. She was referred to the family planning clinic.

DISCUSSION

D.M was a 34 year old Para 3 + 0 gravida 4 who was admitted with severe pre eclampsia at 30 weeks. She had developed PET at 28 weeks and had been managed as an outpatient in a peripheral healthy centre. She was referred to KNH after her blood pressure was noted to be very high with worsening of her symptoms. The blood pressures were controlled at admission then she was sent to the antenatal wards were expectant management was instituted. Three weeks after admission she developed features of impending eclampsia and an emergency caesarean section was done, after the initial doses of Magnesium Sulphate. The outcome was a live male infant who weighed 1250g who scored 7/1, and 9/5. The baby did well and was discharged from NBU after 6 weeks with a weight of 2000grams.

Pre-eclampsia (PET) is one of the hypertensive disorders of pregnancy responsible for significant maternal and foetal morbidity and mortality worldwide. Pre-eclampsia is a pregnancy specific syndrome of reduced organ perfusion secondary to vasospasm and endothelial damage. According to the Working Group of the National High Blood Pressure Education Programme 2000, there are five types of hypertensive disease that include (1):

1. **Gestational hypertension** (formerly pregnancy induced hypertension or transient hypertension) – characterized by a BP of 140/90mmHg or more for the first time during pregnancy but no proteinuria. The BP returns to normal < 12 weeks postpartum. The final diagnosis is only made postpartum.
2. **Pre-eclampsia** - the minimum criteria includes; BP equal or > 140/90mmHg after 20weeks gestation, proteinuria of 1+ (300mg/24hrs) or more.
3. **Eclampsia** – seizures that can not be attributed to other causes in a woman with PET.
4. **Pre-eclampsia superimposed on chronic hypertension** – new onset proteinuria of 300mg/24hrs or more in hypertensive women but no proteinuria before 20wks gestation or a sudden increase in proteinuria or blood pressure or platelet count < 100,000/mm3 in women with hypertension and proteinuria before 20wks gestation.
5. **Chronic hypertension** – BP equal or >140/90mmHg before 20weeks gestation or hypertension first diagnosed after 20weeks gestation and persistent after 12 weeks postpartum.

This patient had a BP of > 140/90mmHg that developed after 20weeks gestation and proteinuria of >1+ (300mg/24hrs). The BP normalised by the tenth postpartum week. She therefore had pre-eclampsia.

Pregnancy-induced hypertension can be divided further into mild and severe forms, with the latter characterized by the following findings; blood pressure equal or greater than 160/110 mm Hg, proteinuria (>5 g/24 h), elevated serum creatinine, seizures, pulmonary oedema, oliguria (<500mL/24 h), microangiopathic haemolysis, thrombocytopenia (platelet count < 100,000/mm3), hepatocellular dysfunction, and intrauterine growth restriction or symptoms of end-organ involvement (e.g., headache, scotomata, or epigastric pain) (1, 2). Importantly the difference between mild and severe pre-eclampsia can be misleading because apparently mild disease may progress rapidly to severe disease (2). Our patient had severe pre-eclampsia that latter progressed to impending eclampsia.

In Kenya, hypertensive disorders are the third commonest cause of maternal morbidity and mortality after haemorrhage and infection (3). According to the National Centre for Health Statistics in 1998, hypertension associated with pregnancy was the most common medical risk factor. The rate is even higher in developing countries, with an estimated frequency of 1 in 100 to 1 in 1700 cases (4, 5). Worldwide the incidence of pre-eclampsia varies widely from 5-15 % (1). In Kenya,

the incidence of pregnancies complicated by hypertensive disorders as reported by Mati varied from 1.5 – 9% and that of pre-eclampsia was 3.7 – 5.4% in another study (6, 7).

Although the cause remains unclear, pre-eclampsia may be initiated by placental factors that enter the maternal circulation and cause endothelial dysfunction resulting in hypertension and proteinuria. (1, 4-6) Chesley (8) summarized several predisposing factors and noted that the order of importance is somewhat arbitrary. Nulliparity, familial history, diabetes, multiple gestations, and extremes of age, pre-existing hypertension, vascular renal disease, hydatidiform mole, and foetal hydrops have all been associated with an increased risk of pregnancy-induced hypertension. Our patient was 34 years old but had none of the other risk factors.

Aetiology of pre-eclampsia is unknown. Several theories have been proposed to explain the pathophysiology of the disease. The immunologic theory finds support in the observation that pregnancy-induced hypertension is most commonly a disease of first pregnancy, increased in multipara women with a new spouse or undergoing donor insemination, and more common in immunocompromised women. (9) Researchers observed that the daughters of mothers with pre-eclampsia were at higher risk than were the daughter-in-laws for this disorder in a single recessive gene fashion (10, 11); however, a multifactoral inheritance could not be ruled out. (12) Other factors include dietary excess and deficiencies, endothelins, endothelium-derived relaxing factors, and vasoactive substances (1).

Severe pre-eclampsia was associated with protein C resistance owing to the factor V Leiden mutation (13). There is an imbalance in the prostacyclin thromboxine ratio and the Rennin-Angiotensin-Aldosterone system. Although the true aetiology of pregnancy-induced hypertension remains elusive, two basic abnormalities are consistently seen; placentation and platelet-vessel wall interaction (9).

Abnormalities of placentation develop early in gestation prior to the onset of clinical disease. (9) Abnormalities of aberrant platelet and vessel wall interaction are thought to be caused by microangiopathy (15). Damage to the endothelial cells of the vessel wall triggers an increase in platelet aggregation, which, in turn, gives way to changes in the production of various coagulation factors, which ultimately results in increased thrombin and fibrin formation. The inciting cause of initial endothelial damage has been thought to result from generalized cellular dysfunction and not an anomaly specific to vessel wall endothelium. The results of assays of various coagulation factors

have been used to predict the severity of the pathology associated with pregnancy-induced hypertension (16).

The pathologic effects of PET can be maternal or foetal (1):

Maternal effects:

- Brain –The basic lesions are oedema, hyperaemia, focal ischemia, thrombosis, and haemorrhage.
- Cardiovascular – increased peripheral resistance hypovolemia with decreased cardiac output, haemoconcentration and extravasation of fluid into the extracellular compartment. This caused by generalised vasoconstriction and increased vascular permeability.
- Renal – reduced renal perfusion and glomerular filtration. Plasma uric acid and creatinine levels are elevated especially in severe disease. Proteinuria is due to increased permeability. The classical lesion in the glomerular capillaries is endotheliosis. Acute renal failure may develop from tubular necrosis.
- Liver – periportal haemorrhagic necrosis which may cause hepatic rupture or may extend beneath the hepatic capsule to form a subcapsular haematoma. HELLP (haemolysis, elevated liver enzymes and low platelets) may ensue.
- Haematological – thrombocytopenia, fragmentation haemolysis decreased clotting factors
- Eye – retinal artery vasospasms or retinal detachment.

Foetal effects: may include (1).

- Growth restriction
- Foetal demise
- Abruptio placenta
- Premature delivery.

The treatment of pre-eclampsia include timing of delivery, prevention of seizures, treatment of hypertension ,fluid management and supportive care for the various end organ complication. Our patient had the disease before 32 weeks, so expectant management was instituted. This involves weighing foetal benefits against maternal risks. Since the only justification for expectant management is to prolong the pregnancy for foetal gain- there is no benefit to the mother. Women who develop PET before 32 weeks are more likely to adverse foetal outcome, such as IUGR (15%-

20%); preterm delivery 50%; and Abruptio placenta (1% - 2%) compared to women diagnosed with pre-eclampsia at 32-36 weeks. They require more antenatal surveillance. Expectant management of patients with severe PET is only for properly selected patients because the maternal and foetal condition could deteriorate.

What is to be monitored; (before 32 weeks with PET)

- BP monitoring at least daily. Daily urine dip-stick evaluation to monitor protein.
- Twice weekly liver enzymes and platelet counts.
- Documentation of symptoms. (Instruct all women to report the onset of severe headache, visual disturbance, altered mental status, epigastric or right upper quadrant pain and any nausea or vomiting.

Foetal monitoring.

- Daily foetal movement count, (foetal kick chart.)
- Serial ultrasounds every three weeks. (BPP, foetal weight estimation, and amniotic fluid estimation). When the BPP is not reassuring then non-stress testing every week and umbilical Doppler flow studies for restrictive index. **Repeat all the above tests if/ when maternal condition deteriorates.**
- Patients are restricted from daily activities but they really don't need complete bed rest.
- Antihypertensive reduce maternal complication but have no effect on the disease progression. Aim to attain a BP bellow 160 mmhg systolic and 90-105 mmhg diastolic.

D.M initially presented with mild PET that progressed to severe PET causing her referral to KNH. Being at 30 weeks with normal blood work-ups she was selected for expectant management to give the fetes time for lung maturation.

She latter developed features of impending eclampsia. (Severe epigastric pain, headache and vomiting). The epigastric pain may have been due some degree of subcapsular haemorrhage. Serial platelet levels done indicated a gradual decline in the levels from 244 to 198 $X10^{9}$. Though they were still within acceptable limits. Renal and liver function tests remained normal. The obstetric scans done showed some level of intrauterine growth restriction. Biparietal diameter corresponding to 31 weeks whiles the abdominal circumference and femoral length corresponds to 27 weeks. Umbilical vessels Doppler studies revealed an elevated restrictive index of 0.788 indicating that if delivery was not done in the near future foetal demise could have occurred. She was delivered by

caesarean section due to the prematurity and IUGR. At delivery the placenta did not have any retro placental clot (no abruption) and no problem was encountered with haemostasis during the delivery.

Pre-eclampsia, remote from term presents a much more difficult management problem. The decision of whether to intervene and deliver a pre-term infant (that may require prolonged intensive care) or to institute expectant management is usually governed by disease severity and length of gestation (1

The role of hospitalization for bed rest in mild pre-eclampsia has been challenged. Instead, some workers have recommended outpatient management with regular blood pressure, weight, foetal kick charts and twice-weekly non-stress tests. The patient is advised to seek immediate attention if she records elevated blood pressure or reduced foetal movements. If the woman is remote from term and becomes normotensive after hospitalization, and without evidence of foetal compromise, outpatient surveillance may be considered. There is no place for conservative management if there are signs of progression to severe pre-eclampsia or foetal monitoring tests becoming abnormal (17). Conservative management of mild disease beyond term is not beneficial to the foetus because utero-placental blood flow is sub-optimal. After 37 weeks gestation, labour should be induced as soon as the cervix is favourable or cervical ripening with prostaglandins may be utilized (1).

The use of anti-hypertensive drugs in attempt to prolong pregnancy or modifying perinatal outcome in pregnancy complicated by the various hypertensive disorders has been of considerable interests. Treatment for early mild pre-eclampsia with antihypertensive remote from term is controversial. Some claim that anti-hypertensive drugs appear to reduce the progression to severe disease by significantly lowering the mean blood pressure but do not improve pregnancy prolongation, gestational age at delivery or birth weight. A meta-analysis on various anti-hypertensives (labetolol, nifedipine isradipine) concluded that anti-hypertensive induced decrease in maternal blood pressure affects foetal growth causing growth restricted infants. (26) Bed rest alone has also little effect on blood pressure or disease progression (17). Our patient was on oral alpha-methyldopa, Hydralazine, magnesium sulphate and bed rest.

The use of Angiotensin converting enzyme (ACE) inhibitors during second and third trimester of pregnancy should be avoided. Reported complications include oligohydramnios, limb contractures, persistent ductus arteriosus, pulmonary hyperplasia, respiratory distress syndrome, prolonged neonatal hypertension and neonatal death (27).

In severe pre-eclampsia, seizure prophylaxis is done preferably with magnesium sulphate. In some centres, diazepam or phenytoin are still being used but are inferior to magnesium sulphate in effectiveness. The use of magnesium sulphate has gained tremendous popularity in our set up. However, magnesium sulphate decreases the beat to beat variability of the foetal heart rate, may inhibit uterine contractions and may cause neonatal hypomagnesaemia. Magnesium sulphate may also inhibit bleeding time and increase blood loss at delivery. Its use has been associated with increase in post partum haemorrhage (17). Though our patient was put on magnesium sulphate, none of these complications was observed.

Induction of labour to effect vaginal delivery has traditionally been considered to be in the best interest of the mother (1). Several concerns, including an unfavourable cervix precluding successful induction of labour, a perceived sense of urgency because of severity of pre-eclampsia and the need to co-ordinate neonatal intensive care has led some practitioners to advocate caesarean delivery. However, many authors advocate vaginal delivery and careful monitoring of oxytocin induction as this was found not to be harmful to even the very low birth weight infants (28). Our patient was delivered by emergency caesarean section due to multiple factors prematurity, very low estimated foetal weight and a poor bishop score) vaginally following induction of labour due to severe pre-eclampsia with features of impeding eclampsia. The labour was uneventful and the outcome was favourable.

Women who develop hypertension during pregnancy should be evaluated during the immediate post partum months and counselled about future pregnancies and also their cardiovascular risks later in life. If by 12 weeks of delivery the hypertension has not resolved, then chronic hypertension may be considered (1, 2).

Generally, the earlier the pre-eclampsia is diagnosed during the index pregnancy, the greater the likelihood of recurrences. Those who develop pre-eclampsia before 30wks have a recurrence risk as high as 40% during the subsequent pregnancy. Those with recurrent pregnancy hypertension are at an increased risk of chronic hypertension compared to those who remain normotensive during subsequent pregnancy (29). However, pre-eclampsia does not cause chronic hypertension. Our patient had a history of IUFD due to PET in the previous pregnancy, and had PET before 30 weeks

in this pregnancy. She has a high chance developing this again at an earlier gestation in the next pregnancy.

Our patient was a multipara who had pre-eclampsia in the preceding pregnancy. After ten weeks post delivery her blood pressure was normal (120/70mmHg) after which she was weaned off the anti-hypertensive. She was refereed to the family welfare clinic for counselling on a family planning method.

