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IABETES IN PREGNANCY-INDUCTION OF LABOUR AT 39 WEKS; LIVE BIRTH

NAME: L.C	IPNO: 1089570
AGE: 38 YEARS	LMP 2/10/05
D.O.A:20.06.06	EDD 9/07/06
D.O.D:10.07.06	GBD 37 WEEKS

Presenting complaint

The above named patient was admitted as a referral from Pumwani maternity hospital with high blood sugar. She had presented there with symptoms of polydipsia and polyuria for 3 months. The random blood sugar done at Pumwani was 19 mmol/l. She had been started on mixtard 20 I.U and 8 I.U every morning and evening respectively.

Fetal movements were reported to be normal. She had normal vision .She had no other complains.

Obstetric history

She was Para 5+1 gravida 7 with her last delivery in 2000.All her deliveries were vaginal deliveries which were uneventful and babies weights ranged 2.6kg- 3.8 kg, all alive and well. She had a miscarriage at 4 months gestation in 2004.

Her LMP was on 2/10/05 and her EDD was on 09/7/06.She was at 37 weeks gestation by dates.

Antenatal care

She was attending her antenatal care at Pumwani maternity hospital. Her blood group is O positive, haemoglobin 9.2g/dl, VDRL negative, HIV negative and urinalysis was normal .she had not started antenatal care

Gynaecology history

Her menarche was at 14 years of age. She had regular menstrual periods lasting 4 days every 28 days. She used Depo-Provera twice in 1993 then traditional methods. She stopped Depo-Provera due to bleeding.

Past medical history

There was no history of prior admission. She was not a known diabetic. No history of any chronic medical illness or any surgery.

Family social history

She was a housewife. She did not take alcohol or smoke cigarettes. There was no family history of diabetes or any other chronic illness.

PHYSICAL EXAMINATION

General examination

She was in good general condition. She was not pale, not jaundiced, not dehydrated and not cyanosed. She had pedal oedema but no sacral oedema. The vital signs were: temperature 36.8 0 c, pulse rate 84/min, respiratory rate 18/ min and blood pressure 112/65 mmHg.

Respiratory system, cardiovascular and central nervous system

These three systems were examined and found to be normal.

Abdominal examination

The abdomen was uniformly distended. There was a sub umbilical midline scar. The fundal height corresponded to term gestation. The foetus was in longitudinal lie, cephalic presentation. The foetal heart rate was 144 b/minute and regular. There was no organomegally.

Pelvic examination

This was not done because it was not indicated.

Diagnosis

A diagnosis of diabetes mellitus at 37 weeks gestation was made

MANAGEMENT

She was admitted for blood sugar control. Daily serial sugars were done as she continued with mixtard insulin 20 I.U and 8 I.U every morning and evening. Serial blood sugars were done and the dose of Insulin titrated accordingly.

Date	FBS	11am	3pm	9pm
First week				
20/06/06	-	-	-	11.6
21	5.4	-	16	-
22	3.4	-	10	8.6
23	7.9	-	12.2	13.2
24	5.8	4.8	12.2	13.3
25	7.1	4.6	10.8	9.4
26	6.1	8.1	-	-
27	11.1	-	7.7	-
Second week				
28/6/06	5.4	11.1	-	13.8
29	4.5	9.4	7.5	11.5
30	6.6	6.3	12.6	9.8
1/7/06	4.0	12	11.7	12.2
2	4.3	4.6	10.2	9.0
3	3.3	8.2	12.6	-
4	4.6	3.5	15.1	8.8
5	3.5	7.6	7.0	7.7
6	4.6	-	6.5	9.5
7	4.1	4.4	6.4	7.0
8	4.2	6.3	6.0	6.4
9	4.5	-	7.2	7.7

Blood sugar remained well controlled. She received nutritional counselling and was put on diabetic diet. Fetal kick chart was commenced to monitor foetal well being and was reassuring. Mid stream specimen of urine for microscopy, culture and sensitivity was done and found to be normal. Bilateral tubal ligation was advised but declined. She was not able to choose any method while in the ward.

She was planned for induction of labour at 39 weeks. Pre induction cervical assessment was done and bishop score was found to be unfavourable.PGE2 3 mg pessary was inserted in the posterior fornix with improvement in bishop score. ARM was done 8 hours later and syntocinon 2.5 I.U in 500mls normal saline commenced.

Progress of labour was good. She had SVD to a life male infant whose Apgar score was 9/1 and 10/5 with a birth weight of 3900g.The neonates random blood sugar was 2.8 mmol.

Her insulin doses were reduced by half to 10 I.U in the morning and 5 I.U in the evening. Post delivery blood sugars were normal. She was discharged home to be followed up with serial blood sugars in the postnatal clinic and latter in the medical clinic.

DISCUSSION

The case presented is of a 38 year old Para 5+1 with gestational diabetes diagnosed at 37 weeks gestation. She was put on mixtard and had good blood sugar control. She was induced at 39 weeks and had uneventful spontaneous vertex delivery. Both mother and baby did well postnatally.

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia resulting from either relative deficiency of pancreatic insulin production, limited insulin release in response to a carbohydrate challenge, or impaired effect of insulin at the cellular level(1).

Gestational diabetes is defined as carbohydrate intolerance of various severities with onset or first recognition during pregnancy (1). The prevalence of overt diabetes mellitus in women of child bearing age in the general population is about 1%.In recent past; the incidence of diabetes complicating pregnancy has increased to about 40% in USA. The incidence was 3.3 % in 2002 of which 90% was attributable to gestational diabetes. The prevalence of type 2 diabetes is rising and many women found to have gestational diabetes are likely to have type 2 diabetes that has previously gone undiagnosed (1). In Kenyatta National Hospital, the incidence of diabetes in pregnancy was found to be 0.15 %(2).

The ACOG classification of diabetes mellitus is as follows: (1)

- Type 1-when there is absolute insulin deficiency
- Type 2-either there is defective insulin secretion or insulin resistance

In pregnancy diabetes is classified as(1):

- Pregestational (overt)-diagnosed before pregnancy
- Gestational-diagnosed during pregnancy

This last group of patients requires further evaluation after pregnancy because the group may include previously undiagnosed type 2 patients identified at the beginning of pregnancy, and normal pregnant women detected during the third trimester after an oral glucose tolerance test (OGTT).Based on this classification, our patient had type II diabetes. Another classification was proposed by Priscilla white almost 40 years ago. This was modified by the American College of Obstetricians and Gynaecologist in 1986 and further modified in 1994.It relates to the onset of diabetes, its duration and the degree of vasculopathy to the outcome of pregnancy(3). At the most favorable end of the scale was gestational diabetes and at the other end were patients with chronic renal failure, most of the type 1 patients with extremely poor pregnancy prognosis. The modified white's classification is as follows:-

Class	Features
Class A1	Diet controlled gestational diabetic
Class A2	Gestational diabetis requiring insulin
Class B	Diabetic onset of 20 years or above, duration at 10-19years, no vascular lesions.
Class C	Diabetic onset at age 10-19, duration of 10-19years, no vascular lesions.
Class D	Diabetic onset of age less than 10 years,duration for more than 20 years or calcification of the vessels of the leg.
Class E	Same as D plus calfication of pelvis vessels
Class F	Any age of onset, any duration with nephropathy
Class H	Any age of onset, any duration with coronary heart disease
Class R	Any age of onset, any duration with proliferative retinopathy
Class T	Any age of onset , any duration with renal transplant

The patient presented had pregestational diabetes with an onset above 20 yrs, duration less than 10 yrs and no vascular lesions. Based on the Priscilla White classification, she was class A2.

An important metabolic change in pregnancy is a decrease in insulin sensitivity which parallels the growth of the fetoplacental unit and facilitates the diversion of glucose to the fetus. In normal pregnancy, insulin secretion increases two to threefold by hypertrophy/hyperplasia of the mother's pancreatic b-cells in order to counteract this decrease in insulin sensitivity. Women who lack this b-cell reserve e.g. women with type 2 diabetes need increasing doses of exogenous insulin, and it is not unusual for requirements to increase threefold by the end of the third trimester. Anticipating this increased insulin requirement is necessary to avoid maternal hyperglycemia and ketoacidosis (4).

In the fasting state, maternal glucose levels are lower in pregnancy than in the non-pregnant state whereas the concentration of free fatty acids, triglycerides and plasma ketones increase. Therefore, a state of relative starvation exists in which glucose is spared for foetal consumption while alternative fuels are used by the mother. Decrease in insulin sensitivity is in part as a result of anti-insulin hormonal activity (human placental lactogen, estrogen, progesterone, cortisol and prolactin). Degradation of insulin is also increased in pregnancy. Inadequate maternal pancreatic insulin response leads to maternal and then foetal hyperglycemia. This typically manifests as recurrent postprandial hyperglycemic episodes. Surging maternal and foetal glucose levels are accompanied by episodic foetal hyperinsulinaemia. Fetal hyperinsulinaemia promotes excess nutrient storage resulting in macrosomia (5).

There is lack of consensus regarding the optimal approach to screening for gestational diabetes. Women with risk factors should have a glucose tolerance test as soon as feasible. If results don't demonstrate diabetes, they should be tested between 24 and 28 weeks gestation(6). The international workshop on gestational diabetes in 1997 recommended that screening for gestational diabetes should be performed between 24 and 28 weeks gestation. This evaluation may be done in one or two steps.

In the two-step procedure, a 50g oral glucose challenge test is followed by a diagnostic100g oral glucose tolerance test (OGTT) if results exceed a predetermined plasma glucose concentration (141-190 mg/dl). In the 1-step approach, the diagnostic 75g test is administered without the preceding 50g test.

When the two-step procedure is used, plasma glucose is measured 1 hour after a 50g oral glucose load without regard to the time of day or time of last meal.

The ACOG 1994 criteria for diagnosis of gestational diabetes using the OGTT are as follows;

	National Diabetic Data Group (1979)	Carpenter and Coustan (1982)
Fasting	105 mg/dl	95 mg/dl
1 – Hour	190 mg/dl	180 mg/dl
2-hour	165 mg/dl	155 mg/dl
3-hour	145 mg/dl	140 mg/dl

Gestational diabetes is diagnosed when two or more values are met or exceeded (1). Our patient had a random blood sugar of 19 mmol which is diagnostic of overt diabetes hence there was no need for OGTT.

Diabetes may be deleterious to pregnancy in a number of ways. Adverse maternal effects include (1,6): -

- Increased rate of abortions
- Four-fold increased risk of preeclampsia and eclampsia.
- Increased risk of infections such as candidal vulvovaginits, UTI, respiratory tract infection, and puerperal sepsis.
- Increased risks of preterm delivery.
- Increased risk s of injury to the birth canal and higher rate of caesarean section (up to 42-72%)
- High incidence of polyhydramnios which at time causes cardio- respiratory symptoms.
- High risk of postpartum haemorrhage than the general population

Foetal effects include(1,6,7): -

- Congenital malformations. The incidence of congenital anomalies in infants of diabetic women is related to the presence of hyperglycaemia induced free radical molecules which are embryotoxic in early gestation
- Caudal regression syndrome and syringomyelia have been characteristically described associated with diabetes; however, they are rare events. Cardiovascular malformations are the most frequent, being over-represented by transposition of the great arteries, truncus arteriosus and tricuspid atresia, followed by genitor-urinary tract, CNS (neural tube defects, anencephaly, and microcephaly) and musculoskeletal anomalies.

- Intrauterine growth restriction-occurs in upto 20% of diabetic pregnancies.
- Unexpected foetal demise- Proposed mechanisms include; decreased PH, increased PCO2 and lactate due to foetal hypoxia in utero from reduced blood volume as a result of villous oedema.
- Macrosomia-Hyperinsulinaemia and hypoxia combined with hyperglycemia results in glucose metabolism through the hexose –monophosphate shunt pathway with resultant increased triglyceride synthesis in foetal adipose tissue.

Preconception counseling should be done for patients with pre-existing diabetes. Conception should be prevented until euglycaemia is achieved. Oral hypoglycemic medication should be discontinued because these may be teratogenic, particularly if taken during organogenesis (first 8 weeks of pregnancy). Patients should be started on insulin preconception or as soon as pregnancy is diagnosed. The patient should be advised to start taking a prenatal vitamin with folic acid before attempting conception. [1] In our patient the diagnosis was made during third trimester.

Pregnancy management of women with pre-existing diabetes is multidisciplinary and will involve dietary therapy, glucose monitoring, exercise, and insulin therapy. The goal of dietary therapy is to avoid single large meals and meals with a large percentage of simple carbohydrates. The diet should provide 25-35kcal/kg body weight. The diet should be 40-50% carbohydrates, 30% fat and 20-30% protein. A total of 6 feedings per day is preferred with 3 major meals and 3 snacks to limit the amount of energy intake presented to the blood stream at any interval. Examples include foods with complex carbohydrates and cellulose, such as whole grain breads and legumes. The goal of exogenous insulin therapy during pregnancy must be to achieve diurnal glucose excursions similar to those of pregnant women who are not diabetic. The optimal glucose levels during pregnancy are fasting levels of 70-90mg/dl (3.9mmol/l-5mmol/l) or 2-hour postprandial values of 120-190mg /dl (6.7-10.6mmol/l) .The patient presented was on diabetic diet and insulin with good glycaemic control.

Because approximately 25% of diabetic patients develop pre-eclampsia, it is imperative to monitor blood pressure, proteinuria and the development of non-dependent oedema closely. Ophthalmologic, cardiac and renal function should be assessed at the initial visit and reassessed during the pregnancy as indicated. A urine specimen should be submitted for culture every trimester so that asymptomatic bacteriuria can be treated in a timely fashion. [1] Determination of maternal serum alpha-fetoprotein level should be carried out at 16 to 20 weeks gestation. A sonogram should be obtained at 18 to 20

weeks gestation to rule out foetal anomalies. Because infants of diabetic mothers are at risk for both macrosomia and IUGR, serial sonograms should be performed 4-6 weekly.

Because diabetic patients carry an increased risk of stillbirth, particularly in the 3rd trimester foetal testing with nonstress test, contraction stress test and biophysical profile is recommended [1, 6, 7] Our patient was on daily foetal kick chart monitoring.

In the absence of any obstetrical complication such as hypertension or abnormalities of foetal growth, the pregnancy should be allowed to go to term and await spontaneous labour. Vaginal delivery is aimed at and caesarean delivery reserved for the usual obstetric indications. However if estimated fetal weight is > 4000g elective caesarean delivery is recommended to prevent shoulder dystocia and birth trauma. [1,3] The estimated foetal weight in our patient was 3500 g and she was allowed to have a vaginal delivery.

If patient goes to spontaneous labour the ACOG recommends the use of regular insulin. Dilution of 25U in 250mls of normal saline is used. The level of blood glucose determines the rate of insulin infusion. Blood sugar is maintained at 3.8-5.O mmol/L. If blood glucose is less than 100mg/dl (5.5mmol/L), no insulin is infused. If blood glucose is between 100- 140mg/dl (5.5 – 7.8 mmol) infuse 1unit/hour. If blood glucose is 141 – 180mg/dl (7.8 – 10.0mmo1/l) infuse 1.5unitis/hr, if blood glucose is 180 -220-mg/dl (10-12.2mmo1/L), infuse 2units/hr. If above 220mg/dl (12.2mmol1/L, infuse 2.5units/hr.

Adequate rehydration is maintained at 125ml/hr of fluids. If blood sugar is below 7.8mmol/L use 5% dextrose in Ringer's lactate. If blood sugar is above 7.8mmol/L use normal saline. After delivery, the regime continues until normal meals are started. When labour is to be induced, the morning dose of insulin is not given and blood glucose is monitored closely. For elective caesarian section a similar infusion of insulin and dextrose is given and blood glucose level determined hourly. Operations are scheduled for early morning when sugar levels are normal. Additional insulin if required is given as bolus injections intra-operatively. In Our patient PGE_2 was inserted in the evening for cervical ripening. ARM was done in the morning followed by syntocinon. The morning insulin dose was omitted.

Neonatal adverse effects include(1,6):-

- Hypoglycaemia: Due to hyperplasia of the foetal B –islets cells induced by chronic maternal hyperplasia.
- Hypocalcaemia. The cause is unknown
- Hyperbilirubinaemia. Factors implicated include preterm birth and polycythaemia with haemolysis
- Cardiac hypertrophy leading to congestive cardiac failure: hyperinsulinaemia implicated in the pathogenesis.
- Respiratory distress syndrome: common with gestational diabetes rather than overt diabetes. fetal hyperinsulinaemia inhibits pulmonary surfactant production and may also interfere with glucorticoid enhancement of lung maturity .Up to 3% of infants develop RDS despite 2:1 lecithin:sphingomyelin ratio in amniotic fluid

The early neonatal period for the baby delivered was uneventful.

There is no single contraceptive method appropriate for all women with diabetes. Diabetes carries a risk of vascular disease, and the estrogens in oral contraceptives statistically increase the risk of thromboembolism, stroke, and myocardial infarction. Progestin only oral or parenteral contraceptives may also be used because of minimal effects on carbohydrate metabolism. There is possible increased risk of pelvic infections with intrauterine devices. Many overtly diabetic women elect postnatal sterilization, and this should be made readily available. [1]

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UNSENSITIZED RHESUS NEGATIVE MOTHER – SVD LIVE BABY

NAME	J. K. N	IP NO.	1092463
AGE	25YEARS	L.M.P:	2.10.05
D.O.A:	5. 07.06.	E.D.D:	9. 07.06
D.O.D:	10.07.06	G.B.D	: 39WEEKS
PARITY:	0+1		

Presenting complaint

She was admitted for induction of labour due to her rhesus negative status at term (39 + weeks).

History of presenting complaint

The patient was well and had no complaints. The foetal movements had remained adequate. She was admitted for induction of labour due to her rhesus negative status at term (39 weeks).

Past obstetric and gynecological history

She was a para 0+1, gravida 2, her LMP was on 2.10.05, and her EDD was on 9.07.06, hence maturity by dates was 39+ weeks. Her 1st pregnancy was in 2001; she had an abortion in December 2005 at 3 months. However her rhesus status was unknown and no Anti-D was given.

Her menarche was at 15 years and her cycle was regular with a length of 28 days and duration of 3 days. She had never used any contraceptives.

Antenatal care

She had attended ANC at KNH from 28 weeks gestation, she came for 5 visits. Her antenatal follow-up was un-eventful. The antenatal profile was as follows: VDRL- Negative; Blood group - O Negative; Hb-10.2 g/dl; Husbands blood group- A positive; Indirect Coomb's Test (ICT) – negative. She received 2 doses of tetanus toxoid.

The antenatal period was uneventful. Weight gain, BP and urinalysis remained normal.

Past medical history

She had never been transfused, a known asthmatic with no recent history of an attack. The patient has history of reacting to penicillin containing drugs.

Family and social history

She was married and stays in Ngong with her husband. She didn't drink alcohol or smoke cigarettes. Her sisters are twins and there is no chronic illness in the family.

Systemic inquiry

Non-revealing

PHYSICAL EXAMINATION

General examination:

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

Observations

The BP was 110/70mmHg, Temperature 36.5^{0}C, pulse 80BPM regular; Respiratory rate was 20 per minute.

Systemic examination.

The central nervous system, respiratory system and the cardiovascular system were essentially Normal

Abdominal examination

The abdomen was uniformly distended. The fundal height was term, cephalic presentation; the descent was 4/5, she had no contractions, the foetal heart was heard and regular at 142 BPM.

Vaginal examination

She had normal external genitalia; the cervix was closed, was 1.5cm long, posteriorly and soft. The pelvis was clinically adequate. The bishop score was 4 hence not favourable.