At present, there are no screening tests for PET that are reliable, valid and economical (Stamilio and colleagues 2000) (19). However a variety of biochemical and biophysical markers have been proposed for the purpose of predicting the development of PET later in pregnancy. These are (1, 17); Angiotensin II infusion, roll over test, elevated uric acid levels, calcium metabolism (hypocalciuria), diminished urinary kallikrein excretion, elevated fibronectin levels, coagulation activation, markers of oxidative stress, immunological factors (cytokines), placental peptides (especially activin A and inhibin A) and Doppler velocimetry of the uterine arteries in second trimester. This patient was not screened with any of these tests.

A variety of strategies have been used in attempts to prevent PET. These include dietary manipulation, low dose aspirin and antioxidants (Vitamin E and C).

REFFERENCES:

1. Cunningham FG, MacDonald PC, Gant NF, et al (eds): Hypertensive disorders in pregnancy. In Williams Obstetrics, ed 22. Stamford, CT, Appleton & Lange, 2005, p 567-618.
2. American College of Obstetricians and Gynaecologists: Hypertension in pregnancy. Technical Bulletin No. 219, January 1996
3. Kirubi L.W, Makokha A.E, Mukolwe B.A, et al. Causes and prevention of maternal mortality in three districts in Kenya, Kilifi, Kirinyaga and Kakamega. *J Obstet and Gynaecol E Centr Afr.* 1998; 14(2): 72-7.
4. The Eclampsia Trial Collaborative Group: Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial. Lancet 345:1455, 1995
5. World Health Organization International Collaborative Study of Hypertensive Disorders of Pregnancy: Geographic variation in the incidence of hypertension in pregnancy. Am J Obstet Gynecol 158:80, 1988
6. Mati J.K. Study on pregnancy hypertension in Kenya. Some aspects of epidemiology, prognosis, differential diagnosis and aetiology. M.Med Thesis, UoN. 1975.
7. Bansal V.P. Pre-eclampsia and eclampsia. A profile of Pumwani Maternity Hospital. E.A Med J. 1985; 62(10): 76.
8. Chesley LC: Epidemiology of preeclampsia-eclampsia. In Hypertensive Disorders in Pregnancy. New York, Appleton-Century-Crofts, 1978, p 17
9. Walker JJ, Dekker GA: The aetiology and pathophysiology of hypertension in pregnancy. In Walker JJ, Gant NF (eds): Hypertension in Pregnancy. London, Chapman & Hall Medical, 1997, p 39
10. Chesley LC, Annitto JE, Cosgrove RA: The familial factor in toxaemia of pregnancy. Obstet Gynecol 32:303, 1968
11. Chesley LC, Cooper DW: Genetics of hypertension in pregnancy: Possible single gene control of preeclampsia and eclampsia in the descendants of eclamptic women. Br J Obstet Gynaecol 93:898, 1986
12. Cooper DW, Liston WA: Genetic control of severe preeclampsia. J Med Genet 16:409, 1979
13. Dizon-Townson DS, Nelson LM, Easton K, et al: The factor V Leiden mutation may predispose women to severe preeclampsia. Am J Obstet Gynecol 175:902, 1996
14. Campbell S, Pearce JM, Hackett G, et al: Qualitative assessment of uteroplacental blood flow: Early screening test for high-risk pregnancies. Obstet Gynecol 68:649, 1986

15. Zeeman GG, Dekker GA, van Geijn HP, et al: Endothelial function in normal and preeclamptic pregnancy: A hypothesis. Eur J Obstet Gynecol Reprod Biol 43:113, 1992
16. Walsh S.W. Preeclampsia and imbalance in placental prostacyclin and thromboxane production. *Am J Obstet and Gynaecol* 1985; 152:331
17. Stamilio D.M., et al. Can antenatal clinic and biochemical markers predict the development of some pre-eclampsia? *Am J Obstet and Gynaecol.* 2000; 182: 589.
18. Walker JJ. Pre-eclampsia. Lancet 2000; 356:1260-5.
19. Roberts JM, Cooper DW. Pathogenesis and genetics of pre-eclampsia. Lancet 2001; 357:53-6.

20. Hadi B, Dies S, Goffinet F et al. Maternal and Perinatal out-come during expectant management of 239 severe pre-eclampsia women between 24 and 33 weeks of gestation. AMJ Obst Gynecol 2004 190:1590- 1597.
21. Sibai BM. Magnesium sulphate prophylaxis. Lessons learnt from resent trails. AMJ Obstet Gynecol 2004; 183: 1520- 1525

21. Hall DR, Odendaal HJ,Kirten GF et al. Expectant management of early onset severe pre-eclampsia; Perinatal outcomes. BJOG 2000;107: 1258- 1264.

22. Von D.P et al. Fall in mean arterial pressure and foetal growth restriction in pregnancy hypertension. A meta- analysis. *Lancet*. 2000; 335: 87.
23. Nightingale F.L. Warning in the use of ACE inhibitors in the second and third trimester of pregnancy. *JAMA.* 1992; 267: 244.
24. Alexander J.M. et al. Severe pre-eclampsia and the very low birth weight infants: Is induction of labour harmful. *Obstet Gynaecol* 1999; 93: 485.
25. National High Blood Pressure Education Programme: Working Group Report on High Blood Pressure in pregnancy. *Am J Obstet and Gynaecol.* 2000; 183: 51.

Obstetric case 5

PYELONEPHRITIS IN PREGNANCY - SUCCESSFUL TREATMENT- LIVE BIRTH

NAME	E.W.	IP NO.	1095032
AGE	18 YEARS	L.M.P	FEB/2006-UNSURE OF DATES
D.O.A	4/11/06	E.D.D	NOVEMBER 2006
D.O.D	25/11/06	G.B.D	38 WEEKS
PARITY	0+0		

Presenting Complaint

E.W presented with complaints of left lumbar pain, frequency of micturation and dysuria for 1 day.

History of Presenting Complaint

She had been well until one day prior to admission when she developed frequency; dysuria and left lumbar pain associated with fever, chills, nausea and vomiting. The pain was dull and continuous, but not rhythmic. The urine was cloudy in colour but normal volume. She had neither vaginal discharge nor bleeding. The foetal movements had remained adequate.

Obstetrics and Gynaecology history

She was a para 0+0, She was not sure of her dates. But she estimated her last menstrual period to have been in February giving her an EDD of November. She experienced quickening in July which corresponds with her being term in November. She had menarche at 15 years; her cycle was regular with a length of 28 days and duration of 4 days. She had never used any method of family planning.

Antenatal Care

She had attended ANC at private care provider in Dagoreti. Her first visit was in early September her fundal height corresponded to 32 weeks by then. Hb – 10.4g/dl; VDRL – Negative; and Blood group not yet done. She had received 2 Tetanus toxoid injections.

Past Medical History

Not significant.

Family and social history

She was single, a student in a computer collage in town. She did not smoke or take alcohol. There was no history of twins or chronic illness in the family.

Systemic inquiry - Non-revealing.

PHYSICAL EXAMINATION

She was in fair general condition afebrile, not pale, had no lymphadenopathy, or oedema.

Observations

The BP was 100/70mmHg, Temperature 37.8^{0}C, pulse 100BPM regular; Respiratory rate was 24 per minute.

Respiratory and cardiovascular systems

Normal.

Abdominal examination

The fundal height was term and the lie was longitudinal with cephalic presentation. There was moderate bilateral renal angle tenderness and mild suprapubic tenderness but no contractions. Foetal heart was heard and regular at 140BPM.

Pelvic examination

She had normal external genitalia; there was no cervical discharge on speculum exam. The cervix was firm posterior, about 1.5cm long with a closed os.

Diagnosis

Acute pyelonephritis at term

MANAGEMENT

She was started on empirical treatment with IV Cefuroxime 750mg 8hourly, paracetamol 1g 8 hourly, buscopan 20mg 8 hourly, and intravenous fluids. Meanwhile investigations were carried out and the results were as follows:

- Urinalysis:- cloudy appearance, Leucocytes ++, casts and epithelial cell 5-7, bacteria scanty, nitrite positive.
- Urine culture, microscopy and sensitivity: - mixed growth, $>10^5$CFU/ml E-coli and proteus species were grown. Organisms were cultured that were sensitive to Cefuroxime, Gentamycin, Nalidixic acid among other drugs.
- Full Haemogram: Hb 10.4g/dl; WBC 10 x109/l neutrophils 75%; Platelets normal; peripheral blood film was reported as normocytic normochromic picture with neutrophilia.
- HVS yeast cells and few epithelial cells NO Trichomonous Vaginalis cells were seen, Gram stain of the same showed gram stain positive cocci and rods. The culture only grew candida albicans.

The initial treatment was continued and her symptoms and general condition improved markedly. Fever subsided, lumbar pains lessened and vomiting stopped. On examination; temperature normalised within 24hrs and renal angle tenderness was less. She is still in the ward to complete her treatment.

DISCUSSION

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, 2, 3). The incidence of acute pyelonephritis in pregnancy is 1 – 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). These 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, and 5). Between 75 -90% of renal infections are caused by bacteria that have P- fimbriae adhesions (13).This patient had E.coli.

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 Gynaecology recommends that a urine culture be obtained at the first prenatal visit (10). 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, 11).

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 (7, 19). 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 labour 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 pyelonephritis. 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 (24, 25).

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 labour (26).

The initial antibiotic selection should be empiric. Based on the fact that the most common offending pathogen is E. coli, sulphonamides, 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 (10).

Women with asymptomatic bacteriuria should be treated with antibiotics such as Nitrofurantoin 100mg daily, sulphonamide, cephalosporin, 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 foetus 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, postcoital 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, and 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 labour (labour onset before 37 weeks of gestation), hypertensive disorders of pregnancy (such as pregnancy-induced hypertension and preeclampsia), anaemia (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. A risk of urosepsis and chronic pyelonephritis was also found (13). Neonatal outcomes associated with UTI include sepsis and pneumonia (specifically, group B streptococcus infection) (22). Untreated urinary tract infection during the first and third trimester of pregnancy is associated with a significant increase in mental retardation and developmental delays in the offspring (30).

REFERENCES

1. Lucas MJ, Cunningham FG. 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 Surv 1995; 50:675-83.
3. Patterson TF, Andriole V. Bacteriuria in pregnancy. Infect Dis Clin North Am 1987; 1:807-22.
4. Barr JG, Ritchie JW, Henry O, 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.
5. McDowall DR, Buchanan JD, Fairley KF.Anaerobic and other fastidious microorganisms in asymptomatic bacteriuria in pregnant women. J Infect Dis 1981; 144:114-22.
6. Kass EH. 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. 9th 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 RE. The significance of eradication of bacteriuria during pregnancy. Obstet Gynecol 1979; 53:71-3.
13. Cunnigham FF; Gant NF; Leveno KJ; 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 DJ, Andrews WW, Goldenberg RL, 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 JW, Heise RH, Naessens JM, Timmerman MG. A study of various tests to detect asymptomatic urinary tract infections in an obstetric population. JAMA 1993; 270:1971-4.

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

17. Peddie BA, Bailey RR, Wells JE. 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.

18. Sanders CC, Sanders WE Jr. Beta-lactam resistance in gram-negative bacteria: global trends and clinical impact. Clin Infect Dis 1992; 15:824-39.

19. Harris RE, Gilstrap LC 3d. Cystitis during pregnancy: a distinct clinical entity. Obstet Gynecol 1981; 57:578-80.

20. Gilstrap LC 3d, Cunningham FG, Whalley PJ. Acute pyelonephritis in pregnancy: a retrospective study. Obstet Gynecol 1981; 57: 409-13.

21. Loughlin KR. Management of urologic problems during pregnancy. Urology 1994; 44:159-69.

22. Mead PJ, Harris RE. The incidence of group B beta hemolytic streptococcus in antepartum urinary tract infections. Obstet Gynecol 1978; 51:412-4.

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

24. Moller M, Thomsen AC, 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.

25. McKenzie H, Donnet ML, Howie PW, Patel NB, Benvie DT. Risk of preterm delivery in pregnant women with group B streptococcal urinary infections or urinary antibodies to group B streptococcal and E. coli antigens. Br J Obstet Gynaecol 1994; 101:107-13.

26. Prevention of perinatal group B streptococcal 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].

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

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

29. 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.

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

Obstetric 6

BARTHOLIN'S ABSCESS IN PREGNANCY: MARSUPIALIZATION DONE

NAME: M.O. IP NO: 1125475

AGE: 19 YEARS LMP 7/04/06

DOA: 25/10/06 EDD 14/01/07

DOD: 29/10/06 GESTATION 29 WEEKS

PARITY 1+0

Presenting complaints

Painful swelling of vulva for 3 days

History of presenting complaints

The patient developed a swelling of the vulva of spontaneous onset. It was rapidly progressive and painful. There was no associated vaginal bleeding or discharge and no changes in micturation.

Past medical history

This was not significant

History of current pregnancy.

Her last menstrual period was on the 7th of April 2006 and an expected date of delivery of 14th of January 2007. She was at 21 weeks gestation by dates. She had not started her antenatal follow-up. The pregnancy had otherwise been un-eventful till then.

Obstetric and Gynecological history

She was a para 1+0. She had a spontaneous vaginal delivery at home to a term live female infant who died at 2 weeks (due to neonatal sepsis).

Menarche was at 16 years with a cycle of 28 days and flow of 4 to 5 days. There is no associated dysmenorrhea. There is no history of previous treatment for sexually transmitted infections. Her sexual behavior is low risk.

Family and Social History

She is married class 8 leaver and lives in Mathare with the husband who is a casual labourer. There is no family history of chronic illness. She does not drink alcohol or smoke cigarettes.

Physical examination

She was in good general condition. She was not pale and was afebrile.

Per abdomen examination

The abdomen was uniformly distended and moving with respiration. The liver and spleen were not palpable. The fundal height corresponded to 28 weeks (which was agreeable for her dates). The foetus was in longitudinal lie and cephalic presentation the fetal heart tones were heard and regular at a rate of 138 beats per minute. There was no abdominal tenderness whatsoever.

Vaginal examination

There was a swelling involving the left labia majora. It was 6x4 cm in diameter and tender. The vaginal walls were healthy and the cervix os was closed. The uterus was of normal size and there was no discharge on the examining finger.

Investigations

Haemogram	*Urea, and creatinine*	*Serology for HIV*
Hb 10.0 g\dl	Urea: 2.4mmol/l	Non-reactive.
RBC 4.40 x 10/l	Creatinine: 57 umol/l	
WBC 5.4 x 10/l		
Platelets 269 x 10/l		

Operation: Marsupilization

The patient was put under General anaesthesia and placed in lithotomy position. The perineum was cleaned and draped. It was noted that the bartholins abscess had burst already. An elliptical incision was made at the mucocutaneous junction (just extending from the opening on the abscess). Locules of puss were broken using the small finger. About 10 cc of purulent material was removed. The edges of the abscess were the everted and held in place with interrupted catgut sutures (marsupilization). Haemostasis was achieved and General anaesthesia was reversed.

Post-operative period

The patient was allowed to take orally when fully awake and was put on oral antibiotics, Erythromycin and Flagyl. She was also put on analgesics and asked to do twice daily saline sitz bath for a week.

The post-operative period was uneventful and she was discharged on 29/10/06

Follow-up

She was advised to come to our Antenatal clinic for further follow up.

DISCUSSION

The patient presented was a para 1+0 who was admitted with a bartholins abscess at 29 weeks gestation. Marsupilization was done with uneventful recovery and she was discharged on erythromycin and Flagyl to be seen in the antenatal clinic in 2 weeks time.

Bartholins abscess is a common condition in women of reproductive age (15-49years) According to Mumia the condition formed 1.7 % of the admission to acute gynecological ward at Kenyatta National hospital. Ndede found an incident of 1.9% in the wards. (1,6).

Bartholin's glands were first described by Caspar Bartholin's, a Dutch anatomist, in paired glands that, through their secretion of fluid, maintain the moisture of the vaginal mucosa.

The Bartholin's glands, or the major vestibular glands, are situated beneath the vestibule on either side of the vaginal introitus and measure 0.5x 1.0 cm in diameter. They lie just inferior and lateral to the bulbocavernosus muscle partially covered by the vestibular bulbs.The gland ducts are 1.5 to 2 cm long and open on the sides of the vestibule just outside the lateral margin of the vaginal orifice. The secretion of the Bartholin's glands is a clear, viscid and stringy mucoid substance with an alkaline pH. Secretion occurs during sexual activity. After the age of 30, however, the glands undergo involution and become atrophic and shrunken.Obstruction of the main duct of the Bartholin's gland results in retention of secretions and cystic dilation. Infection is an important cause of obstruction. Infection of the gland commonly occurs at 20-29 years of age, in multiparous women and those of low socio economic status (1). Our patient was 19 years old, para 1+ 0 and of low socio economic status

Ethnicity or marital status does not play a role in development of bartholin's abscess. Prior sexually transmitted infection with gonorrhea or Chlamydia is usually present and bartholin's abscess may

be considered a sexually transmitted infection. Recurrent infection or abscess may be due to scarring of the gland duct by previous infection.

The bacteria involved in bartholin's abscess are the same as those of pelvic inflammatory disease (2,5). Most have both aerobic and anaerobic pathogens, some will have a single organism while in some no pathogen will be isolated on culture. Both Neisseria gonorrhoea and Chlamydia have been isolated from Bartholin's abscess (2,3). Other organisms include E.coli, proteus, Streptococcus faecalis, Staphylococcus aureus, trichomonas and Candida albicans (3,5).

Infection of the Bartholin's gland usually presents with acute symptoms with painful unilateral labial oedema, tenderness and dyspareunia. The surrounding tissues become oedematous and inflamed and a fluctuant mass is usually palpable. This was the case in our patient. Fever occurs in approximately one third of patients. However she did not have a fever.

Differentials include chancroid, gonorrhoea, syphilis, vaginitis and genital warts.

Blood tests are not necessary to evaluate an uncomplicated abscess. Serology for syphilis may be considered. Cultures are rarely useful in treatment. Fluid and cervical specimens may be tested for N. gonorrhoea and Chlamydia species.

Cysts of the Bartholin's gland are sterile and need no treatment during pregnancy. If the cysts are large enough to cause difficult delivery then needle aspiration can be a temporary measure for these. Our patient had an abscess and not just a simple cyst.

A number of surgical techniques are available for the treatment of Bartholin's abscess (4, 7). These include incision and drainage, marsupilization and insertion of a Word catheter.

Word catheter placement

First described in 1952, the technique was modified by Word in 1964. A small catheter is placed in the abscess cavity through a stab incision in the mucosa. This allows for drainage and fistula formation when left in place for several weeks. Simplicity is the techniques main advantage(4).

Incision and drainage

This technique consists of a wide incision and packing.

Marsupilization

Marsupilization avoids excising the gland with the abscess and preserves the secretory function of the gland for lubrication. The procedure is performed under local, regional or general anaesthesia(4). A wedge shaped, vertical incision is made in the vaginal mucosa over the center of the abscess outside the hymenal ring. The incision should be as wide as possible to enhance the

post-operative patency of the stoma. After the abscess is opened and drained of its contents the lining of the abscess is everted and approximated to the vaginal mucosa with interrupted sutures of number 2-0 delayed absorbable material. Sitz baths are encouraged from the 3rd ad 4th day (4).

Marsupilization has a recurrence rate of 10-15%. The four main goals of surgical treatment of Bartholin's abscess are as follows (4).

1. Adequate drainage of the infected gland and abscess.
2. The gland should be preserved so that its secretory function is maintained.
3. Prevention of recurrence by creation of a new ostium or fistula to replace the damaged or occluded duct.
4. Prevention of complications such as necrotising fasciitis.

Antibiotics are also used to treat infection. In pregnancy these could lead to ascending infection to cervicitis (from chlamydia, Neisseria gonnorhoea) that could lead PPROM and chorio-amnonitis. For this reason broad spectrum antibiotics should be used in the management of bartholins abscess in pregnancy.

Prevention of Bartholin's abscess is by prevention of sexually transmitted infections (4).

References

1. Aghajamian A, Bernstein L, Grimes DA: Bartholin's duct abscess and cyst: A case control study. South Med J 1994; 87: 26.
2. Brook I: Anaerobic and aerobic microbiology of Bartholin's abscess. Surg Gynaecol Obstet 1989; 169:32.
3. Wren MWD: Bacteriological finding in culture of clinical material from Bartholin's abscess. J Clin Path 30: 1025-27, 1977.
4. Andersen PG et al : Treatment of Bartholin's abscess: Marsupilization versus incision, curettage and suture under antibiotic cover. A randomized study with 6 months follow up. Acta Obstet Gynecol Scand 1992; 71: 59.
5. Lee VH, Ranken JJ, Alpert et al: Microbiological investigations of Bartholin's gland abscess and cyst. Am J Obstet Gynecol 1977; 129: 150.
6. Mumia JA: Bartholin's abscess at Kenyatta National Hospital. Mmed Thesis. University of Nairobi. 1981.
7. Heah J. Methods of treatment of cysts and abscesses of Bartholin's gland. BJOG 1984;63(6):820-4.

Obstetric 7

SUCCESSFUL VAGINAL BIRTH AFTER CAESAREAN SECTION (VBAC) - LIVE BABY

NAME: M.W IP NO. : 1125571

AGE: 22 YEARS L.M.P: 12/01/06

D.O.A: 26/10/2006. E.D.D: 8/10/06

D.O.D: 31/10/2006 GESTATION: 39+ WEEKS

PARITY: 1+1

Presenting complaint

She was admitted to labor ward from home with history of lower abdominal pains that were increasing in frequency and intensity.

History of presenting complaint

MW was admitted with complaints of intermittent regular lower abdominal pains radiating to the back which were increasing in intensity and frequency. There was no P.V bleeding or drainage of liquor. Fetal movements were appreciated and were described as normal. There were no urinary symptoms.

Past obstetrics and gynaecologic history

She was a para 1+1 with 1 living child. Her first delivery was in 2002.The delivery was a cesarean birth due to breech presentation in labour(KNH) and the outcome was a live male infant, birth weight-3.3kgs , with good apgar scores and the baby was alive and well. Her pueperium was uneventful and she was discharged home on the 3rd post operative day. In 2005 she had a complete abortion at 3 months, with uneventful recovery.

She attained menarche at 14 years. Prior to her pregnancy, her periods were regular of 4 days duration and frequency of 27-30 days. She had used combined oral contraceptive pills in between her pregnancies.

History of present pregnancy

Her last menstrual period was 12/01/2006 and her expected date of delivery was 8/10/2006. She had been supervised at city council clinic in town during her antenatal period where she was booked at 28weeks gestation and she had six visits.

The antenatal profiles were as follows: Hb: 12.4g/dl, Blood group: A+ve, HIV –ve and VDRL-ve. Antenatal period was uneventful.

Past medical history

Nil of significance.

Family and social history

She was a married woman who stayed with her husband and children in Buruburu. She never drunk alcohol nor smoked. There was no family history of chronic illnesses.

Review of systems

This was non-contributory.

Physical examination

General examination findings

She was in good general condition, she was not pale, not jaundiced and had no oedema. The vital signs were as follows: Pulse: 88/min Resp.rate:18/min BP: 100/60mmHg temp: 36.2^{0}C

Abdominal examination

The abdomen was uniformly distended and pfannestiel scar was visualized. Fundal height was term, the lie longitudinal, the presentation was cephalic and the descent was 4/5. Fetal heart was heard and regular at 140 bpm. There were no contractions observed. There was no scar tenderness and fetal parts were not easily palpable. Estimated fetal weight was 3000grams.The liver and spleen was not palpable.

Vaginal examination

The external genitalia were normal. There was no drainage of liquor or bleeding through the introitus. The cervix was admitting a tip of a finger but seemed well effaced, central and soft. The pelvis was found to be adequate with a diagonal conjugate of 13.5cm, The 4 knuckles could fit in the inter-tuberous space.

Other systems were essentially normal.

Diagnosis

An impression of a para 1+1 with one previous Caesarian section scar in early labor at term with an adequate pelvis was made.

MANAGEMENT

She was admitted and counseled for VBAC and likewise counseled on the possibility of an emergency cesarean section. The mother and the primary nurse were informed to report about scar tenderness in between uterine contractions. IV access was obtained and blood for grouping and cross match was drawn. 2 units of whole blood were requested. She was started on 5% dextrose and advised to stay nil by mouth. Informed consent for emergency caesarian section was obtained in case of failed 'trial of scar' and the obstetrician, anesthesiologist and the maternity theatre staff were alerted. She was advised to lie on her left lateral position. Strict partograph was instituted with half hourly monitoring of maternal vital signs and fetal heart tones.

She was reviewed 4hrs later and was found to have 3 contractions in 10mins lasting about 30secs and the descent was 3/5.There was no scar tenderness and fetal heart was heard and regular. On VE the cervix was 4cm dilated with no caput and no molding and the head was in occiput anterior position. She was maintained on 5% dextrose and allowed to progress in labor.

Four hours later she felt the urge of bearing down and the cervix was found to be 10cm dilated. She was wheeled to delivery room where she delivered by spontaneous vertex delivery and the outcome was a live male infant apgar score of 9 at 1min and 10 at 5min with a birth weight of 2710 grams. No gross abnormality was noted. Second stage of labor lasted 30 minutes. The placenta was delivered by controlled cord traction. It was expelled complete with membranes and was found to be grossly normal. The uterus was not explored. Estimated blood loss was 450ml and the perineum was intact. Third stage lasted 15 minutes.

She was given intramuscular injection of 10mg of syntocinon with delivery of the placenta. Her post delivery pulse was 80/min and of good volume and the blood pressure was 110/60 mm/Hg.

She was discharged from labor ward 2hrs later during which examination findings were as follows: She was not pale, no edema, the breasts were soft and not active, the uterus was well contracted at 20wks and the lochia was rubra and minimal. There was no calf swelling or tenderness.

Immediate pueperium was uneventful and she was discharged home 2days later for review in postnatal clinic after 6 weeks.

DISCUSSION

M.W was a Para 1+1 Gravida 3 with one previous Caesarean section scar. She was admitted at 39+ weeks in labor and she had a successful vaginal birth after caesarean section (VBAC). She delivered by SVD, the outcome being a live male infant apgar score of 9 at 1min and 10 at 5min with a birth weight of 2710 grams. Both the mother and baby did well and were discharged home.

Patients with prior cesarean deliveries require special management, both antenatally and in labor and delivery. Early in their prenatal care, patients are catalogued according to the preexisting risk factors for both successful VBAC and uterine rupture. If uncertain about the prior uterine incision facts, operative notes from patients' cesarean delivery are obtained. When all obstetric history is obtained, patients are counseled regarding the risks and benefits of undergoing a trial of labor after cesarean delivery, and the particular mode of delivery is planned with patients (1). This was the case with JN whereby details of her previous surgery were obtained. Counseling was done and VBAC was agreed upon.

"Once a cesarean, always a cesarean." From the time they were spoken in 1916 to the New York Association of Obstetricians & Gynecologists over the ensuing 50-60 years, these words reflected most of obstetricians' management of patients with a prior cesarean delivery. Data from developed as well as developing countries indicate a steady rise in caesarean section rates over the years. Among the developed countries, the lowest caesarean section rates have been recorded in Japan and Netherlands (6-7%). In the USA and Canada, the rate was 25% in 1988, rising from less than 5% in the early 1970s. Only 3% of live-born infants were delivered vaginally after the mother had undergone a prior cesarean delivery (2). Rates are known to be higher among private patients e.g. in

San Paulo, Brazil, rates as high as 75% have been recorded among the private patients. In developing countries rates vary from one region to another. In Nigeria the rate is 6.9% in Ilum and 19.8% in Ibadin. In Kenyatta National Hospital, it was 17.8% in 1980 of which 59.8% were repeat sections (20). In 1989, the rate was found to be 21.1% (21).

Although attempts at a trial of labor after a cesarean birth have become accepted practice, the rate of successful vaginal birth after cesarean delivery (VBAC), as well as the rate of attempted VBACs, has decreased during the past 10 years. Whereas 40-50% of women attempted VBAC in 1996, as few as 20% of patients with a prior cesarean delivery attempted a trial of labor in 2002 (3).

Several factors have contributed to this decline. As practitioners experience complications related to managing patients undergoing trials of labor after cesarean delivery, they are less likely to allow new patients to undergo a trial of labor. In addition, 1999 guidelines from the American College of Obstetricians and Gynecologists (ACOG) clearly state that patients undergoing a trial of labor after cesarean delivery require the presence of an obstetrician, an anesthesiologist, and/or a staff capable of performing an emergency cesarean delivery throughout the patient's active phase of labor (4).