Diagnosis: para 0 +1 unsensitized Rhesus Negative mother at term with an unfavourable bishop score was made.

Diagnosis: para 0 +1 unsensitized Rhesus Negative mother at term with an unfavourable bishop score was made.

MANAGEMENT

She was admitted to the antenatal ward to a wait purchasing of Prostaglandin E_{2a} pessary for induction of labour. Six hours latter A Prostaglandin E_{2a} 3mg pessary was inserted into the posterior fornix. The foetal heart rate was monitored half hourly. On review after 8 hrs, she was comfortable with no lower abdominal pains; the cervix was admitting a finger but still posterior. The second prostaglandin pessary was inserted. She complained of lower abdominal pains 6 hour latter. On examination she was having 2 contractions every 10 minutes each lasting 20 seconds, foetal heart was regular at 136/min, the presenting part was 3/5 up. On vaginal examination the cervix was 4 cm dilated and she was draining clear liquor. She was started on a partograph and syntocinon 5IU in 5% dextrose10 drops per minute (DPM) to be increased by 10 DPM every ½ hour till a maximum of 60 DPM or till 3 strong contractions every 10 minutes each lasting over 40 seconds were achieved. On the next review after 4 hours, she reported an urge to bear down. On examination she was found to be in second stage of labour. She progressed well, and had SVD to a live female infant, whose birth weight was 3200 gm, Apgar score of 9/1, 10/5 and 10/10.

At delivery, the cord was clamped immediately and cord blood was taken for blood group, Haemoglobin level, and direct coomb's test and bilirubin levels. The infant was taken to NBU for observation. The mother was stable. The baby's blood group was O Rhesus positive, the direct coomb's test was negative; the Haemoglobin and bilirubin levels were normal. The baby was discharged from NBU after 24 hours with no complications. The mother opted not to be given anti-D since she had no desire for another baby.

Postnatally, the mother and baby did well and they were discharged home to be reviewed in the postnatal clinic after six weeks, she was found to be well and referred to the family planning unit for a method. She was advised of the need to inform her next health obstetrician of her status. To be given ant-D incase any pregnacy lose in future.

DISCUSSION

The case presented is of a 30 year old para 0+1 unsensitized Rhesus D negative mother who delivered a live female baby by spontaneous vertex delivery (SVD). The baby's blood group was O positive but the mother opted not to be given anti-D since she had no desire for another baby.

In the red blood cells, there are about 250 recognized antigenic factors of which the most common are ABO, Rhesus, Kell, Lutheran, Duffy, Kidd, P and MNs. Landsteiner and Weiner discovered the Rhesus factor in 1940 the presence of which makes an individual Rhesus positive and its absence Rhesus negative. The Rhesus factor antigens are lipoprotein antigens confined to red blood cell membranes and are inherited independently of all other blood group antigens (1, 2).

Fisher and Race found out that , there are three pairs of antigens (Dd Cc Ee) which are inherited in two sets of 3, one set from each parent. They are located in the short arm of chromosome 1. The gene that makes a person Rh-positive is D. Inheritance is by a Mendelian dominant manner; i.e. a homozygous Rh-positive father (DD) will pass the D gene to all his offspring, whereas a heterozygous Rh-positive father (Dd) will have an equal chance of having a Rh-positive or Rh-negative baby by a Rh-negative mother (2,3,12,13). The rhesus antigens Cc Ee are considered to be of lower immunogenicity than the D antigen which is responsible for severe haemolytic disease of the new born.

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

The initial response of a rhesus negative individual to rhesus positive blood is the formation of IgM antibodies which do not cross the placenta barrier; subsequently IgG antibodies are formed that cross the placenta. (Rhesus antigens are well developed by 30 days gestation) or after a transfusion of Rh-positive blood (2, 3). Our patient had no history of previous blood transfusion. Foetal cells do enter the maternal circulation but in small amounts, which are destroyed by maternal immune

system before provoking an antibody reaction especially where the ABO blood group of the mother and foetus are incompatible. In the general population, foetal red cells have been detected in maternal blood in 7% of women in the first trimester, 16% during the second trimester and 29% during the third trimester with no apparent predisposing factor. The result of this antepartum foeto-maternal haemorrhage is an overall rate of Rhesus sensitization before labour of about 1 to 2% before delivery. Foeto-maternal haemorrhage has also been detected following abortion or ectopic pregnancy (2, 3, 12, 13). Other conditions which may increase the chances of foeto-maternal haemorrhage are caesarean delivery, multiple gestation, and antepartum haemorrhage, manual removal of placenta, external cephalic version, chorionic villus sampling and amniocentesis. Our patient had none of these conditions or procedures.

The amount of red blood cells necessary to cause isoimmunization in a rhesus negative patient is not known. As little as 0.1ml of Rh-positive red blood cells has been shown to sensitize Rh-negative volunteers. Overall, about 16% of Rh-negative women will become alloimmunized by their first Rhesus incompatible (ABO compatible) pregnancy if not treated with Rhesus immunoglobulin. Of these, 1.5-2% of the isoimmunization will occur intrapartum, 7% within 6 months of delivery and the remaining 7% early in the next pregnancy most likely as a result of amnestic response (3).

The risk of Rhesus isoimmunization seems to be less than 2% after infusion of relatively small volume (<30mls) of Rhesus incompatible cells as would occur in multiple deliveries. With infusion of a large volume (>200ml) the risk is slightly greater than 8%. However at no volume, does there seem to be 100% immunization risk (1, 2).

Whether or not a rhesus-positive foetus immunizes an at-risk rhesus-negative woman, depends on a number of factors; first, 30%of Rh-negative individuals appear to be immunologic 'non responders' who will not become sensitized, even when challenged with large volumes of Rh-positive blood. Second, ABO incompatibility diminishes the risks of alloimmunization to about one-tenth of that when they are ABO-compatible. In our patient the mother was type O and the infant was type O hence the ABO grouping was compatible. Third, there is some variation in the strength of the rhesus antigenic stimulus, depending on the rhesus genotype of the foetal blood, e.g. the Cde/cde genotype seems to be relatively 'strong'. Fourth, the volume of foetal blood entering the maternal circulation is important, with 0.25ml representing a critical sensitizing volume and with the likelihood ad severity of sensitization increasing with greater volumes (4).

The initial (primary) Rhesus immune response is slow to develop, usually requiring 6-12 weeks and sometimes up to 6 months. It is predominantly immunoglobulin M (1gM). This does not cross the placenta due to the high molecular weight. Subsequently, 1gG antibodies are formed and these cross from mother to foetus and cause haemolysis, hydrops fetalis, and kernicterus depending on the extent of haemolysis. (2,3,12,13) Risk factors that predispose to feto-maternal haemorrhage include amniocentesis, abortion, abdominal trauma, abruptio placenta, caesarean section, antepartum haemorrhage and manual removal of placenta (1,3).

The introduction and widespread use of anti-D gamma globulin has made the frequency of sensitized pregnancy to decline. This immunoglobulin prevents Rhesus isoimmunisation by competitive inhibition.

All the antigenic sites are covered or blocked from the lymphoid cells by the antibody. It may also interfere with the foetal red cells antigen processing by maternal macrophages, thus preventing the initiation of immune response (1, 7).

The management of Rh-negative women involves early detection of their sensitization status. It is generally recommended that each mother should have her ABO blood group and Rhesus status checked at the first antenatal visit of each pregnancy. If the woman is Rh-negative, she should have an indirect coomb's test to check for antibodies. The paternal ABO and Rhesus blood group is also checked. In unsensitized cases, like our patient, indirect Coomb's test is repeated at 28 weeks if negative, anti-D immunoglobulin 300ug is given .Our patient did not receive Ant- D antenatally. A repeat antibody screening is done again at 35 weeks gestation. If the test remains negative the patient is delivered at 40 weeks gestation to avoid risks of sensitization. The routine administration of anti-D after delivery or abortion prevents up to 95% of Rhesus sensitisation. There is 1-6% failure rate of prophylactic anti D when given after delivery compared to 0.1% when given antenatally (8, 9). The standard recommendation is to give anti D within 72 hours postpartum, but women at risk who have not received this regimen within 72 hours would still be treated. In fact some authors recommend treatment up to 28 days postpartum (3).

Where the mother is sensitised the maternal antibodies should be quantified. If the titres remain below 1:16, further measurements are repeated at timely intervals of 2 to 4 weeks and serial, amniocentesis and ultrasound assessments of the foetus are done. When the baby is being compromised ultra-sound features of severely anaemic foetus are skin oedema, ascites, pericardial

or pleural effusion. For titres above 1:16, a further assessment with amniocentesis or foetal blood sampling to assess the disease of haemolysis is performed. After amniocentesis, the amniotic fluid is analysed for amounts of bilirubin, which provides an indirect measure of haemolysis. The amniotic fluid is analyzed by spectrophotometry plotted on a semi-logarithmic versus gestation (Liley's chart). Further management will depend on level of haemolysis based on Liley's chart and gestational age.

Zone I generally indicates an unaffected foetus or one who will have a mild disease but a D negative foetus is also a possibility.

In zone II the foetus is at moderate to severe risk of haemolytic disease. A repeat amniocentesis or foetal blood sampling may be required to establish the actual condition of the foetus.

In zone III the foetus is severely affected and death within one week to ten days may be expected unless intrauterine transfusion or delivery is effected (1, 2). Direct intravascular transfusion into the circulation (umbilical vein, hepatic vein or intra-cardiac) is done under ultrasound guidance. This has improved the outcome of foetuses with severe anaemia and hydrops fetalis. Due to lack of these facilities perinatal mortality and morbidity remains high among the isoimmunised women in our set up. In Kenyatta National Hospital a perinatal mortality of 600 per 1000 was reported (11).

Other modes of treatment which have been tried unsuccessfully include plasmaphoresis, immunosuppression with high dose of steroids, promethazine and D-positive erythrocyte membranes in enteric coated capsules to induce T- suppressor cell formation (3, 9). For the patient who has been sensitized and has had repeated pregnancy losses artificial insemination by Rhesus D negative donor sperm can be done. OUR patient had only had one pregnancy loss which could have unlikely been from alloimmunization because it was the first pregnancy.