This was the case in our patient whereby informed consent for emergency caesarian section was obtained and the obstetrician, anesthesiologist and the maternity theatre staff were alerted.

Many obstetric practices and healthcare institutions have adopted a separate consent form for patients wishing to attempt a VBAC. While consent helps to formalize counseling, documentation of the overall risks quoted to the patient, specifically mentioning the individual's risk factors is all that is truly necessary. However, because of medical-legal concerns, formalized written consent forms, and even video-taped counseling-consent interactions, are used by clinicians (5).

The predictors of a successful trial of labor after cesarean delivery

Maternal characteristics:

Maternal age has also been examined in several studies in VBAC literature. Adjusting for confounding factors, women older than 40 years who have had a prior cesarean delivery have an almost 3-fold higher risk for a failed trial of labor compared with women younger than 40 years.(6) The patient was 22 years old and this could have contributed to the success of VBAC. Maternal race and ethnicity have been examined as a predictor for VBAC in the setting of trial of

labor and have not generally been noted to be a strong predictor. However, in the recent Maternal-Fetal Medicine Unit (MFMU) Cesarean Registry, both Hispanic ethnicity and African American ethnicity were associated with lower rates of successful trial of labor. Whether this is due to actual biologic reasons or rather ethnicity acting as a proxy for some other factor or factors remains to be elucidated. However this was not the case in our patient despite being of African origin (7).

Birth weight:

Birth weight greater than 4000 g is associated with an almost 4-fold higher risk of cesarean birth among nulliparous women. Several studies have demonstrated a difference in VBAC rates between patients with a birth weight greater than 4000 g and those with a lower birth weight. Consistent with these findings, several studies have demonstrated a higher failure of a trial of labor with increasing birth weight (8).

M.W delivered a 2710 grams baby .Besides, her previous vaginal delivery weighed 3400 grams.

Obstetric history and Indications for prior cesarean delivery:

Obstetric history is enormously important in terms of risk factors for a successful trial of labor. Predictors for increased success include a nonrecurring indication for prior cesarean delivery (e.g., breech presentation, placenta previa) and prior vaginal delivery. A history of cephalopelvic disproportion (CPD), failure to progress, no prior vaginal deliveries, or a prior cesarean delivery performed in the second stage of labor are negative predictors of success in a subsequent trial of labor

Several studies have examined indications for prior cesarean delivery as a predictor of outcome in a subsequent trial of labor. In all studies, CPD had the lowest VBAC success rate (60-65%). Fetal distress (e.g., non reassuring fetal testing) had the second lowest success rate of VBAC (69-73%). Non recurrent indications, such as breech birth, herpes, and placenta previa, were associated with the highest rates of success (77-89%).

Failure to progress, CPD, or dystocia as indications for prior cesarean delivery are also associated with a higher proportion of patients not attempting a trial of labor after cesarean birth. In a meta-analysis of the existing literature prior to 1990, Rosen et al demonstrated that women whose prior

cesarean delivery was performed for CPD were twice as likely to have an unsuccessful trial of labor (9).

In this patient, the positive predictor for success of VBAC was breech presentation as the non recurring indication for previous c-section.

Prior vaginal delivery:

Patients with a prior vaginal delivery have higher rates of successful VBAC compared with patients without a prior vaginal birth. A 2000 study by Zelop et al demonstrated that patients with a prior vaginal delivery had a 0.2% rate of rupture compared with 1.1% for patients with no prior vaginal delivery.

Furthermore, women with a successful VBAC have a higher success rate in a subsequent trial of labor compared with women whose vaginal delivery was prior to cesarean delivery. In an unadjusted comparison, patients with 1 prior vaginal delivery had an 89% VBAC success rate compared with a 70% success rate in patients without a prior vaginal delivery. Among patients with a prior VBAC, the success rate is 93% compared with 85% in patients with a vaginal delivery prior to their cesarean birth but no prior VBAC. (10)

Cervical dilation at prior cesarean delivery:

In one study, the degree of cervical dilation in the prior delivery was directly associated with the likelihood of success in the subsequent trial of labor. For example, 67% of patients who were dilated 5 cm or less at the time of their delivery had a successful VBAC compared with 73% of patients who were dilated 6-9 cm. The success rate is much lower for patients whose labor arrested in the second stage; only 13% of patients who were fully dilated at the time of their prior delivery had a successful VBAC.

In a similar study, patients who had their prior cesarean delivery in the first stage of labor had a lower rate of cesarean delivery than those who had their prior cesarean delivery in the second stage of labor (11).

Gestational age:

Increasing gestational age is associated with a decreased rate of successful VBAC. Three potential factors are related to the association of increasing gestational age with an increased rate of cesarean

delivery: increasing birth weight, increased risk of fetal intolerance of labor, and increased need for induction of labor. However, in a recent study that controlled for both birth weight and induction/augmentation of labor, gestational age of greater than 41 weeks was still associated with failed VBAC (12).In this patient, gestation age was 39 weeks making it probable for successful VBAC.

Interpregnancy interval:

The timing between pregnancies has recently become an interesting predictor for a number of obstetric outcomes, VBAC success among them. Several studies have demonstrated that the shorter the amount of time between the cesarean delivery and the subsequent delivery, the higher the rate of uterine rupture. Commonly, thresholds of 18 and 24 months have been examined. Adjusted odds ratios range from 2.5-3 for an increased rate of uterine rupture in the women with less time between deliveries. The biologic plausibility here is related to the amount of time required for the uterine scar to heal completely. In one analysis, women who had an interpregnancy interval of more than 18 months had an 86% chance of VBAC success, while women whose interpregnancy interval was less than 18 months had a VBAC success rate of 79%. This difference was not statistically significant, and whether interpregnancy interval does have any effect on success or rather has an effect on the risk for uterine rupture is unclear (14). In our patient, the interpregnancy interval was 4 years thus reducing the risk of uterine rupture.

Other factors to look at when considering trial of scar:

Classical uterine incision:

Unquestionably, practitioners do not feel safe allowing a patient who has had a prior classical uterine incision (i.e., a vertical incision that has extended above the insertion of the round ligaments) to undergo a trial of labor. Patients with a prior classical uterine incision have a higher rate of uterine rupture in subsequent pregnancies. Because these patients can sustain a uterine rupture prior to labor, they are often delivered at 36-37 weeks' gestation. Although available data are limited, the risk of uterine rupture in this group of patients is estimated at 6-12% (15).

Low transverse (Kerr) uterine incision:

Most babies delivered abdominally are delivered through a transverse incision in the lower uterine segment (Kerr uterine incision). In several large retrospective cohort studies, the reported rate of uterine rupture is 0.3-1%. Rates of 0.5-1% (1 in 200 to 1 in 100) are commonly used to counsel

patients with no other additional risk factors. This patient had a lower uterine segment c-section thus the risk of uterine rupture was markedly reduced (15)

Low vertical (Krönig) uterine incision:

Retrospective cohort studies have demonstrated that the risk of uterine rupture is no greater for patients who have had a vertical incision in the lower uterine segment than those who have had a transverse incision. The rate of uterine rupture from these studies is 0.8-1.3%. When comparing patients with prior Krönig uterine incisions to patients with low transverse incisions, no statistical difference exists in either univariate or multivariate analyses controlling for the confounding factors of obstetrical history, induction of labor, birth weight, and length of labor (15).

Unknown uterine incision:

When an obstetrician cannot obtain an operative report of a patient's prior cesarean delivery, obstetric history may be helpful in determining the type of uterine incision. For example, a patient who underwent a cesarean delivery for a breech presentation at 28 weeks' gestation has a much higher risk of a vertical uterine incision than the patient at term with arrest of dilation.

Because most cesarean deliveries are via low transverse incisions, the risk of uterine rupture for patients with an unknown uterine scar is usually similar to that of patients who have had a prior transverse incision. Several studies examining this issue have demonstrated that the rate of rupture for patients with an unknown uterine incision is approximately 0.6%. A case-control study of patients with and without uterine rupture did not find unknown uterine incision to be a risk factor compared with low transverse incision (Leung, Farmer, et al, 1993) (15). However in our patient, the details of the previous surgery were well documented in the discharge sheet.

Number of prior cesarean deliveries:

Patients with more than 1 prior cesarean delivery are at increased risk of uterine rupture. The unadjusted rate of uterine rupture for patients with 2 prior uterine incisions ranges from 1.8-3.7%. A recent analysis demonstrated that when potential confounding variables (e.g., prior vaginal delivery) are controlled for, patients who have had 2 prior cesarean deliveries have 5 times the risk of uterine rupture compared with patients who have had only 1 prior cesarean delivery (16).

M.W had one previous scar which made her suitable for VBAC.

Single-layer uterine closure:

While traditionally the uterine incision had been closed in several layers, in the 1990s, physicians at many institutions began closing the Kerr incision in a single layer. Because the lower uterine segment is quite thin, a single layer often afforded adequate haemostasis. Several recent studies have compared women whose uterine incision was closed in a single layer with those whose uterine incision was closed in 2 layers. An adjusted odds ratio of 3.95 for uterine rupture has been estimated for those women who only have a single-layer closure (17). No details were available about the number of layers that MW had during closure of her uterus in the previous surgery thus it may not be possible to attribute the success of VBAC to this factor.

Prior infection:

A recent study demonstrated that women who had an infection at the time of the cesarean delivery have an increased rate of uterine rupture in a subsequent trial of labor. The assumed causal mechanism is poor healing of the uterine incision secondary to the infection (18).

M.W had uneventful pueperium in her previous birth and no sepsis was reported thus making her suitable for VBAC in her current pregnancy.

Clinically, the patient is observed closely for signs of uterine rupture. Harbingers of uterine rupture include the following:

- Acute abdominal pain
- A popping sensation
- Palpation of fetal parts outside the uterus upon Leopold maneuvers
- Repetitive or prolonged fetal heart rate deceleration
- High presenting part upon vaginal examination
- Vaginal bleeding

Any of these findings are treated as a possible uterine rupture until another source for the finding has been identified. Rupture requires immediate delivery (19).

MW was observed for these factors while in labor and they were absent thus labor was allowed to progress.

REFERENCES

1. Granovsky-Grisaru S., Shaya M., Diamant Y.Z.: The management of labor in women with more than one uterine scar: is a repeat cesarean section really the only "safe" option? J Perinat Med 1994; 22(1): 13-7
2. Flamm B.L.: Once a cesarean, always a controversy. Obstet Gynecol 1997 Aug; 90(2): 312-5
3. Porreco R.P., Thorp J.A.: The cesarean birth epidemic: trends, causes, and solutions. Am J Obstet Gynecol 1996 Aug; 175(2): 369-74.
4. American College of Obstetricians and Gynecologists: ACOG criteria set. Ambulatory care criteria set: obtaining informed consent. Number 26, August 1997. Committee on Quality Assessment. Int J Gynaecol Obstet 1997 Dec; 59(3): 279-80.
5. American College of Obstetricians and Gynecologists: ACOG practice bulletin. Vaginal birth after previous cesarean delivery. Number 5, July 1999 (replaces practice bulletin number 2, October 1998). Clinical management guidelines for obstetrician-gynecologists. Int J Gynaecol Obstet 1999 Aug; 66(2): 197-204
6. Landon M.B., Leindecker S., Spong C.Y. et al: The MFMU Cesarean Registry: factors affecting the success of trial of labor after previous cesarean delivery. Am J Obstet Gynecol 2005 Sep; 193(3 Pt 2): 1016-23.
7. Dodd J., Crowther C.: Vaginal birth after Caesarean versus elective repeat Caesarean for women with a single prior Caesarean birth: a systematic review of the literature. Aust N Z J Obstet Gynaecol 2004 Oct; 44(5): 387-91
8. Elkousy M.A., Sammel M., Stevens E. et al: The effect of birth weight on vaginal birth after cesarean delivery success rates. Am J Obstet Gynecol 2003 Mar; 188(3): 824-30
9. Eglinton G.S.: Effect of previous indications for cesarean on subsequent outcome. In: Phelan JP, Clark SL, eds. Cesarean Delivery. New York, NY: Elsevier; 1988: 476-83.
10. Caughey A.B., Shipp T.D., Repke J.T. et al: Trial of labor after cesarean delivery: the effect of previous vaginal delivery. Am J Obstet Gynecol 1998 Oct; 179(4): 938-41
12. Caughey A.B., Musci T.J.: Complications of term pregnancies beyond 37 weeks of gestation. Obstet Gynecol 2004 Jan; 103(1): 57-62
13. Gyamfi C., Juhasz G., Gyamfi P., Stone J.L.: Increased success of trial of labor after previous vaginal birth after cesarean. Obstet Gynecol 2004 Oct; 104(4): 715-9

14. Huang W.H., Nakashima D.K., Rumney P.J. et al: Interdelivery interval and the success of vaginal birth after cesarean delivery. Obstet Gynecol 2002 Jan; 99(1): 41-4

15. Grubb D.K., Kjos S.L., Paul RH.: Latent labor with an unknown uterine scar. Obstet Gynecol 1996 Sep; 88(3): 351-5

16. American College of Obstetricians and Gynecologists: ACOG criteria set. Ambulatory care criteria set: obtaining informed consent. Number 26, August 1997. Committee on Quality Assessment. Int J Gynaecol Obstet 1997 Dec; 59(3): 279-80

17. Bujold E., Bujold C., Hamilton E.F. et al: The impact of a single-layer or double-layer closure on uterine rupture. Am J Obstet Gynecol 2002 Jun; 186(6): 1326-30.

18. Shipp T.D., Zelop C., Cohen A., Repke J.T.: Post-cesarean delivery fever and uterine rupture in a subsequent trial of labor. Obstet Gynecol 2003 Jan; 101(1): 136-9

19. Wilson A.L.: Labor and delivery after cesarean section. Am J Obstet Gynecol 1951 Dec; 62(6): 1225-33.

20. Karanja J.K. A review of caesarean delivery in KNH in 1980. M.Med thesis, UoN 1982.

21. Bodaya C.,Bartha T.L., Rodrique I. et al. Trial of scar labour after caesarean section in patients with or without previous vaginal delivery. Int J. Obstet Gynecol. 1992;38:285-289.