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Obstetrics case 14

FOETAL DISTRESS

NAME: E W. IP NO. : 1104172

AGE: 21 YEARS LMP: 22/9/05

D. O. A.:16/7/06 E.D.D: 29/6/06

D. O. D.:20/7/06 GESTATION 40 WEEKS

PARITY: 0+1

Presenting complain

Patient presented with complains of labour pains for three hours prior to admission.

History of presenting complains.

The patient had been well till three hours prior to admission when she started experiencing lower abdominal pains which intermittent and increasing in frequency and intensity. She gave no history or draining liquor.

Past Medical History

She had no history of admission or surgical operations. There was no history of chronic illness in the family.

Obstetric and gynaecological history.

She was a para 0+1, having had spontaneous complete abortion in 2005 at 22 weeks. Her LMP was on 22/9/05, EDD was 29//06, and hence maturity by date was 40weeks.

Her menarche was at 12 years and her cycle length was at 28 days. Her periods lasted 3-4 days and were regular and normal flow. She had never used any methods of contraception.

Antenatal care.

She attended antenatal care at Kenyatta national Hospital from her 36th week of gestation. The antenatal period was uneventful.

Her ANP were as follows;

Haemoglobin level 10g/dl

VDRL was negative.

HIV test was negative.

Blood group A positive.

Family and social history

She is a married housewife who stays in Maringo. She does not drink alcohol or smoke cigarettes. There is no history of chronic illness or twinning.

PHYSICAL EXAMINATION.

She was in good general condition, she was not pale or febrile, and had no oedema.

Observation.

The BP was130/80 mmHg, temperature 32.6°C, pulse 88BPM regular, respiratory rate was 18/min.

Abdominal examination.

The fundal height was term. The lie was longitudinal and presentation was cephalic which was engaged. Foetal heart was heard and was irregular. She had moderate contractions, 2 in 10minutes lasting over 30 seconds each. Clinically estimated foetal weight was 300g.

Vaginal examination

She had a normal external genitalia; the cervix was 4cm dilated, soft central and partially effaced. The membranes were bulging and intact but the cord not felt. Artificial rapture of membrane was done using a sterile kocher and meconium stained liquid grade 3 was obtained.

A diagnosis of foetal distress in labour was made.

MANAGEMENT

The patient was asked to lie in the left lateral position and oxygen was maintained. She was told that she will have to undergo a caesarean section. She gave an informed consent. She had her pubic hair shaved. Blood was taken for group and crossmatch. She was given atropine 0.6mg intravenous before she was taken to theatre. In the theatre she was explained to about the procedure and anaesthesia was given. She was put in supine position and catherized.

Her abdomen was cleaned with chlorohexidine and iodine and then she was draped. She was given general anaesthesia .Subumblical midline incision was made. The rectus layer was dissected with scissors. The abdominal cavity was parked with abdominal packs, a lower transverse uterine

incision was made, and a live male infant who weighed 3 kg was delivered and scored 6/1, 8/5, 8/10. There was meconium stained liquor grade 3.The umbilical cord and placenta were normal. The placenta was delivered by controlled cord traction. The cause of distress was not apparent in intraoperative findings. The uterus was sutured into two layers with the achievement of haemostasis. The abdominal packs were removed, the instrument and gauze count was made ad was correct.

The abdomen was closed in two layers, the rectus and the skin. Vulvovaginal toilet was done. The anaesthesia was reversed successfully. She remained stable throughout recovery period. She was put n pethidine 100 mg 8 hourly for analgesia, penicillin 2 mega units 6 hourly and gentamycin 80 mg 8 hourly for prophylaxis. The baby was taken to the newborn unit for observation and joined with the mother after two days in stable condition. She stayed in the ward for 4 days and remained instable condition. She was discharged home on Brufen for pain control. She was booked to be seen after two weeks in the postnatal clinic.

DISCUSSION

The term foetal distress is too broad and too vague to be applied with any precision to clinical situations. It may be defined as progressive fetal asphyxia that, if not circumvented will result in decompensation of physiological responses (primarily redistribution of blood to preserve oxygenation to vital organs) and cause permanent central nervous system and other damage or death (1). Foetal distress may be acute or chronic. Skilful monitoring will detect some degree of foetal compromise in at least 20% of all obstetric patients. Normal parturition is an asphyxiating event for the fetus (2, 3, 4).

The metabolic derangements signaling acute fetal distress are hypoxia, hypercapnia, and acidosis. Usually asphyxia, or hypoxia, is induced by only three mechanisms: decreased maternal uteroplacental blood flow, decreased maternal oxygenation, and decreased umbilical blood flow. During a moderate asphyxic episode, then fetus can compensate by the diversion of blood flow to the most vital organs, by limiting oxygen consumption over a prolonged period, (e.g. postmaturity) or by shifting to anaerobic metabolism. If the asphyxic stress is severe, prolonged, or becomes more pronounced, these compensatory mechanisms ultimately fail, and fetal distress becomes evident.

In chronic fetal compromise, reduced placental perfusion result from maternal conditions such as pregnancy induced hypertension, chronic hypertension, diabetics, severe heart disease, long term pulmonary shunting or residence at high altitude. Placental causes of chronic fetal compromise

include premature placental aging and diabetic mellitus. Fetal causes include the postmaturity syndrome, multiples gestation, twin to twin transfusion, congenital anomalies, maternal fetal transfusion and erythroblastosis fetalis (3).

Causes of acute fetal compromise include (3)

- Maternal – hypotension (supine hypotension syndrome), hypoxia, impaired respiration, PET, shock, and sickle cell crises
- Uterine - hypertonia or polysystole, excessive oxytocin, uterine rupture
- Placental – abruptio placenta, placenta previa, premature placental aging.
- Cord – prolapse, ruptured vasa previa, tight or short cord, true knot
- Fetal – cardiac failure, congenital anomaly, haemorrhage, isoimmunization

The objective of monitoring the foetus in labour is to detect foetal abnormalities at a stage when they are reversible. The current modalities for the monitoring of the foetus are intermittent auscultation using the Pinard stethoscope or a portable Doppler fetal heart detector, cardiotocography (continuous electronic fetal monitoring), colour and quality of amniotic fluid and foetal blood sampling. Biophysical profile, moulding of the foetal head and caput formation serve as accessories to monitor the foetus.

It is easier to define a normal fetal heart rate pattern than an abnormal one. Analysis of the pattern should always be performed systematically, with assessment of the baseline rate and variability, and the presence or absence of accelerations and decelerations. A reassuring baseline fetal heart rate lies between 110 and 160 beats/min, heart rate variability of 5 - 25 beats/min, accelerations (increases in rate of more than 15 beats/min for more than15 s) and no decelerations. No foetal compromise is present when there is absence of any abnormality of foetal rate (FHR) or rhythm and no response to uterine contractions other than early decelerations (2). Our patient had a baseline fetal heart rate ranging between 110 and 152 beats/min.

The commonest fetal heart rate decelerations are synchronous with contractions, and if small (less than 40 beats/min) are called early, while if large (more than 40 beats/min) are called variable. Variable decelerations also tend to vary in amplitude and timing in relation to the contractions,

hence their name. The commonest cause of variable decelerations is cord compression, rather than hypoxia per se.

However, hypoxia can develop if the cord compression is severe enough and prolonged enough to reduce substantially the blood flow through the umbilical artery. This is particularly important if there is meconium in the amniotic fluid, as the resultant gasping can cause meconium aspiration syndrome. In addition, some studies have suggested that the large fluctuations in fetal blood pressure that result from prolonged and severe intermittent cord compression can result in intracranial haemorrhage, and thus in long-term handicap.

Therefore, variable decelerations should not be regarded as innocuous, and should always be considered an indication for requesting the presence of the paediatrician at delivery. Late decelerations of the fetal heart rate are less common, but almost invariably indicate a degree of hypoxaemia in the fetus. They occur because the reduced oxygen tension in the blood supplying the fetal coronary arteries leads to the generation of lactic acidosis secondary to anaerobic metabolism in the myocardium, which has the effect of slowing the fetal heart rate (5). A cardiotocograph done on our patient revealed variable decelerations.

Measurement of pH in capillary scalp blood may help to identify the foetus in serious jeopardy. The pH of the foetal capillary scalp is normally lower than umbilical venous blood and approaches that of umbilical arterial blood. The following protocol has been recommended to try and confirm foetal distress: If pH is greater than 7.25, then labour is observed. If the pH is between 7.20 and 7.25, pH measurement is repeated within 30 minutes. If the pH is less than 7.20, another scalp blood measurement is taken immediately and the mother taken to the operating room for preparation for surgery. If the pH is still low immediate caesarean delivery is performed (7). In our setup, foetal scalp blood is not done and hence it is difficult to objectively determine the foetus that is actually having foetal distress. Foetal blood sampling was not done in our patient.

Obstetricians have long realized that detection of meconium during labour is problematic in the prediction of fetal distress or asphyxia.

Three theories have been suggested to explain fetal passage of meconium: the pathological explanation proposes that fetuses pass meconium in response to hypoxia, and that meconium

therefore signifies fetal compromise. Alternatively, in utero fetal passage of meconium may represent normal gastrointestinal tract maturation under neural control.

Third, passage of meconium could also follow vagal stimulation from common but transient umbilical cord entrapment and resultant increased peristalsis.

The presence of thick meconium in labour particularly in association with post-term pregnancy, oligohydramnios and poor foetal growth has been associated with increased risk of acidemia which then increases the risk of meconium aspiration (4). Ramin (1996) hypothesized that the pathophysiology of meconium aspiration syndrome includes, but is not limited to, fetal hypercabia, which stimulates fetal gasping leading to aspiration of meconium (3). Meconium staining without foetal heart abnormalities or foetal scalp abnormalities and with labour progressing well seems to have no great significance. Meconium in the presence of complicated labour with foetal heart rate abnormalities has a greater risk of foetal hypoxia than either meconium alone or foetal heart rate abnormalities alone (8). Our patient had meconium stained liquor grade 2 with foetal heart rate irregularity and bradycardia.

Intrauterine resuscitation (IUR) for foetal compromise will help improve the condition of the foetus and may help avoid unnecessary intervention. Intrauterine resuscitation measures include:

1. Maternal position change - turning the patient on her left lateral position usually improves fetal heart rate abnormalities caused by aorta-caval compression. However in some cases the abnormality may be caused by cord compression which is unrelieved or made worse in the left lateral position. Right lateral or knee elbow position must then be tried. A good rule in the labour room is that all patients should recline as much as possible in a semi lateral position (9).
2. Intravenous fluids – a rapid intravenous infusion of 1 liter of non glucose containing crystalloid is commonly recommended as part of IUR. Most evidence at present suggests that an intravenous preload reduces the FHR changes after epidural blockade. Ephedrine is the drug of choice if hypotension due to conduction anaesthesia is significant or persistent. In the past, bolus doses of hypertonic dextrose were used for the management of foetal distress. But the use of dextrose has been shown to be of little value (6).

3. Oxygen administration - supplemental oxygen to the mother results in improved foetal oxygenation, assuming that placental exchange is adequate and umbilical cord circulation is unobstructed.

4. Discontinuation of Oxytocin – decreased uterine activity permits better placental perfusion.

5. Tocolysis - agents such as Beta-adrenergic agonists (terbutaline) and nitroglycerin can be administered to decrease uterine activity in the presence of uterine hypertonus with non reassuring foetal heart rate patterns. As a single intervention, tocolysis is probably more useful than maternal oxygen inhalation.

6. Amnioinfusion - should be considered for repetitive, non reassuring variable decelerations. The therapeutic goal is expansion of the amniotic fluid volume. Amnioinfusion has been shown to the useful in the management of variable decelerations in fetuses with oligohydramnios and subsequent cord compression. Amnioinfusion has also been used for dilution and lavage of meconium (6).

7. Maneuvers for umbilical cord prolapse – positioning the patient in the elbow knee position or semi prone (Sims) position, manual elevation of the presenting part per vaginum or insertion of a foley catheter into the bladder and instillation of 0.5L of fluid to elevate the presenting part. A steep trendelenburg position does not relieve aorta-caval compression and may cause maternal respiratory difficulty (9).

8. If maternal acidosis is the cause of foetal acidosis, administering bicarbonate to the mother may benefit both patients.

9. Vibroacoustic stimulation (VAS) or foetal scalp stimulation may be used to induce accelerations in foetal heart rate that indicate the absence of acidosis.

If there is continued foetal distress despite conservative treatment, immediate delivery is mandatory. Obstetric judgment must dictate how the delivery will be accomplished in accordance with the presentation, station, position, dilatation of the cervix and presumed fetal status. If caesarean section is chosen, it must be done rapidly (2).

The form of handicap most typically associated with intrapartum asphyxia is cerebral palsy, with a generalized motor deficit and relative intellectual sparing (5).

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Obstetric case 15

PRETERM PROM AT 32 WEEKS GESTATION – PRETERM LABOUR, LIVE BIRTH

NAME: L.K.
IPNO: 1102642
AGE: 21 YEARS
LMP:16/11/05
DOA: 26/06/06
EDD: 23/08/06
DOD: 7/07/06
GESTATION. 32 Weeks
PARITY: 1+0

Presenting Complaint

She presented with a 20 hours history of drainage of liquor.

History of the presenting Complaint

She was well until the day before admission when she had a sudden gush of fluid from the vagina while walking. The fluid was colorless and flowed down the legs to the floor. There were no associated abdominal pains and no accompanying per vaginal bleeding. There was no history of trauma, dysuria or frequency of maturation. Fetal movements were present.

Past Obstetric and Gynaecologic History

She was a para 1+0. Her previous pregnancy was in 2001, she had a spontaneous vertex delivery at term to a male infant whose birth weight was 3.5kg and was alive and well.

She attained menarche at 16 years. She had regular menses after every 28 days lasting 3 days. There was no history of contraceptive use.

History of present pregnancy

She was a para1+0 and her LMP was on the 16/11/05 an EDD of 23/8/06 giving a maturity of 32 weeks. She had not started attending antenatal clinic. Antenatal profiles done while admitted were normal; hemoglobin 10.1 g/dl, negative VDRL and HIV tests and blood group B positive. An ultrasound scan done on 11th May revealed a single viable intrauterine fetus with an average computed gestational age of 15 weeks 5 days. There were no fetal anomalies.

Past Medical History

She had no significant medical or surgical history.

Family and Social History

She was a single house-help who lived in Easghlie. Her first child lives with her mother in Makueni. She did not smoke cigarettes or drink alcohol. There was no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in good general condition, not pale, afebrile, not jaundiced, and had no oedema. She had a pulse rate of 84 beats per minute, a respiratory rate of 20 breaths per minute, a blood pressure of 110/60 mmHg and a temperature of 36.0^0C.

Systemic Examination

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

Abdominal Examination

The abdomen was distended and moving with respiration. There were no areas of tenderness. The liver and spleen were not palpable. The uterus corresponded to 30 weeks gestation. The foetus was in longitudinal lie and cephalic presentation and the descent was 4/5. The foetal heart tones were heard and regular at 140 beats per minute. There were no contractions felt.

Speculum Examination

She had normal external genitalia. The vaginal walls and the cervix were healthy. There was a pool of clear fluid in the posterior fornix and active drainage of liquor was noted from the cervical os on valsava maneouvre. There was no cord prolapse.

Diagnosis

A diagnosis of premature rupture of membranes at 31 weeks gestation was made.

Investigations.

1. Obstetric Ultrasound scan showed a single viable intra-uterine pregnancy at 33 weeks with normal liquor. Normal fetal movements and fetal cardiac activity was at 145 beats per minute. Gross fetal parts were normal.

2. Haemoglobin. WBC 10.8 x 10^9 /L
   Hb 10.7 g/dl
   Platelates 192 x 10^9/L
3. Urine for microscopy culture and sensitivity no growth obtained
4. High Vaginal Swab(HVS) wet preparation= no pus cell, no T Vaginalis, no RBC (red blood cells), no yeast cells. Culture (HVS) no growth obtained.

MANAGEMENT

She was admitted to the admitting antenatal wards for conservative management. She was put on bed rest and oral Erythromycin 500mg 8 hourly and metronidazole 400mg 8 hourly. Intramuscular Dexamethasone was given 12mg 12 hourly, 2 doses to facilitate fetal lung maturation. A high vaginal swab was taken for microscopy culture and sensitivity. Vital signs were taken 4 hourly and daily examination of the abdomen for any palpable tenderness, examination of the liquor for any fowl smell or change in colour. No signs of sepsis were noted during the period for conservative management.

Ten days after admission she went into spontaneous labour and was augmented with 5 IU of intravenous syntocinon in 500mls of dextrose 5% which was titrated to contractions. Labour progressed uneventfully and she had a spontaneous vertex delivery to a live female infant who weighed 2300g and had an Apgar score of 9 and 10 in 1 and 5 minutes respectively. She was allowed to room in immediately.

She and the baby remained stable postnatally. She was discharged home on the third post natal day through the post natal clinic.

Post natal follow up

She was seen in the postnatal clinic after 6 weeks and had no complaints. Examination findings were normal. She was yet to choose a method of contraception because currently dint have a partner, she was however encouraged to take with her some condoms.

DISCUSSION

L.K. was a 21 year old para 1+0 who presented with premature rupture of membranes at 32 weeks gestation. She was admitted for conservative management and ten days latter went into spontaneous labour and had an SVD to a live female infant who weighed 2300g and had a good Apgar score.

Premature rupture of the membranes is defined as spontaneous membrane rupture that occurs before the onset of labor. When spontaneous membrane rupture occurs before 37 weeks' gestation, it is referred to as preterm PROM. Preterm PROM is further sub-divided into "previable PROM," which occurs before the limit of viability (less than 23 weeks), "preterm PROM remote from term" (from viability to about 32 weeks' gestation), and "preterm PROM near term" (approximately 32–36 weeks' gestation) (1).

PROM occurs in approximately 10.7% of all pregnancies. In approximately 94% of cases, the fetus is mature. Premature foetuses (1000-2500g) account for about 5% of the total number of cases while immature foetuses (<1000g) account for less than 0.5% [(2)]. The incidence of PROM at Kenyatta National Hospital has been cited as 6.2 to 9.3%. (3,4,5)

Eighty five percent of neonatal morbidity and mortality is as a result of prematurity and preterm premature rupture of membranes (PPROM) is responsible for approximately 30% of all preterm births(6). Even with conservative management, 80% of women with preterm PROM remote from term will deliver within 1 week of membrane rupture. The incidence of dysfunctional labour, chorio-amnonitis, cesarean delivery, post-partum haemorrhage, endometritis, and neonatal infection are higher with PROM compared with term deliveries.

The frequency and severity of neonatal complications after preterm PROM vary with the gestational age at which rupture and delivery occur, and are increased with perinatal infection, abruptio placenta, and umbilical cord compression (due to Oligohydroamnos)(7). Chronic Oligohydroamnos may lead to compression deformity of the fetus, these include; facial deformities (low set flat ears, Down sloping tip of nose, slopping chin), deformities of the extremities (flexure contractures of the elbows, knees, and feet) and pulmonary hypoplasia. This appearance similar to potters syndrome. Infants with compressional deformities die due to the chest-lung hypoplasia (7).