Obstetric 8

RUPTURED UTERUS IN 1 PREVIOUS SCAR-SUBTOTAL HYSTERECTOMY

NAME: T.N IPNO: 1082730.

AGE: 41 YEARS L.M.P: NOT SURE

D.O.A:28/03/06 QUICKENING; SEPT 2005

D.O.D:06/04/06

PARITY: 3+0G4

Presenting complain

She presented with abdominal pain for the last 13 hours and per vaginal bleeding for four hours prior to admission.

History of presenting complain

She had gone to a traditional birth attendant (TBA) for delivery at the onset of labour pains. While at the TBA she started having PV bleeding which was increasing in amount.13hours later she had not delivered ands she decided to seek care in KNH.There had been no history of PV bleeding during the pregnancy. She reported no fetal movements for 10 hours prior to admoission. Initially she had been having labour like pains but later on she reported to have develope4d generalized pain.

History of the current pregnancy

She attended ANC in Embakasi health centre. She had attended 4 times. Antenatal profiles were not done. The card was not available for perusal

Obstetrics and gynaecology history

She was Para 3+0 gravida 4.Her first delivery was by cesarean section in 1992 due to breech presentation in labour at term. The baby succumbed after 3 days. Her second delivery was VBAC at Pumwani in 1994 to a live male infant who is well while her third delivery was also VBAC in a private clinic in 1997.The child is well. Birth weights for both babies were not known. She was not sure of her dates. Her menarche was at 16 years, had irregular cycles after 22- 34 days and bleeding lasted 3 days. She had used depo provera and stopped 2 years ago due to persistent bleeding. She never sought advice from health workers.

Past medical history

This was not significant.

Family and social history

She divorced in 1994.She was a form four leaver and was a vegetable vendor. She did not smoke or drink alcohol.

PHYSICAL EXAMINATION

General examination

She was sick looking, pale, restless with cold extremities. Her pulse was thready and the rate was 112 in a minute. The BP was 90/50 mmhg.

Respiratory, cardiovacular and CNS

Were essentially normal.

Abdominal examination

The abdomen was uniformly distended, term with generalized tenderness. Fetal parts were easily palpable and there was no fetal heart heard.

Speculum examination

There was active bleeding which was noted together with numerous clots. The cervix was not visualized.

Diagnosis

A diagnosis of antepartum hemorrhage secondary to ruptured uterus was made.

MANAGEMENT

IV access was established and fluid resuscitation commenced. Grouping and cross matching was done and 4 units of whole blood and 2 units of fresh frozen plasma requested. Informed Consent was obtained and she was wheeled to theatre for laparotomy. The abdomen was opened via midline sub umbilical incision. There was haemoperitoneum; the fetus was in the peritoneal cavity FSB weighing 3150g, ruptured uterus through the previous scar with extension to and involvement

of the upper posterior segment and the bladder dome. A decision to do subtotal hysterectomy was reached and done. The bladder was repaired in 2 layers. A drain was left insitu and an indwelling urethral catheter inserted to be retained for 14 days. Intraoperatively she was transfused 4 units of whole blood and 1 unit of fresh frozen plasma. General anesthesia was uneventful.

Post operative care

She had an uneventful postoperative course. She was appraised of the intraoperative findings and the surgery done. The drain was removed after 48 hours and this time there was no active drainage noted.

The check HB at day 3 was 8.2g% and was put on haematinics. She was put on bromocriptine because her breasts became engorged. She was discharged home on day 6 for follow up in the postnatal clinic after I week.

Post natal follow up.

She was reviewed after one week after discharge. The operation site was well healed. The urethral catheter was removed. She was advised about yearly Pap smear screening.

DISCUSSION

Presented is N.M, 43 years Para 4+0, who had rupture of uterus while in labour assisted by traditional birth attendant. Subtotal hysterectomy was done and repair of bladder. She was transfused 4 units of whole blood.

Uterine rupture is defined as dissolution in the continuity of the uterine wall any time beyond 28 weeks of pregnancy. Injury to the wall of the uterus in the early months is called perforation either instrumental or perforating hydatidiform mole [1].Our patient had rupture of uterus at term.

Rupture of the pregnant uterus is a potential obstetric catastrophe and a major cause of maternal death. In the USA the maternal mortality rate as a result of ruptured uterus is 4.2% while the perinatal mortality rate is about 50-75 %.[2, 3]. At Kenyatta National Hospital, ruptured uterus was responsible for 3% of maternal deaths in the period 1995-1999[4].

The incidence of uterine rupture is reported to be between 1:1148 and 1:2250 deliveries in the USA [2]. In the Nairobi Birth Survey of 1983, the incidence of uterine rupture was 0.06 % [5].

Uterine rupture is classified as either complete where all layers of the uterine wall are separated or incomplete where uterine muscle are separated but visceral peritoneum is intact[3]. Incomplete rupture is also referred as uterine dehiscence or occult rupture[2].Morbidity and mortality are appreciably greater when rupture is complete[3]. Our patient had complete uterine rupture and the baby was found outside the uterus.

Risk factor for uterine rupture include history of prior hysterotomy (caesarean section, myomectomy, metroplasty, cornual resection), trauma (motor vehicle accident, rotational forceps, extension of a cervical laceration), uterine over-distension (hydramnios, multiple gestation, macrosomia), uterine anomalies, placenta percreta, choriocarcinoma and grandmultiparity. Other operative procedure that may have damaged the uterus are vigorous curettage, induced abortion and manual removal of the placenta[1,2,3] .The case presented had a previous caesarean section scar.

Ruptures usually occur during the course of labour. One notable exception is scars from classical caesarean section (or hysterotomy), one third of which rupture during the third trimester before term and before the onset of labour. Other causes of rupture without labour are placenta percreta, invasive mole, choriocarcinoma and cornual pregnancy[2].Our patient ruptured while in labour.

During labour improper administration of an oxytocic agent or an inept attempt at operative delivery vaginal delivery may cause rupture of the uterus. Other maneuvers that impose risk of rupture are internal podalic version and extraction, difficult forceps, destructive operations and maneuvers to relieve shoulder dystocia[2].Tumultuous labour, excessive fundal pressure or violent bearing down efforts and neglected obstructed labour increase the risk of uterine rupture. Causes of obstructed labour include contracted pelvis, fetal macrosomia, and brow or face presentation, hydrocephalus or tumors involving the birth canal[2] .Its difficult to know the circumstances surrounding uterine rupture for our patient. However the birth attendant was not skilled and labour was prolonged.

There are no reliable signs of impeding uterine rupture.[2, 3]

The warning signs of rupture of scar during labour are foetal heart rate abnormalities, maternal tachycardia, and vague pain continuing even in between uterine contractions, suprapubic

tenderness, vaginal bleeding and gross hematuria [1,2].Our patient presented with continuous abdominal pains and vaginal bleeding.

The classic findings of spontaneous uterine rupture during labour are suprapubic pain and tenderness, cessation of uterine contractions, disappearance of fetal heart tones, recession of the presenting part and vaginal haemorrhage followed by signs and symptoms of hypovolaemic shock and haemoperitoneum [2].
Our patient was in shock, the abdomen was tender, fetal parts easily palpable and there was no fetal heart.

Depending upon the state of the clinical condition, either resuscitation is to be done followed by laparatomy or in acute conditions resuscitation and laparatomy are to be done simultaneously.
Following laparatomy, three surgical options are available: -[2,3]

- Hysterectomy. This is the preferred treatment. Either total or subtotal hysterectomy can be employed depending on the site of rupture and the patient's condition.
- Repair and sterilization is mostly done in patients with a clean cut scar rupture having desired number of children.
- Repair of the rupture without tubal sterilization is mostly applicable to a rupture where the margins are clean and more children are desired. Repair is done by excision of the fibrous tissue at the margins. In such cases, however, there is chance of peritonitis and septicemia. Elective caesarean section in subsequent pregnancy is mandatory because of the high risk of rupture estimated to be about 20%.

In long neglected and badly infected cases survival may be improved by limiting the surgical procedure to repair of the rupture and antibiotic rupture [2].Our patient was done subtotal hysterectomy.

In cases of scar separation without bleeding following VBAC, exploratory laparatomy is not indicated [3].

Complications of ruptured uterus are haemorrhage, shock, postoperative infection, ureteral damage, thrombophlebitis, amniotic fluid embolus, disseminated intravascular coagulation, pituitary failure, and death. If the patient survives, infertility or sterility may results.[2, 3]

Prevention of uterine rupture includes skilled attendance, proper administration of oxytocin during labour, foetal weight estimation to avoid traumatic delivery, proper assessment prior to use of assisted operative techniques and proper closure of caesarean section incision [2].

REFERENCES

1. Dutta DC: Rupture of the Uterus. In: Textbook of Obstetrics, 6th edition, 2004, New Central Book Agency (P) Ltd, Calcutta, India Pg 427-432.
2. Claydon CS, Pernoll ML. Third Trimester Vaginal Bleeding. In: DeCherney AH, Nathan L. (Eds). Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th edition, 2003, McGraw-Hill, Pg 365-367
3. Cunningham FG Gant NF Leveno KJ et al (Eds). Obstetric Haemorrhage.Rupture of the uterus In: Williams Obstetrics, 22nd edition, 2005. McGraw-Hill Companies, Pg 836-838.
4. Oyieke J.B.O., Obore, S.and Kigondu, C.S.Millenium development goal 5:A review of maternal mortality at the Kenyatta National Hospital ,Nairobi. East Afr.Med.J.2006;83:4-9
5. Mati JKG, Aggarwal VP, Sanghvi HCG, et al. The Nairobi Birth Survey III-Labour and Delivery. J Obtet Gynelol Centr. Afr 1983 2(2):47-56.

Obstetric 9

HYPOTHYROIDISM IN PREGNANCY.

NAME: AK IPNO 1108014

AGE: 36 LMP: 01/11/2005

DOA: 01 /08/ 2005 EDD 08/08/2006

DOD: 06 /08/06 GESTATION 37+ WEEKS.

PARA 4+0

Diagnosis: Hypothyroidism in pregnancy

The patient was admitted through the ANC clinic as a 3 previous scar at term with hypothyroid goitre on thyroxine for elective c/s.

History of current pregnancy.

Her last menstrual period was on 1st Nov 2005and her expected date of delivery. She was therefore at a gestation of 37 weeks.

She was followed up in a private clinic in Nairobi. She was noted to have goitre in early pregnancy. Thyroid function tests done which revealed a raised TSH and a low free T_4 with no other symptom of hypothyroidism. Her blood pressure and cardiac functions have been normal through the pregnancy.

A.K was put on thyroxine 25 mg once a day at a gestation of 12 weeks. These was later increased to 50mg and maintained at this through the pregnancy. Report of the serial thyrotropin and serum T4 levels were not available in the file.

Antenatal profile done 15/03.06 Vdrl negative, P24 negative, HEP B negative, B/G AB+ve, HB 13.7 g/dl. RBC was normocytic normochromic picture.

Urinalysis no leucocytes, nil proteins, ketones sugars. Microscopy no pus cells.

She only came to KNH antenatal clinic once to book for delivery and also informed as of her desire to have sterilization during the planed delivery.

Past Obstetric and gynecological history.

She was Para 4+2. Her last delivery was in 2001 to a live male infant by elective cesarean section due to 2 previous scars at term, the boy is alive and well. In 1997 she delivered a live female infant by cesarean section due to prolonged labour who is alive and well. In 1999 she had a fresh still birth

due to cord prolapse a cesarean section had been done to try save the baby. A.K had an abortion in 2004 at 14 weeks at this time she had not been evaluated for hypothyroidism and she had not noted the anterior neck mass she now has. She attained her menarche at 14 years. Her menses last for 4 days and come after every 28days; they are regular with no associated dysmenorrhea. She had used oral contraception between the first pregnancy and the second. She had not done a pap smear before.

Past medical history

She had no significant past medical or surgical history.

Family social history.

She was a housewife who stays in Embakasi. Her husband is a policeman . She neither drinks alcohol nor smokes cigarettes. There is no history of chronic illnesses in the family.

Physical examination

General examination

She was in good general condition .She was not pale, not jaundiced, not wasted, no lymphadenopathy and no edema. She had an anterior neck mass that was central and diffuse but moved on swallowing. It had no bruit was heard over it. She was relaxed, not apprehensive and did not have exophthalmos. Vital signs were normal.

Respiratory/cardiovascular and CNS

All were normal.

Abdomen

It was uniformly distended with a midline subumbilical scar. The fundal height corresponding to term pregnancy. The fetus was in longitudinal lie and cephalic presentation. Head was high. Fetal heart was 138 beats/min and regular

Vaginal examination

Was not indicated

Investigatiosn done (*15*/03/06)

Thyroid function test

T3 1.0 nmol/l (0.9-2.5 nmol/l)

T4 87 nmol/l (50-130 nmol/l)

TSH 1.2 mol/l (0.3-7 mol/l)

Hemoglobin 13.3g/dl (15/07/2006)

Renal function tests.(15/07/2006)

Urea 2.0mmol/l

Sodium 138mmol/l

Potassium 4.2 mmol/l

Chloride 98mmol/l

Creatinine 63. umol/l

Obstetric ultrasound done on (19/05/06) showed a single fetus in cephalic presentation. Fetal maturity at 29 weeks and 4 days EDD 31/07/2006. Heart rate at 150 beats /minute and estimated fetal weight of 1313 grams. There is no fetal body or neural tube abnormality noted. The liquor is adequate and there are normal fetal movements seen. The placenta is fundal anterior and not low lying. BPP 10/10 Restrictive index of 0.63. there is no uterine wall abnormality.

Conclusion: Normal single intra-uterine pregnancy at 29 weeks and 4 days.

Diagnosis

Hypothyroidism at 39 weeks in III previous scar now euthyroid.

MANAGEMENT

She was admitted to the labour ward for elective caesarean section. She was scheduled for an elective caesarean section the following day. She gave consent for caesarean, 2 units of blood were crossmatched. Haemoglobin was 13.3g/dl and urea, creatinine and electrolytes were normal. Half an hour before surgery she was premeditated with atropine 0.6mg intramuscularly.