The etiology of premature rupture of membranes is multifactoral in nature. In any given patient, one or more pathophysiologic processes may be evident.[8] It is clear that maternal enzymes,

maturational and mechanical forces, chorion-amniotic membrane phospholipids content, collagen disruption, amnion cell cytokines, induced by fetal signals and bacterial phospholipases, and collagenases all play a major role in the onset and progression of PROM. Enzymes with collaginolytic activity alter the structure of the collagen fibrils and leading to mechanical strain, breakage and exposing the membranes to bacterial invasion. These enzymes include: trpsin, collagenases, metalloproteases, gelatinase, and protioglycanase and cystein protinases. Maturational factor are involved because there is decreased membrane collagen content has been demonstrated in the setting of preterm PROM and with increasing gestational age. In pregnancies complicated with PROM there is a noted decrease in type III collagen which causes the loss of the membrane elasticity and strength. Similarly there is increased hydrophobicity of the membranes with increasing cervical effacement and dilatation. . Other factors associated with preterm PROM include lower socioeconomic status, familial history of premature rupture of membranes, cigarette smoking, sexually transmitted infections, prior cervical conization, prior preterm delivery, prior preterm labor in the current pregnancy, uterine distention (e.g., twins, hydramnios), cervical cerclage, amniocentesis, dietary deficiencies in ascorbic acid, zinc or copper deficiency and vaginal bleeding in pregnancy. Each of these may be associated with preterm PROM through membrane stretch or degradation, local inflammation, or a weakening of maternal resistance to ascending bacterial colonization.

The common important path-way used by all these forces is: the amnion producing cytokines (IL 6, IL8) in conjunction with fetal IL-1 beta these induces the production of prostaglandins some being potent oxytocytics. In many cases, the ultimate cause of premature membrane rupture is unknown. [8]

Diagnosis of membrane rupture is based on a history of gush of watery fluid or persistent wetness, demonstrated by a pooled amniotic fluid in the posterior vaginal vault or leakage from the cervical canal on examination with a sterile speculum. A fern pattern in dried amniotic fluid (on a glass slide for 10 minutes), a PH of the fluid of more than 7 as seen by colour change of Nitrazine paper. The Nitrazine test can be "falsely positive (colour change of Nitrazine paper from yellow to blue)" if the vaginal pH is increased by blood or semen contamination or alkaline antiseptics, or if bacterial vaginosis is present. The fernning test should be performed on a sample collected from the posterior fornix or lateral vaginal sidewall to avoid cervical mucus, which may also yield a false positive result. Other plausible causes of vaginal discharge (e.g., urinary incontinence, vaginitis, cervicitis, mucous "show", semen, vaginal douches) should be excluded if the diagnosis is unclear. Should

initial testing be negative but a clinical suspicion of membrane rupture remain, the patient can be retested after prolonged decumbency or alternate measures can be considered. Ultrasound evaluation may prove useful if the diagnosis remains in doubt after speculum examination. The diagnosis of membrane rupture can be confirmed unequivocally with ultrasound-guided amnioinfusion of indigo carmine (1 mL in 9 mL of sterile normal saline), followed by observation for passage of blue fluid per vaginum. Cervico-vaginal screening for fetal fibronectin has been suggested as a marker for preterm PROM when the diagnosis remains in doubt after initial speculum examination. However, it is not recommended for routine practice. [7] Our patient presented with history of gush of watery fluid and on speculum examination there was pooling of amniotic fluid in the posterior vaginal fornix and fluid was also observed draining from the cervical os on valsava maneuver.

Digital cervical examinations should be avoided, this has been shown to decrease latency and increase infectious morbidity (9, 10).

Initial laboratory studies should include a complete blood count with differential. In preterm pregnancies, urinalysis culture and sensitivity should be done.

Ultrasounds scan for foetal size and amniotic fluid index and amniocentesis in some cases to determine foetal lung maturity and the presence of infection may be done (7).

In management PROM the important factor to consider are; the gestational age and the availability of neonatal support services, presence or absence of labour, presence of overt or subclinical infection, stability of fetal presentation and the heart rate tracing, the degree of fetal lung maturation and the degree of cervical effacement and dilatation. The two basic options are immediate delivery and expectant management (7). The principle hazards of expectant management are: ascending infections, umbilical cord prolapse, umbilical cord compression due to Oligohydroamnos and abruption placenta. The principle hazards of immediate delivery are complications of prematurity, including respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), sepsis, necrotizing enterocolitis (NEC), thermal instability, metabolic derangements, apnea, bradycardia, patent ductas arteriosous, (PDA), and poor feeding. Of all these the 4 most likely to lead to neonatal death are RDS, IVH, sepsis and NEC, all which are significantly more likely to occur at gestation below 32 weeks than those after 32 weeks. Although there is no apparent neonatal benefit to conservative management after membrane rupture at term, there is a potential for neonatal benefit when conservative management of preterm PROM is undertaken for the immature fetus. The woman with preterm PROM should be evaluated clinically for evidence of advanced labor,

chorioamnionitis, abruptio placenta, and fetal distress. Those with advanced labor, intrauterine infection, significant vaginal bleeding, or non-reassuring fetal testing are best delivered regardless of gestational age(7). Our patient did not have any of the above there were no features of chorioamnionitis and the fetal status was reassuring.

When preterm PROM occurs at 34–36 weeks' gestation, the risk of severe acute morbidity and mortality occurring is low when expeditious delivery is pursued. Corticosteroids are generally not given to accelerate fetal pulmonary maturity. If fetal pulmonary immaturity is suspected at 32 to 33 weeks, an option would be to treat conservatively with close fetal monitoring, adjunctive antibiotic therapy, and antenatal corticosteroid administration for fetal maturation(11). L.K was given the corticosteroids to aid in the fetal lung maturation.

The stable gravida with preterm PROM remote from term is generally best served by conservative management in an attempt to prolong pregnancy and reduce the risk of gestational age– dependent morbidity in the newborn.

Conservative management generally consists of initial prolonged continuous fetal (nonstress test, biophysical profile, fetal heart rate monitoring) and maternal monitoring combined with subsequent modified bed rest to increase the opportunity for amniotic fluid re-accumulation and spontaneous membrane sealing. Complete pelvic rest and avoidance of digital pelvic examinations should be undertaken to reduce the risk of intrauterine infection and enhance latency (7, 10).

The combination of fever with uterine tenderness and/or maternal or fetal tachycardia, a rising white blood cell count in the absence of another source of infection, are suggestive of intrauterine infection and should lead to expeditious delivery. If amnionitis is suspected but the diagnosis is equivocal, amniocentesis should be considered with Gram stain, amniotic fluid white blood cell count (AF-WBC), leukocyte esterase test and glucose level (7).

There appears to be a role for adjunctive aggressive antibiotic therapy with erythromycin and amoxicillin or ampicillin during conservative management of preterm PROM remote from term. The combination of erythromycin and extended spectrum ampicillin– clavulonic acid in a lower risk population near term does not appear to be beneficial and may be harmful (7, 12). This latter regimen is not recommended. Intrapartum group B streptococcus prophylaxis should be given to carriers, regardless of prior therapy.

It is well established that administration of antenatal corticosteroids to women at high risk of delivering an immature preterm infant is one of the most effective obstetric interventions directed to

reduce perinatal morbidity and mortality. The current NIH consensus conference recommendations regarding corticosteroid administration in the setting of preterm PROM are that a single course of betamethasone (12 mg IM, two doses every 24 hours) or dexamethasone (6 mg IM, four doses every 12 hours) be given during conservative management of preterm PROM before 30–32 weeks' gestation because of the potential for reduction of intraventricular hemorrhage (10).

There continues to be discussion regarding the efficacy of corticosteroids in the setting of preterm PROM in the prevention of RDS. It has been suggested that antenatal corticosteroids may be inappropriate because: 1) women with preterm PROM will deliver too quickly to accrue the potential benefits; 2) preterm PROM itself might induce fetal pulmonary maturation, thereby making corticosteroids unnecessary; and 3) antenatal corticosteroids might delay the diagnosis or increase the risk of perinatal infection through their immunosuppressive effects.

There are inadequate data upon which to make firm recommendations regarding tocolytic therapy in the setting of preterm PROM. It is plausible that short-term pregnancy prolongation with prophylactic tocolysis could enhance the potential for corticosteroid effect and allow more time for antibiotics to act against subclinical decidual infection.

Women presenting with PROM before viability should be counseled regarding the impact of immediate delivery and the potential risks and benefits of conservative management.

Home management of carefully selected patients with PPROM is an option that has comparable maternal and neonatal outcomes to the traditional hospital management (11).

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Long obstetric commentary

CORRELATES OF HIV POSITIVE MOTHERS CHOICE OF INFANT FEEDING OPTIONS ON THE FIRST POSTNATAL DAY AT ST MARY'S MISSION HOSPITAL (LANGA'TA)

ABSTRACT

Introduction

Ninty percent of HIV among children is due to MTCT. In Kenya we have 100,000 chidren infected yearly. Children under 5 account for 7.7 % of mortality from HIV world wide.

The majority of these children are infected perinatal. About 16% of these transmissions are attributed to breast feeding. Infant feeding options are important to PMTCT of HIV.

With safer infant feeding practise up to 40% of post natal transmission can be prevented. Knowledge of the determinants of infant feeding options will help in formulating ways of reinforcing the safer infant feeding option.

Objective:

The aim of this study was to find out the practice of infant feeding among seropositive mothers in St Mary's Mission Hospital, Nairobi.

Methodology

This was a cross sectional survey done in the post natal wards of St Mary's Mission Hospital. A total of 124 of HIV positive mothers who delivered at the hospital and gave consent for the study were interviewed between March 2006 and October 2006. The interview was done via pretested questionnaire on the first postnatal day. A structured and pre-tested questionnaire was used to collect the information from the respondents on the first postnatal day.

Results

One hundred and twenty four HIV seropositive mothers were interviewed. The mean age of the HIV positive mothers in this study was 28.7 years (SD +/- 4.206 years, range 20-40 years). Most of the mothers,104(83.9%) came to know of their HIV status during this index pregnancy). Counseling for infant feeding options had been conducted for 115(92.7%), the majority had had upto two sessions. The choices opted for were exclusive breast feeding in 63(50.8%), replacement feeding in 58(46.8%) and mixed feeding in 3(2.4%). Majority 53(91.4%) of those who chose replacement feeding cited PMTCT as a reason for their opting for that method, while those who chose exclusive breast feeding did so because of it being socially acceptable.

Women with higher education were 3.5 time more likely choose replacement feeding than women with primary school education ($p = 0.024$). Women who chose replacement feeding were 3.05 times

more likely to have delivered by caserean section compared to those who choose exclusive breast feeding. (p= 0.009). The age, parity, level of acceptance of HIV status and economic status did not have a statistical significance in the choice of the method of infant feeding on the first post natal day. Women who chose replacement feeding anticipated financial difficulty 17 times more than those who chose exclusive breast feeding.

Conclusion

Mode of delivery and foreseeable difficulties has influence in the choice of infant feeding methods. Other socioeconomic statuses do not have statistical significance in influencing the choice. Most mothers knew their status in this pregnancy. There was high acceptance of HIV testing among ANC clients. Women with tertiary education had three fold increased chance of chosing replacement feeds.

INTRODUCTION:

In Africa children under 5 years account for 7.7% of the HIV/AIDS mortality. Ninety percent of the HIV infection among children is from mother to child transmission.

Breast feeding increases infant HIV infection by 14%; and almost all postnatal infant infection is from mother's breast milk. In the absence of intervention the risk of mother to child transmission is (5-10%) during the antenatal period, (10-20%) during labour and (5-20%) with breast feeding for more than 6 months. The overall risk of transmission where breast feeding is for 6 months is (25-35%), when breast feeding is for 18 to 24 months it is (30-45%) (1,2,3).

In Kenya; HIV incidence among children under 5years is 100,000 annually (2, 3, 5). HIV seroprevalence among antenatal women is estimated at (12 %) Kenya, (11.4%) KNH (Ong'ech)(2, 3). Recent study show a decline in sero-prevalence now estimated at 8.7 %(average nation wide).

Sub Saharan Africa bears 75% of HIV/AIDS disease burden (4); estimated 24 million persons. Prevention of mother to child strategies has demonstrated ability to reduce MTCT to 2%. Safe infant feeding practices reduce infection by 40%. To transit from knowledge to practice requires an understanding of factors that influence HIV sero positive mothers' choice of feeding options.

This study reviews factors that influenced choice of infant feeding practice among HIV seropositive mother at St Mary's Mission Hospital Langat'a, Nairobi

LITERATURE REVIEW

QUANTIFYING THE RISK OF HIV TRANSMISSION THROUGH BREASTFEEDING.

According to available data the early 1990's the estimated additional risk of transmission from breastfeeding above that of transmission during pregnancy and delivery among women with established HIV infection was roughly 15% when breast feeding is continued for 2 years or more. (7, 8, 9). More recent data confirm that the cumulative probability of HIV infection at two years of breast feeding was 70% and for formula feeding 36%. The transmission rate attributable to breast feeding was 16.2% at 2 years (10, 11).

In a South African study they found a 4% rise in the risk of HIV transmission with every 6 month of breastfeeding (12).Infant a who receive other feeds and are also breastfed (mixed feeding) are significantly more likely to be infected at 15 months, (36%) more likely to be infected than those on exclusive breast feeding and (25%) compared to the replacement feeding infants (12).

PREVENTION MOTHER TO CHILD TRANSMISSION OF HIV.

The UNAIDS/WHO/UNICEF guidelines for preventing MTCT, are

1. Early access to adequate antenatal care (ANC),
2. Counseling and HIV testing for women and their partners(VCT),
3. ARV during the peri-natal and postnatal period. (AZT and NVP) for the mother and baby.
4. Intrapartum care with the a of preventing MTCT at this stage.
5. Counseling on the best available feeding option for infant to HIV infected mothers.
6. Support HIV infected mothers through safe practice of chosen feeding method.

ARVs therapies are used to prevent vertical transmission of HIV during pregnancy, labour, delivery and postpartum/postnatal periods. Regimens commonly used include AZT and NVP (14). Elective caesarean sections and non-operative vaginal delivery, avoiding episiotomies and rupture of membranes for more than 4 hours before birth are important midwifery practices that reduce intrapartum transmission (15).

Safe infant feeding practices reduce MTCT by 5-20% (8,11). It is clear that although breast feeding, relative to replacement feeding carries an increased risk for MTCT of HIV, this risk is influenced by many factor. They include duration and pattern of breastfeeding, maternal breast health and immunological status (9,16). Exclusive breastfeeding (in the first 4-6 months) carry a significantly lower risk in transmission than mixed feeding.

Replacement feeding however carries an increases the risk to infants' health, it is associated with increased infant mortality from diarrheal diseases and respiratory infections. In a modeling exercise Kuhn et al estimated that when infant mortality rate is greater than about 40/1000 live births, proving formula milk to HIV infected women would result in excess number of the deaths arising from

formula use being approximately the same or greater than the number of the HIV infections that may have been prevented (17, 18, 19). . Exclusive breast feeding is a safer option to mixed feeding. However maternal mastitis, high viral load advanced disease stage increase the risk of MTCT in breastfed infants. When replacement feeding is acceptable, feasible, affordable, sustainable, and safe avoidance of all breastfeeding by HIV infected mothers is recommended (17).

Where safe replacement feeds are deemed not to be possible, breastfeeding must be modified to make it as safe as possible (20, 21 and 22).

Safe breastfeeding practices

a) Avoiding breast feeding from cracked or bleeding nipples.
b) Providing post-natal ARVs prophylaxis to infants.
c) Heat treatment of breast milk (especially with no mixed feeding)
d) Exclusive breastfeeding for the first 4-6 months with rapid weaning.
e) Reducing the period of breastfeeding (at least 3 months of exclusive breast feeding).
f) Proper positioning and latching during breastfeeding.
g) Practicing safe sex during breastfeeding period, .
h) Seeking medical care immediately for breast problems or when the baby has mouth problems (e.g. oral thrush).

BARRIERS TO THE UPTAKE OF REPLACEMENT FEEDING

When replacement feeding is acceptable, feasible, affordable, sustainable and safe, avoidance of all breastfeeding by HIV infected mothers is recommended.

In resources poor setting, poverty lack of clean water and social-cultural factors make replacement feeding difficult. (12, 13, 19). Key factors influencing the uptake of a method are disclosure of HIV sero status and counseling support (23, 24).

Mothers tend to keep their status to themselves. Lack of disclosure to spouse locks out the financial support that is required for replacement feeding. When the feeding strategies is obvious to the community and neighbors, the woman may be subject to judgment and interrogation, Spouse/partner involvement is important for successful intervention, and to a larger extent community involvement (24, 25).

Counseling matters – A study done in Tanzania about the informed choice of infant feeding method by HIV infected women, as recommended by UNAIDS/WHO/UNICEF Guidelines, found that the choices of safe infant feeding was compromised by:

1. Quality of infant feeding counseling given(directive counseling was used)
2. counselors knowledge
3. lack of time to cope with a positive HIV test result,
4. Lack of follow-up support, regardless of the socio-economic status.

The risks and benefits of the options open to HIV infected mothers were complicated for both the counselors and the mothers. Counselors needed additional training in non-directive counseling and infant feeding options to ensure a better quality of advice-giving and support follow up of women at home (26).

STUDY JUSTIFICATION

HIV/AIDS in a growing world wide epidemic seriously affecting women in the reproductive age: with increased risk of mother to child transmission,

MTCT of HIV is increased by breast feeding by (14-16%).Almost all children with HIV are infected through mother to child transmission. Translating this knowledge to practices that support mothers through decision making of safe infant feeding options is key to PMTCT progress. Knowledge of the determinants of infant feeding options will help us in formulating ways of reinforcing the safer infant feeding option.

Infant HIV infection adds to substantial burden to families', communities and health care systems. Feeding options for infants of HIV infected mothers is complex.

This study proposes to review the factors that determine decision on feeding options for infants of HIV infected mothers for we cannot change the habits which we do not know.

HYPOTHESIS:

1. Socioeconomic status influences the feeding options among sero positive mother at St Mary's Mission Hospital Langat'a.

ANTI HYPOTHESIS

1. Socioeconomic status may not influences choice of infant feeding methods.

OBJECTIVES:

BROAD OBJECTIVES:

To review the factors that influences the choice of infant feeding method among seropositive mothers at St. Mary's Hospital Langata.

SPECIFIC OBJECTIVES:

1. To find out the impact of social / economic factors on the choice and practice of infant feeding among seropositive mothers.
2. To find out whether the level of acceptance of the mothers HIV status has an influence on the infant feeding method practiced
3. To access the level of spouse involvement i.e. spouse disclosure of the sero status, psycho-economic support and participation in choosing the preferred method of infant feeding.
4. To determine the quality of counseling received by the mothers about infant feeding options.

METHODS AND MATERIAL

Study Design

This was a cross-sectional survey.

Study population

The study was conducted among sero-positive women in the postnatal wards at the St. Mary's Mission Hospital, Nairobi.

Study Area

This study was carried out at St. Mary's Mission hospital Maternity Unit. This is a relatively new 250 bed catholic hospital situated in Langata division of Nairobi. It opened its doors in the year 2000. The obstetric and gynecology unit is composed of a Monday to Friday antenatal and gynecology clinic, maternity ward, post natal ward and part of the female surgical ward. The hospital has about 750 new mothers booking in the antenatal clinic, 900 deliveries and 260 caesarean sections monthly. All mothers are offered counseling on HIV testing antenatally. The proportion of mothers accepting to be tested for HIV is 85% and 9% of them are seropositive. HIV testing is free of charge for the mother but their partners pay 350/= ksh for the test. Mothers who are seropositive for HIV are encouraged to do a CD4 cell count this costs 500/= ksh. Counseling about mode of delivery and infant feeding option is offered antenatally. Mothers who choose elective ceserian delivery (PMTCT purpoeses), pay

5,000/= ksh theatre fee just as any other patient going for a c/s for any other indication. Infant formular is neither provided nor subsidized by the hospital. Mothers who opt for this method are counseled on the whether this is affordable and sustainable in there economic circumstances.