In theatre, vulvovaginal toilet and aseptic catheterizations were done. The abdomen was cleaned and draped with sterile towels. Under general anaesthesia, the abdomen was open through a repeat midline incision. A lower uterine segment caesarean section was performed The outcome was a live female infant with an Apgar score of 9 in 1 minute and 10 in 5 minutes and weighed 2700g. The infant was reviewed by the pediatrician and was found to be normal. The placenta was delivered and was complete and grossly normal. The uterus was closed and haemostasis achieved. Bilateral tubal ligation was done. The abdomen was closed in layers after ascertaining swabs and

instruments count. Vulvovaginal toilet was done and the catheter removed. She had an estimated blood loss of 400ml.

Post operative management

The vital sign were observed half hourly until she was fully conscious, one hourly for 4 hours and then 4-hourly thereafter. She was put on intravenous fluids, 1 litre of dextrose alternating with normal saline 8-hourly for 24 hours.

Analgesia was through intramuscular pethidine 100 mg 8-hourly for the first 24 hours then oral ibuprofen 400 mg 8-hourly. She was also put on intravenous augmentin for 48 hours and was then changed to orals. She continued with her thyroxine at 50 mg once daily.

The patient did well postoperatively. She was informed of the BTL that had been done as per her consent. They were discharged home through the post-natal clinic. She was to be seen in one week's time with a thyroid function hormonal profile to taper down her drugs. She was encouraged to breast feed and that the drugs will have no effect on her baby.

Post-natal follow-up

She did not come back to the clinic for her follow-up.

DISCUSSION

Maternal hypothyroidism occurs in 2.5% of all pregnancies. In the USA an estimated 1-2% of all pregnant women are on levothyroxine (1). Most of these develop in the reproductive age group and are auto-immune related disorders of the thyroid gland(58%) have thyroid anti-bodies (1, 2, 7). They may be goitrious hypothyroidism or atrophic hypothyroidism. Our patient had the goitrious type.

These could also be divided into Overt and sub-clinical hypothyroidism. When the free Thyroxine is below the lowest 10^{th} percentile and the TSH is above the reference range of 0.15-2.0mu/l then these is overt hypothyroidism. Subclinical hypothyroidism is when the thyroxine levels are within normal but the thyrotropin levels are elevated (1).

During early pregnancy the maternal thyroid physiology undergoes some well defined changes. These include an approximate doubling of the thyroglobulin binding protein concentration due to the increase to in estrogen levels as well as increase in the plasma concentration by 30-40%. These changes lead to an increase in the total thyroxine pool especially in the first trimester. These increases may be provided for largely by the stimulation of thyrotropin induced by the human chorionic gonadotropin hormone. In subjects with hypothyroidism there is a peak in thyrotropin at 8-10 weeks followed by a rapid down ward tread in free thyroxine. These suggest the important role of the human chorionic gonadotrophic hormone –thyroid axis in healthy women is to increase the thyroid hormone pool during the second trimester (1, 2).

In normal women there is a slight increase in free thyroxine levels and a reduction in thyrotropin occurs at 9-12 weeks of gestation. However in general the thyrotropin levels remains within the normal range throughout pregnancy despite the estimation of 30-40 percent increase in the thyroxine requirements (2, 3).

The sole source of thyroid hormones for the fetus from conception till 13 weeks is the pregnant woman. After the 13 week the fetal thyroid gland is developed enough to assume that role. These hormones play a critical role in the brain development. Trials on animals have shown that thyroid insufficiency in pregnant rats disrupted the migration of neurons in the fetal cortex and hippocampus leading to aberrant locations of neurons in adult offspring's brains. Children born to mothers with high thyrotropin levels at 17 weeks who were not treated for hypothyroidism had 7 points lower in IQ than matched controls. A prospective cohort study has shown that the low levels of free thyroxin at 12 weeks are associated with impaired psychomotor development at 10 months (3, 4).

Two thyroid hormones are available for treatment of hypothyroidism. These are Levothyroxine and Liiothyronine. Levothyroxine is the commoner one used. Liiothyronine can be used when quick onset of action is required, but it is less desirable for chronic use because it requires frequent dose adjustment and it also produces a transient elevation in triiodothyronine concentration. Levothyroxine requirement s increase early in pregnancy in most women with primary hypothyroidism,, reaching a plateau at 16-20 weeks of gestation (4, 5) . These are at a value approximately 47% higher than in non pregnant states, these high requirements persist through out pregnancy (5).

Given the importance of the mother being euthyroid for normal fetal cognitive development it is suggested that patients who are already known to be hypothyroid to increase the dosage by 30% as soon as they confirm that they are pregnant. Dose should be adjusted using the free serum thyroxin levels but not the TSH levels. Even partial treatment for hypothyroidism during pregnancy is thought to be beneficial to the off-spring. Infant neurodevelopment is not adversely affected by hypothyroidism during the first trimester (4, 5).

Hypothyroidism may lead to infertility and early pregnancy losses are not treated (2), Our patient had a pregnancy lose in the first trimester prior to these pregnancy. She was not evaluated during that pregnancy it is possible that this abortion was caused by the hypothyroid state.

Maternal complications associated with hypothyroidism are: Anemia, Post partum hemorrhage, Cardiac dysfunction, Pre-eclampsia toxemia, and abortions (2, 3, , 7).

Fetal complication s associated with hypothyroidism is; Intrapartum fetal distress, prematurity and low birth weight, congenital malformations, fetal death and perinatal death. Already alluded to is the impaired cognitive and psychosocial function of the off-spring (1, 2).

Routine screening of pregnant women for hypothyroidism is still a subject of debate in the developed countries (2). In our setting the screening is only done when there is a suggestive history or previous pregnancy losses.

REFERENCES.

1. *Alejando O.A and Offie P. Hypothyroidism during pregnancy. Canadian Family Physician.* (Mother Risk update). April 2004.
2. Allan MC, Haddow JE et al. maternal thyroid deficiency and pregnancy complications. ; Implication of a population based screening. J Med screen 2000 7:127-30
3. Lavado AR, Auso E, et al. Detection of thyroxine in cavities of human embryos in the first trimester. J Clin Endocrin Metab 1993 77; 1719-22
4. Haddow JE et al. Maternal thyroid hormone deficiency and subsequent neuro-psychological development of the child. N Engl J med 1999; 341: 549-55
5. ChopraIJ, Baber K. Treatment of primary hypothyroidism during pregnancy, is there an increase in thyroxin requirement during pregnancy. Metabolism 2003;52 :122-128
6. Erik K et al. Timming and magnitude in increase of levothyronine dosage during pregnancy in women with hypothyroidism. N Engl J med abstract 2004
7. Kriss Poppe and Daniel Glinoer. Thyroid auto immunity and hypothyroidism before and during pregnancy. Human reproductive updates. 2003 vol 9,No 2, pp149-151

Obstetric 10

CARDIAC DISEASE IN PREGNANCY- EMERGENCY CAESEREAN DELIVERY AND BTL- LIVE BABY

NAME: W.N IPNO : 1100690

AGE : 29 YEARS L.M.P:17/11/05

D.O.A: 20/06/06 E.D.D:24/08/06

D.O.D :14/08/06 GESTATION: 31 WEEKS

PARITY: 1+0

History of presenting complaints

She was admitted as a referral from Thika District Hospital with a diagnosis of cardiac disease in pregnancy in congestive heart failure. She presented with history of worsening dyspnoea and palpitations one week prior to admission. She also had orthopnoea and was coughing. Dyspnoea and palpitations began early in pregnancy and progressively worsened as pregnancy progressed. She was not known to have cardiac disease.

Obstetric history

She was Para 1+0 gravida 2.This delivery was by caesarean section in 1993 at Kisii District Hospital. She did not know the indication and discharge summary was not available. The boy was alive and well. Her LMP was on 17/11/05 and her EDD was on 24/8/06.She was at 31 weeks gestation by dates.

Antenatal care

She had not started antenatal care

Gynaecology history

Her menarche was at 16 years of age. She had regular menstrual periods lasting 4 days every 26 days. She was on Norplant from 1995 to 20005 when she removed it in order to conceive.

Past medical history

There was no history of prior admission. No history of any chronic medical illness or any surgery.

Family social history

She was a single lady working in a sisal company in Juja. She did not take alcohol or smoke cigarettes. There was no family history of chronic illness.

PHYSICAL EXAMINATION

General examination

She was sick looking. She had moderate pallor, not jaundiced and not cyanosed. She had pedal oedema but no sacral oedema. There was no finger clubbing or petechial haemorrhage. The vital signs were: temperature 37.1 0 c, pulse rate 114/min, respiratory rate 26/ min and blood pressure 102/65 mmhg.

Respiratory system

She was dyspnoeic. On auscultation there were coarse basal crepitations bilaterally.

Cardiovascular system

The pulse rate was 114b/minute.It was regular, of good volume and non-collapsing.

The jugular venous pressure was elevated. The precordium was hyper reactive. The apex beat was in 5th intercostal space lateral to the midclavicular line. The first and second heart sounds were heard. There was a diastolic murmur best heard at the mitral area.

Abdominal examination

The abdomen was uniformly distended. There was a sub umbilical midline scar. The fundal height corresponded to 30 weeks gestation. The foetus was in longitudinal lie, cephalic presentation. The foetal heart rate was 152 beats per minute and regular. There was tender hepatomegally 2 cm below sub costal margin.

Pelvic examination

This was not done because it was not indicated.

Diagnosis

A diagnosis of cardiac disease grade IV at 31 weeks in one previous scar was made.

Investigations

Haemogram:-Haemoglobin 7.8g/dl, WBC 5.3x109/l, neutrophils 76%, platelets256x109/l

Urinalysis: normal

Urea and creatinine: urea 5.6mmol/l, creatinine 60 umol/l

Blood group: O positive

VDRL : negative

HIV: negative

ECG: sinus tachycardia

Echocardiogram: There was a large left atrium with severe mitral stenosis and severe tricuspid regurgitation. The ejection fraction was 77%.there was pulmonary hypertension with MPA of 100mmHg.There were no vegetations seen.

MANAGEMENT

The patient was management for congestive cardiac disease in labour ward acute room. She was propped up and put on oxygen. She was put on frusemide 80 mg twice daily, digoxin 0.25 mg once daily, atenolol 12.5 mg once daily and haematinics. Grouping and cross matching was done. Her condition improved and she was transferred to the antenatal ward for continuation of care.

She was transfused 3 units of packed red cells. She was reviewed by a cardiologist, who recommended that she continue with the medication she was on. She was for weekly haemogram, blood urea and electrolytes and urine culture every fortnight. Respiratory and cardiac examination was done every day. Contraception was advised and agreed to bilateral tubal ligation. A decision was made to deliver by elective caesarean section at term .She remained functionally in grade III during the admission period. At 37 weeks gestation she went into spontaneous labour and the lie was found to be transverse. She was transferred to labour ward and she gave consent for emergency caesarean delivery and BTL. Consultant anaesthetist was asked to review the patient and recommended that the delivery be done in main theatre which was near ICU in case she would need ICU care. Caserean section was performed as described earlier. The outcome was a life female infant who scored 8/1, 9/10 and weighed 2250g.Delivery was uneventful. After the operation, general anaesthesia was reversed and the patient was transferred to HDU for observation.

Postoperative care

She was stable throughout the 24 hour observation in HDU after which she was transferred back to the acute room in labour ward. She was observed there for 24 hours the transferred to the postnatal ward. She stayed in the postnatal ward for 14 days. During this period she was put on intravenous Augmentin 1.2 grams 8 hourly for 10 days and continued on Digoxin 0.25mg daily, Lasix 40mg orally once daily and ranferon 10 mls twice daily. She was discharged through cardiac clinic for follow-up.

DISCUSSION

W.N was 29 years old para1+0, one previous scar admitted with cardiac disease in pregnancy at 31 weeks. She delivered at 37 weeks by emergency caesarean section due to transverse lie in one previous scar in labour with good outcome. Bilateral tubal ligation was done at caesarean delivery.

Cardiac disease is a rare but potentially serious medical disease that complicates approximately 1 percent of all pregnancies (1, 2, 3). At Kenyatta National Hospital the incidence of cardiac disease was reported to be 0.5% in 1969 (4). In a later study in 1982, Ngotho reported an incidence of 0.9% in KNH (5).

In Kenya majority of the patients were found to be in the age group of 20-24 years (5).

Cardiac disease is associated with increased maternal and fetal morbidity and mortality. In the USA, cardiac disease is the third leading cause of death in 24-44 year old women (1).

The aetiology of cardiac disease is variable. Cardiac disease may be congenital (operated or unoperated) or acquired. Acquired diseases include Rheumatic heart disease, hypertensive heart disease, coronary, thyroid, syphilitic, and kyphoscoliotic cardiac disease, idiopathic cardiomyopathy, cor pulmonale, constrictive pericarditis, heart block and isolated myocarditis (1, 2). In the developing countries rheumatic heart disease with valvular lesion is the most common cause of cardiac disease.

Ngotho in his study found that rheumatic heart disease was responsible for 84.6% of cardiac disease in pregnancy while 12.9% were congenital heart disease (5). Similar findings were reported by

Sequiera and Ojiambo in 1969 at Kenyatta National Hospital who found that 95% of cases being of rheumatic heart disease (RHD) origin (4). Congenital heart disease accounts for approximately 50 % of women with cardiac disease in pregnancy in the UK thanks to advance in paediatric cardiac surgery. Rheumatic heart disease is rarely seen in developed countries due to use of antibiotics in the treatment of streptococcal throat infections (1, 2, 3).

The dominant lesion in rheumatic heart disease is mitral stenosis accounting for 90% of rheumatic valvular problems (1, 2, 4). Significant problems may be anticipated if the valvular area falls below the normal 8cm sq to 4 cm sq. A valve with an area of 2 cm sq or less will require surgical valvotomy (2).

Irrespective of the underlying condition pregnancy imposes a significant burden on the heart due to normal physiological changes that occur. Both blood volume and cardiac output increase by 40-50 percent. Dynamic changes also occur during labour when each uterine contraction leads to auto transfusion of 300 - 500 ml of blood back into the systemic circulation. The sympathetic response to pain and anxiety during labour causes further elevations in heart rate and blood pressure. Cardiac output thus increases by as much as 34% during contractions and 12% between contractions. Following delivery, redistribution of blood volume and relief of venocaval compression leads to an increase in cardiac output of 60 - 80% followed by rapid decline to pre-labour values by 1 h after delivery. High-risk periods include the end of the second trimester, labour and the immediate postpartum period (1, 2). Because significant haemodynamic changes are apparently early in pregnancy women with severe cardiac dysfunction may experience worsening of heart failure before midpregnancy. In others heart failure develops after 28 weeks when pregnancy induced hypervolaemia is maximal (1).

Maternal mortality mostly occurs in conditions that restrict an increase in pulmonary blood flow typically pulmonary hypertension and mitral stenosis. Examples of this condition are the Eisenmengers syndrome with a mortality of 25-50% and Fallots tetralogy where the mortality is about 5 percent (1).

Fetal outcome in heart disease depends on severity. In general there is an increased incidence of growth restriction due to relative hypoxia and preterm delivery. Fetal outcome is especially poor in cases of cyanotic heart disease with fetal loss as high as 49 percent (1, 2).

The risk of fetal congenital heart disease is 8 per 1000 live born babies. The risk is increased to 5 percent if a parent is affected (1, 2).

Diagnosis of cardiac disease in pregnancy is challenging because some of the physiological changes mimic heart disease. This include breathlessness, increase in pulse, edema in lower extremities, functional systolic murmur. Symptoms of heart disease during pregnancy are: progressive dyspneic or orthopnea, nocturnal cough, haemoptysis, syncope and chest pain. Suggestive clinical findings include: cyanosis, clubbing of fingers, persistent neck vein distension, systolic murmur grade 3 and above, diastolic murmur, unequivocal cardiomegally, persistent arrhythmia, persistent split second sound and pulmonary hypertension (1).

Diagnostic investigations include electrocardiography, echocardiography and shielded chest x-ray. However the cardiovascular changes in pregnancy cause a challenge in interpretation. Due to the elevation of the diaphragm with advancing pregnancy, there is a 15 left axis deviation seen in the SCG with mild ST changes in the inferior lead. Some normal pregnancy induced changes in echocardiography include trivial tricuspid regurgitation, increased left atrial size and increase in left ventricular outflow cross sectional area. Echocardiography is the Gold standard as it allows accurate diagnosis of both structural and functional cardiac factors (1).

The degree of functional disability is graded according to the following New York Heart Association classification (1):

Grade I Uncompromised. Patients with cardiac disease but no limitation of physical activity

Grade II Slightly compromised. The patients have cardiac disease and slight limitation of physical activity. The patients are comfortable at rest but ordinary physical activity causes symptoms.

Grade III Markedly compromised. Patients have cardiac disease with marked limitation of activity. The patients are comfortable at rest but symptoms occur with less than ordinary physical activity.

Grade IV Severely compromised. Patients have cardiac disease with symptoms even at rest.

The grading is clinical and depends on cardiac response to physical activity with no relationship to the extent of the heart lesion. Patients with pure mitral stenosis and those who have had previous cardiac failure or cardiac surgery are classified as Grade 1V.Our patient was classified as grade IV.

The American college of Obstetricians and Gynaecologists has further classified the various heart diseases into 3 groups according to the risks of mortality during pregnancy (1). These are:

Group 1: Low Risk-Mortality<1%

These include; atrial septal defects, ventricular septal defects, patent ductus arteriosus, corrected tetralogy of Fallot, bioprosthetic valve, pulmonary or tricuspid disease and mild mitral stenosis (NYHA disease 1 and 11)

Group 2: Moderate Risk-Mortality 5-15%

This is further divided into 2A and 2B.

2A- These include; symptomatic mitral stenosis(NYHA classes III and 1V),aortic stenosis,aortic coarctation without valvular involvement, uncorrected tetralogy of Fallot, previous myocardial infarction, Marfans syndrome with normal aorta.

2B-These include; mitral stenosis with arterial fibrillation and artificial valve.

Group 3: High risk-Mortality 25-50%

These include pulmonary hypertension, aortic coarctation with valvular involvement and Marfans syndrome with aortic regurgitation.

Based on ACOG classification our patient was in the high risk group.

The management calls for team approach involving obstetrician, cardiologist, paediatrician and anesthesiologists (1, 2, 3). Where preexisting disease is known preconception care should be provided. The woman should undergo a cardiology and obstetric evaluation and genetic counseling. Assessment of the disease and the likely maternal and foetal risks including risk of worsening and risk of transmission to the child should be discussed with her (1, 2, 3).

Patients with high risks should be advised to terminate pregnancy in 1st trimester if possible. Pregnancy termination may be justified in conditions like: pulmonary hypertension, dilated cardiomyopathy, Eisenmenger syndrome, marfan syndrome, any uncorrectable lesion in functional

classes III or IV refractory to medical management(6). The objective of the antenatal care is to avoid aggravation of her cardiac condition.

Factors which may aggravate her disease are anemia, infections especially urinary and respiratory infections, hypertension, anxiety, arrthymia, thromboembolic disease and rigorous activity. She should be advised to; have adequate bed rest, low salt diet(2gm/day), avoid contact with any persons with respiratory infection and if possible be vaccinated against pneumococcal and influenza infections Bed rest reduces cardiac output hence reducing the strain on cardiovascular system. She should be on haematinics supplementation and on surveillance for infections. Cigarrete smoking is prohibited because of its cardiac effects as well as its propensity to cause upper respiratory tract infections. Our patient did not smoke cigarettes (1, 2).

Anticoagulant drugs may be needed in pregnancy because of recurrent deep vein thrombosis, pulmonary embolism, rheumatic heart disease with atrial fibrillation, prosthetic valves or cyanotic congenital heart disease (7). Grade I and 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. They are seen every 2 weeks and at 36 weeks they are admitted to await delivery. Grades III and IV patients are usually confined in the wards until after delivery. Alternate day weighing, weekly hemoglobin levels and fortnightly cultures are recommended for those admitted. The haemoglobin should be maintained at or above 12g/dl(1,3). Infections and anaemia must be treated rigorously if detected. The development of heart failure in pregnancy is a very ominous sign and must be prevented from happening. Digoxin and diuretics are indicated in heart failure (2).

Patients with cardiac disease should be delivered vaginally at term unless there are obstetrical indications for caesarian section (1, 3). They should await spontaneous onset of labour. Vaginal delivery is advocated because it is associated with less morbidity and mortality. Caesarian section carries risk of hemorrhage and infection which is less well tolerated incases of cardiac disease (2). During labour, the patient should be propped up; vital signs should be taken after every 30 minutes during first stage of labour and every 15 minutes in the second stage of labour. Pulse rate of 100 beats /minute or more or the respiratory rate above 24/minute when associated with dyspneic may

suggest impending ventricular failure and need for intensive medical management. Urine output should be recorded hourly as a decrease may be an early sign of cardiac failure (1, 3).

Morphine should be used to allay anxiety and pain. Epidural analgesia may be used but under a senior anaesthetist due to risk of maternal hypotension. The patient is put on oxygen and prophylactic antibiotics. The American heart association recommends intravenous ampicillin 2.0g and gentamycin 1.5kg, 30-60 minutes before delivery and repeated 8 hours later. The practice in our unit is to give the patients a full course of antibiotics because of the high risk of infection in our set up. Artificial rupture of membranes is done when she is in established labour. vaginal examinations are minimized (1). These two measures are undertaken to reduce risk of infective endorcarditis. Intravenous fluids should be carefully monitored to avoid fluid overload and pulmonary edema.

The ACOG recommends use of a central line to guide infusion of fluids (1, 3). In our setup intravenous fluids are restricted. Incase uterine contractility is not adequate syntocinon should be given using oxytocin pump. In our set up a high syntocinon dose of 10 IU in 500 mls of fluid is given, followed by IV frusemide.

Second stage of labour should be shortened by elective assisted delivery to minimize increase in blood pressure with bearing down. Episiotomy is performed once the head is on the perineum (1).

The most dangerous time for the development of congestive cardiac failure or pulmonary edema is immediately after delivery.[1] Active management of third stage is recommended. Postpartum hemorrhage is prevented by uterine massage and oxytocin. Ergometrine is avoided because it is associated with intense vasoconstriction, hypertension and heart failure. A bolus of intravenous frusemide immediately after delivery may offset the haemodynamic changes of sudden mobilization and redistribution of fluids. Our patient received arsenide bolus in theatre. She did not develop congestive cardiac failure

After delivery, the patients are observed for 24 hours in labour ward acute room and if there are no complications they are transferred to the antenatal wards and observed for 10-14 days. This is because the immediate pueperium is the period of maximum risk of decompensation. Prophylactic antibiotics, analgesics and haematinics are continued postpartum. The patient is monitored for

congestive heart failure, infective endocarditis and thromboembolic disease. Our patient was put on antibiotics, analgesics and haematinics. She was observed for 10 days and none of the above complications arose.

Before discharge contraception should be discussed with the patient to limit family size and for optimal spacing of children. They should be advised the need to complete their family size early as cardiac disease worsens with age (age induced deterioration of cardiac function). Barrier methods and progesterone only pill is advisable for those who desire another child or do not opt for tubal ligation. When the family size is complete tubal ligation is the optimal choice. Alternatively vasectomy can be offered to the spouse if desired. Combined pills predispose to thromboembolism while intrauterine devices predispose to infection (1, 2, 7).

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REFERENCES

1. Cunningham FG, Gant NF, Leveno KJ, Gilstrap LC, Hauth JC, Wenstrom KD. (Eds). Cardiovascular Diseases. In: Williams Obstetrics, 21st Edition, McGraw Hill, 2005:1019 – 1028.
2. Stuart C. and Christoph Lees. Medical diseases complicating pregnancy. Heart disease Obstetrics by Ten Teachers.17th Edition. Bookpower 2000;pg 241-245
3. Grewal M, Biswas MK, Perloff D. Cardiac, Haematologic, Pulmonary, Renal and Urinary Tract; Disorders in Pregnancy. In: DeCherney AH, Nathan L. Current Obstetric and Gynaecologic Diagnosis and Treatment 9th Edition McGraw-Hill Companies 2003; 387-409.
4. Sequiera R. F.; Ojiambo H.P. The heart in pregnancy. EAMJ 46 (1); 18, 1969.
5. Ngotho D. K. Cardiac disease at Kenyatta National Hospital. Mmed Thesis University of Nairobi, 1982.
6. Brokner ME, Hillis LD, Lange RA.Congenital Heart Disease in Adults. Engl J Med 2000; 342:334.
7. Carabello BA,Crawford FA.Valvular Heart Disease.NEngl J Med 1997;337:32

Obstetrics 11

TITLE: HIV IN PREGNANCY: LIVE BIRTH

NAME: M.W.C
AGE: 31 YEARS
IPNO: 1110314
PARITY: 0+1
GBD: 38 weeks

DOA: 21/09//06
DOD: 26/10/06
LMP: 31/12/05
EDD: 08/10/06

Presenting complaint

Admitted for elective caesarean section for PMTCT at 38 weeks

History of presenting illness

The patient was admitted via the antenatal clinic for elective caesarean section at 38 weeks for prevention of mother to child transmission of HIV. She was diagnosed to be HIV positive in 2004

Antenatal period.

She had been followed up by a private Obstetrician from 6 weeks gestation. She was referred to KNH at 33 weeks for antiretroviral therapy.

The antenatal profile was; haemoglobin 10.4g/dL, VDRL was negative, Blood group O positive and Elisa for HIV was positive. The couple was discordant

Her CD4 count on 13/05/06 at 19 weeks gestation was 283 cells/mm and the viral load was 5730 copies/ml. She was not on ARVs .Haematinic supplementation was done throughout pregnancy and she was on balanced diet. Obstetric ultrasound done at 10 weeks showed a single intrauterine pregnancy. Anomaly scan at 19 weeks was normal.

In the clinic she was advised on prevention of mother to child transmission. She was also counseled on the need for antiretroviral therapy for herself. Liver function tests, full haemogram and urea, creatinine and electrolytes were normal. She was started on combivir 1 tablet twice daily (zidovudine 300mg and lamivudine150 mg) and kaletra (lopinavir and retrovir) three tablets twice a day. She was counseled on adherence. Mode of delivery was discussed and she agreed on elective

caesarean section. Infant feeding options was advised and she opted for exclusive breast feeding. She was followed up till 38 weeks when she was admitted for elective caesarean delivery.

Obstetric history and gyaecology history

She was Para 0+1, gravida 2. She had spontenous complete arbortion 2 years prior to this pregnancy.

Se attained menarche at 13 years and had regular cycles lasting 5 days every 21 days. She used pills on and off for contraception. Pap smear done 2005 was normal.

Past medical history

Not significant

Family and social history

She was married. She was not working at present. She did not smoke or drink alcohol.

PHYSICAL EXAMINATION

General examination

She was in good general condition .She was not pale, not jaundiced, no oral thrush, no lymphadenopathy and no edema. Vital signs were normal.

Respiratory/cardiovascular and CNS

All were normal.

Abdomen

It was uniformly distended with a fundal height corresponding to term pregnancy. The fetus was in longitudinal lie and cephalic presentation. Head was high. Fetal heart was 138 beats/min and regular

Vaginal examination

Was not indicated

Diagnosis

HIV positive mother at 38 weeks gestation by dates.

MANAGEMENT

She was admitted to the labour ward for elective caesarean section. She was scheduled for an elective caesarean section the following day. She gave consent for caesarean, 2 units of blood were crossmatched. Haemoglobin was 12.2 g/dl and urea, creatinine and electrolytes were normal. Half an hour before surgery she was premeditated with atropine 0.6mg intramuscularly.

In theatre, vulvovaginal toilet and aseptic catheterizations were done. The abdomen was cleaned and draped with sterile towels. Under general anaesthesia, the abdomen was open through a Pfannestiel incision. A lower uterine segment caesarean section was performed The outcome was a live male infant with an Apgar score of 9 in 1 minute and 10 in 5 minutes and weighed 2150g. The placenta was delivered and was complete and grossly normal. The uterus was closed and Haemostasis achieved. The abdomen was closed in layers after ascertaining swabs and instruments count. Vulvovaginal toilet was done and the catheter removed. She had an estimated blood loss of 400ml.

Post operative management

The vital sign were observed half hourly until she was fully conscious, one hourly for 4 hours and then 4-hourly thereafter. She was put on intravenous fluids, 1 litre of dextrose alternating with normal saline 8-hourly for 24 hours. Analgesia was through intramuscular pethidine 100 mg 8-hourly for the first 24 hours then oral ibuprofen 400 mg 8-hourly. She was also put on intravenous augmentin for 48 hours and was then changed to orals. She continued with her antiretroviral drugs.

The baby was given syrup niverapine on the day of delivery and AZT syrup twice a day for 4 weeks.The mother had opted to practice exclusive breat feeding, for which she was counseled and coached on how to do it safely.The patient did well postoperatively. They were discharged home through the high risk postnatal clinic in 2 week's time.

Post-natal follow-up

When she was seen in the post-natal clinic after 2 weeks, she had no complaints and the baby was well and exclusively breast feeding .The wound was well healed with good uterine involution. She

was then given a 4 weeks return date. During the following review, she was doing well and was advised on contraception in which she opted for the progesterone only pill. She was referred to her obstetrician for family planning and return visit to the high risk clinic at 3 months

DISCUSSION

Acquired immunodeficiency syndrome (AIDS) was first described in the United States in 1981 among homosexual men with defective cellular immunity (1). In Kenya the first case was reported in 1984 (2).

HIV is a single stranded RNA retrovirus. A viral aetiological agent in AIDS was first demonstrated in 1985 and the virus was initially referred to as HTLV-3 (3). HIV virus replicates by using reverse transcriptase enzyme to translate its genomic RNA into DNA copy. The viral DNA is then inserted as a provirus into the host cell DNA.The CD4 site serves as a receptor for the virus. The lifespan of an infected cell is shortened; hence the number of T-cells declines insidiously and progressively and eventually results in profound immunosuppression (1).

HIV/AIDS is a major public health problem in Kenya with the national prevalence in 2003 being 7% and the prevalence among women been 9 % almost twice that of men (4).

The modes of HIV infection are contact with body fluids and mother to child. In Kenya heterosexual contacts account for 90% of new infection while about 10% are spread from mother to child (5). Transmission from male to women is thought to occur more readily than female to male because of high viral concentration in semen than in vaginal secretion, larger vaginal surface area and that coitus causes more introital mucosal breaks than in the penile skin. Other factors that increase the risk in heterosexual exposure include high risk sexual partner s and presence of STDS (6). The patient presented had most likely acquired HIV by sexual contact since she had not been transfused blood at any time.

Mother to Child transmission (MTCT) accounts for 90% of pediatric HIV infections in Kenya. Approximately 60,000 HIV positive births take place annually in Kenya (5).

The risk of transmission varies during pregnancy, delivery and postnatally.The overall risk of transmission without interventions is 15-40%.During pregnancy, transmission across placenta accounts for 10-20%, labour and delivery account for 35-50% while breastfeeding carries a

transmission rate of 30-40%.The risk is higher during labour and delivery because the risk of fetal maternal hemorrhage is accentuated (7).

The risk factors for MTCT of HIV are: high viral load, viral genotype and phenotype, low CD4 count, recent HIV infection,vaginal delivery, rupture of membranes for more than 4 hours,prematurity,duration of breastfeeding, mixed feeding, breast disease, vitamin A deficiency,STIs,anaemia,chorioamnitis,smoking,unprotected sexual intercourse, invasive procedures, first twin ,oral thrush (5,8).

Tests used in the diagnosis of HIV are divided into antibody detection, antigen detection, viral nucleic acid and viral culture. The main test is ELISA antibody detection with sensitivity of up to 99 % (1, 6). A positive ELISA test can be confirmed by the western blot. Locally most PMTCT programmes use simple rapid test. The Ministry of Health, Kenya recommends 2 different types of rapid test on all clients. Incase of discordance a third test "tie breaker "or ELISA is done. The advantage of the rapid test is that it's easy to perform and give results in less than 10 minutes (5). The patient presented was found to be HIV infected using rapid test done as part of the antenatal profile.

All pregnant women should be counseled and encouraged to tested for HIV infection to allow them know their HIV infection status for their own health and to reduce the risk of prenatal HIV infection (5). An opt in approach is used in KNH where women are notified that a HIV test is part of a comprehensive set of antenatal profiles and may decline testing if they don't want.Acceptabilty rates locally are high. A study conducted by Kiarie in 1994 reported that the rates of acceptance for HIV testing among ANC clients in Umoja,Jericho and Dandora city council clinics was 99.4%(9). Statistics from KNH PMTCT programmes show acceptability rates of 99% (10).

Theoretically, pregnancy being an immunosuppressive state is expected to worsen the progression of HIV. In developed countries pregnancy does not appear to change the course of HIV infection in women. According to the US Public Health service guidelines (2003) maternal morbidity and mortality are not increased by pregnancy (1). However local studies have shown that maternal morbidity and mortality is increased.

HIV is associated with adverse fetal outcomes such as abortion, IUGR, prematurity, increased perinatal morbidity and mortality (1, 6, 8). Antenatal management of HIV infected mothers should include nutritional supplementation, lifestyle and behavior change, treatment and prophylaxis of opportunistic infections,haematinics,screening for STIs,malaria chemoprophylaxis and provision of antiretrovirals.Counselling should be done on couple testing, safe sex and positive living (5). Good nutritional status reduces the risk of MTCT and progression of HIV infection (7). Nutrition counseling was given to our patient.Haematinic supplementation was done and partner was tested and found to be negative. They were counseled on safe sex.

Specific interventions to reduce MTCT are use of antiretroviral drugs, elective cesarean delivery and not breastfeeding. The benefits of the use of antiretroviral drugs are well documented in the literature. The ARV regimes are described below (1).
Zidovudine (AZT) long course (PACTG 076).300mg P.O B.D starting from 14 weeks, 2mg/kg at the start of labour, then1mg/kg/hr till delivery. For the baby, syrup 2mg/kg QDS for 6 weeks.(68% effective)

- Zidovudine short course.300mg P.O BD from 36 weeks,300mg P.O 3 hourly during labour(50% effective)
- Nevirapine 200mg P.O during labour, 2mg /kg syrup for the baby within 72 hours after delivery (HIVNET 012) (47% effective with breast feeding).

In our unit AZT 300mg P.O BD is given from 28 weeks, then 600mg and nevirapine 200mg start at labour onset and single dose nevirapine 2mg/kg for the baby within 72 hours. Those women who are more than 36 weeks gestation or those in early labour nevirapine 200mg is given at onset of labour and baby is given single nevirapine dose as above.
HAART is indicated for those mothers with CD4 counts of less than 350 or in WHO HIV stage IV starting from 16 weeks gestation (PMTCT).HAART regime in our unit is AZT 300mg BD, lamivudine (3TC) 150 mg BD and nevirapine 200mg OD for 2 weeks and increased to BD dosing if liver function tests are normal (10). Those with CD4 counts more than 200 nevirapine is avoided due to risk of severe hepatotoxicity (5).

The WHO HIV Staging is shown below (11).

WHO STAGING SYSTEM FOR HIV INFECTION AND DISEASE IN ADULTS AND ADOLESCENTS

Clinical Stage I 1. Asymptomatic 2. Generalized lymphadenopathy Performance scale 1: asymptomatic, normal activity
Clinical Stage II 3. Weight loss <10% of body weight 4. Minor mucocutaneous manifestations (seborrhoeic dermatitis, prurigo, fungal nail infections, recurrent oral ulcerations, angular cheilitis) 5. Herpes zoster within the last five years 6. Recurrent upper respiratory tract infections (i.e. bacterial sinusitis) And/or performance scale 2: symptomatic, normal activity
Clinical Stage III 7. Weight loss >10% of body weight 8. Unexplained chronic diarrhoea, >1 month 9. Unexplained prolonged fever (intermittent or constant), >1 month 10. Oral candidiasis (thrush) 11. Oral hairy leucoplakia 12. Pulmonary tuberculosis 13. Severe bacterial infections (i.e. pneumonia, pyomyositis) And/or performance scale 3: bedridden <50% of the day during last month
Clinical Stage IV: 14. HIV wasting syndrome 15. Pneumocystic carinii pneumonia 16. Toxoplasmosis of the brain 17. Cryptosporidiosis with diarrhoea >1 month 18. Cryptococcosis, extrapulmonary 19. Cytomegalovirus disease of an organ other than liver, spleen or lymph node (e.g. retinitis) 20. Herpes simplex virus infection, mucocutaneous (>1month) or visceral 21. Progressive multifocal leucoencephalopathy 22. Any disseminated endemic mycosis 23. Candidiasis of oesophagus, trachea, bronchi 24. Atypical mycobacteriosis, disseminated or pulmonary 25. Non-typhoid Salmonella septicaemia 26. Extrapulmonary tuberculosis 27. Lymphoma 28. Kaposi's sarcoma 29. HIV encephalopathy And/or performance scale 4: bedridden >50% of the day during last month

Elective caesarean delivery when viral loads exceed 1000copies/ml has been shown to reduce MTCT by one half (1, 5). This intervention is practiced in our set up. Unfortunately viral loads measurements are not done readily available due to cost hence all patients who choose to have elective caesarean section are offered without viral load assessment. Our patients viral load was done at 19 weeks and HAART initiated at 34 weeks. It's unlikely that in 4 weeks we achieved complete suppression of the virions.

For HIV positive mothers who opt vaginal delivery, intrapartum interventions include; use of partogram to avoid prolonged labour, vaginal cleansing with chlorhexidine 0.25% after every vaginal examination, artificial rupture of membranes at cervical dilatation of 7cm or more unless the progress of labour is abnormal where early rupture is indicated. Routine episiotomy is not recommended. Suction of the baby is not recommended and the cord should be clamped immediately and should not be milked (5).

Breast milk increases the risk of neonatal transmission of HIV and in general is not recommended in HIV positive women (1). Counseling on infant feeding options according to W.H.O. and local guidelines should be done to all HIV positive women. Those opting to breastfeed should be encouraged to exclusively breastfeed for six months with rapid weaning (1, 5). The patient presented opted to exclusively formula feed.

Postpartum care includes care of breast and lochia, review after one week to assess and reinforce adherence to infant feeding option, revisit at 6 weeks postpartum for Pap smear and initiation of family planning. HIV infected women should initiate a reliable contraceptive method by 2-4 weeks postpartum. They can use all modern methods of contraception. Dual method of contraception is recommended to reduce chances of infection with other STIs and HIV (5). Further visits are scheduled at 3 and 6 months to assess adherence to infant feeding, CD4 and HIV clinical monitoring. On the 6 month visit the clients are send to Comprehensive care Centre for continued care and support (10).

REFERENCES

1. Cunningham FG, Gant NF, Leveno KJ, Gilstrap LC Hauth JC, Wenstrom KD. Acquired Immunodeficiency Syndrome. In: Williams Obstetrics, 21st Edition, McGraw Hill companies, 2001: 1498 - 1504.
2. Republic of Kenya, MOH, Sessional paper No.4 of 1997 on AIDS in Kenya 1997.
3. Ratner L.et al.HTLV 111 and LAV are variants of the same AIDS virus nature 313:646 1985.
4. Central Bureau of Statistics (CBS) [Kenya], Ministry of Health (MOH) [Kenya] and ORC Macro. 2004. Kenya Demographic and Health Survey 2003. Calverton, Maryland: CBS, MOH, and ORC Macro.
5. Republic of Kenya, Ministry of Health.2005: Clinical guidelines for the management of HIV infected pregnant women and prevention of mother to child transmission (MTCT) of HIV in Kenya
6. DeCherney A.H., Nathan L. (Eds): HIV in pregnancy.Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th edition, McGraw-Hill Companies, 2003:745-748.
7. K.Koigi et al.Guidelines on antenatal HIV testing and management. Division of Obstetrics and Gynaecology, Nairobi Hospital.2004.
8. U.S Department of Health .A guide to the clinical care of women with HIV.2001 Edition.
9. Kiarie J.N.The acceptability of prenatal HIV screening.M.Med thesis UoN.1996.
10. Protocol for PMCT in KNH and Pumwani Maternity Hospital
11. WHO. Treat 3 Million by 2005. Scaling Up Antiretroviral Therapy In Resource-Limited Settings: Treatment Guidelines for a Public Health Approach, 2003 Revision. World Health Organization, Geneva, 2004

REFERENCES

1. Cunningham FG, Gant NF, Leveno KJ, Gilstrap LC Hauth JC, Wenstrom KD. Acquired Immunodeficiency Syndrome. In: Williams Obstetrics, 21st Edition, McGraw Hill companies, 2001: 1498 - 1504.
2. Republic of Kenya, MOH, Sessional paper No.4 of 1997 on AIDS in Kenya 1997.
3. Ratner L.et al.HTLV 111 and LAV are variants of the same AIDS virus nature 313:646 1985.
4. Central Bureau of Statistics (CBS) [Kenya], Ministry of Health (MOH) [Kenya] and ORC Macro. 2004. Kenya Demographic and Health Survey 2003. Calverton, Maryland: CBS, MOH, and ORC Macro.
5. Republic of Kenya, Ministry of Health.2005: Clinical guidelines for the management of HIV infected pregnant women and prevention of mother to child transmission (MTCT) of HIV in Kenya
6. DeCherney A.H., Nathan L. (Eds): HIV in pregnancy.Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th edition, McGraw-Hill Companies, 2003:745-748.
7. K.Koigi et al.Guidelines on antenatal HIV testing and management. Division of Obstetrics and Gynaecology, Nairobi Hospital.2004.
8. U.S Department of Health .A guide to the clinical care of women with HIV.2001 Edition.
9. Kiarie J.N.The acceptability of prenatal HIV screening.M.Med thesis UoN.1996.
10. Protocol for PMCT in KNH and Pumwani Maternity Hospital
11. WHO. Treat 3 Million by 2005. Scaling Up Antiretroviral Therapy In Resource-Limited Settings: Treatment Guidelines for a Public Health Approach, 2003 Revision. World Health Organization, Geneva, 2004