Inclusion Criteria

All consenting sero-positive postnatal patients that were in the postnatal ward that had been counseled before about infant feeding option.

Exclusion criteria

Those who decline to participate in the study

Very sick patients not able to participate in the study.

Where there is fetal demise.

SAMPLING

Selection of study subject

Using the inclusion and exclusion criteria stated above, study subjects will be obtained from, postnatal wards, and postnatal wards (using convenient sampling). All eligible women during the study period were enrolled.

Sample size

From a recent study, (Ongech KNH 2004) the prevalence of correct practice on breast feeding among HIV positive mothers was 84% while only 16% had mixed feeding. Given a confidence interval of 95% a level of precision of 5%, the minimum sample size for the study is obtained using the following formula.

SAMPLE SIZE CALCULATION

Sample size calculation will be determined by the formula.

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

Where

N= the desired sample size

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

p- Prevalence of HIV among antenatal mothers at St Mary's is 9%

Q =1 – p

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

$$\frac{1.96^2(0.9)\,(1-0.9)}{(0.05)^2}$$

Therefore n=128

METHODOLOGY

Data collection

Postnatal patients in the 2 postnatal-wards who were HIV positive were identified by the principal investigator assisted by the nurses working there. Each patient was taken to a private room where privacy and confidentiality were ensured. The investigator introduced herself to the client and a written consent for inclusion to the study was obtained .Data was collected using questionnaires using both open and structured questions. Only number codes and not patients names were used for identification on the questionnaire.

Pretesting

Piloting of questionnaire was conducted by administering the questionnaire to ten selected patients (those fulfilling the inclusion criteria). The responses were noted and ambiguous and sensitive questions corrected.

Data management and Analysis

The open ended questionnaire was coded for each question depending on the expected response for data entry. All the completed questionnaires were collected from the interviewers and kept by the principal investigators. Strict confidentiality was maintained at all times. Name identifiers and consent form were kept different from the questionnaire.

The data collected was entered into a computer for analysis using the SPSS 11.0 version package. Data was analyzed using Chi Square (II^2) , Fishers exact test and odds ratio.

ETHICAL CONSIDERATION

Permission to carry out the study was sought and approval obtained from Kenyatta National Hospital Ethical Committee as well as the administration of St. Mary's Mission Hospital.

Written consent was sought and obtained from patients in order to participate in the study.

Information obtained was treated with care to maintain utmost confidentiality. Patient's names were entered into the consent form but were kept separate from the questionnaire for purposes of confidentiality.

The information was used only for the intended purpose of the study. The information did not jeopardize the patients' treatment and follow up in any way.

RESULTS

Women that delivered at St Mary's Mission Hospital Langata between October 2005 and October 2006 were 10073 of who 324 were HIV positive. These gives as sero-prevalence rate of 3.2% among the postnatal mothers. 148 mothers who were HIV positive delivered had a cesarean delivery. At total of 8781 new antenatal mothers were seen in the ANC all were counseled on HIV testing, 91.2% accepted to be tested. Among those who were tested 767 were HIV positive giving as a sero-prevalence rate of 9.5% among the ANC attendants.

We sampled 124 HIV infected postnatal mothers with live babies between March and October 2006. They were interviewed and information was collected from all Clients who gave consent. These were entered into a structured questionnaire by the help of a counselor. A number (5) declined giving consent, wanting another HIV test and some questioning the confidentiality of the study. Though it had been well explained to then the intents of the study and that the information obtained would only be used for the purposes of study, just as stated in the structure consent form.

The preferred methods were exclusive breastfeeding for 6 months having 63 (50.8%) and replacement feeding 58(46.8%). Only 3 (2.4%) did prefer mixed feeding. The mixed feeding group was not included in the analysis.

Table 1: Distribution and Preferred feeding option by age, marriage and education.

(n=124)

Characteristic	Distribution N (%)	Exclusive breast feeding (%)	Replacement feeding (%)	P-values			
Age (mean /SD)	*28.17 (+/- 4.2)*	28.40 (+/- 4.4)	28.03 (+/- 4.0)	0.638			
Marital status							
Single	25(*20.2*)	13(*52.2*)	12(*47.8*)	0.994			
Married	99(*79.8*)	50(*52*)	46(*48*)				
Type of marriage							
Monogamous	86(*83.8*)	43(*53.7*)	37(*46.3*)	0.324			
Polygamous	16(*16.2*)	7(*43.7*)	9(*56.3*)				
Education				**OR**	**95%(CI)**	**P value**	0.028
Primary	30(*24.2*)	19(*67.8*)	9(*32.2*)	ref	95%	-	
Secondary	53(*42.7*)	30(*56.6*)	23(*43.4*)	1.393	0.543-3.577	0.491	
Tertiary	41(*33.1*)	14(*35*)	26(*65*)	3.538	1.292-9.631	0.014	

Table 1 shows the distribution by age, marriage and education, it also shows analysis of the same with regard to the preferred feeding options. The mean age of the study population was 28.17 years (SD+/-4yrs); 99(79.8%) were married. Majority of the married were in monogamous (83.8%) marriages. The population was reasonably educated with 75.8% having at least completed secondary school. Education had an association of statistical significance (p, 0.028) with infant feeding method chosen.Those who had tertiary education had 3.54 fold increased uptake of replacement feeding than those who had primary education while those who had secondary education were 1.39 times more likely to chose replacement feeds .The age did not have any statistical significance (p 0.638,)in determining the choice with, neither did marriage (p 0.994) nor type of marriage (p 0.324).

N/B *The number in the analysis for replacement and exclusive is 121 since 3 patients had mixed feeds and are not included in the analysis.The marital status was pooled into two to enable the analyisis since there would be only one in each cell for the separated group that were included in those who are single.

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Table 2: Distribution and preferred feeding option by socioeconomic status.

(n=124)

Characteristic	Distribution N (%)	Exclusive breastfeeding (%)	Replacement feeding (%)	p-value
Employment status				
Employed	45(*36.3*)	23(*51*)	22(*49*)	0.510
Non employed	79(*67.7*)	40(*51*)	36(*49*)	
Income				
Less than 5000	34(*23.4*)	19(*61.3*)	12(*38.7*)	0.311
More than 5000	90(*76.6*)	44(*48.9*)	46(*51.1*)	
Housing				
Permanent	92(*74.2*)	47(*51.0*)	45(*49*)	0.433
Semi permanent	32(*25.8*)	16(*55.1*)	13(*44.9*)	
Permanent; rooms				
Single room	27(*29.3*)	16(*59.2*)	11(*40.8*)	0.426
One bedroom	45(*48.0*)	23(*51.1*)	22(*48.9*)	
Two and above	20(*21.7*)	8(*40*)	12(*60*)	
Water source				
Reliable tap water	108(*87.1*)	55(*52.3*)	50(*47.7*)	0.577
Other sources	16(*12.9*)	8(*50*)	8(*50*)	

Table 2 depicts the socio-economic characteristics of the study population. Forty five (36.3%) of the mothers were employed while 79(67.7%) were not-employed. More than half of those not employed were house wives. Most of the respondents 92(74.2%) lived in permanent houses; 45(48%) of those in permanent houses were single rooms; ninety 90(76.6%) of the correspondents earned more than 5000 shillings per month. Fifty two (41.9%) spent less than 500 shillings on water, 35(28.2%) got water free of charge and 23(18.5%) paid between 500 and 1000 shillings per month fourteen (11.3%) paid more than 1000 shillings per month.

These socioeconomic characteristics had no association of statistical significance with the infant feeding method chosen; employment (p 0.510), income (p, 0.311) type of housing (p, 0.433), number of rooms in the permanent house (p, 0.426) while source of water (p, 0.577)..

*Eighty six (69.4%) spent more than 500 shillings on energy, sixty six 53.2% used paraffin, charcoal and gas for cooking (combination of fuel source), 26(21%) used paraffin alone, 5(4%) used charcoal alone 27(21.8%) used gas alone.

Table 3: Distribution and preferred feeding option by obstetric characteristics.

Characteristic	Distribution N (%)	Exclusive breastfeeding (%)	Replacement feeding (%)	P – values +/- OR
Parity (Mean SD)	1.97 (+/- 0.95)	1.32 (+/-0.47)	1.36 (+/-0.48)	0.609
Mode of delivery *Caesarean* *Vaginal*	 54(*43.5)* 70(*56.5*)	 20(*37*) 43(*64.2*)	 34(*63*) 24(*35.8*)	 0.009 OR 3.05 95%(1.36-6.89)
Birth weight (Mean SD)	3.21 (+/-0.47)	3.23 (+/-0.43)	3.195 (+/- 0.52)	0.638

Table 3 shows the obstetric characteristics of the study population and the way this affected the choice of infant feeding

The mean parity of the study population was 1.97(SD+/- 0.95) Majority of the women were Para 2+0; 49(39.5%), while Para 1+0 (1st time mother) were 44(35.5%) so those with parity of one and two combined were 93(75%).25(20.2%) were para 3, four (3.2%) were para 4 while parities five and six had one (0.8%) each. Mean party was 1.97+/-0.95 in general, of the mothers who chose exclusive, their mean parity was 1.32 +/- 0.47, while those who chose replacement had 1.36+/- 0.48 as their mean parity.in terms of the influence on the choice , p value of 0.609 was found using the analysis of variance, showing that it did not significantly influence the choice statistically. Most of these pregnancies were at term at the time of delivery 99% and the mean birth weight was 3.21 (SD 0.47) majority (62.9%) had birth weight of more than 3 kilograms. It did not statiscally have any power in influencing the choice with p value of 0.638 calculate from analysis of variance

Mothers opting for replacement feding were 3 times more likely to have been delivered by cesarean section compared with exclusive breast feeding.

Table 4: **Distribution and preferred feeding option by timing of HIV testing and outcome.**

Characteristic	Distribution N (%)	Exclusive breastfeeding (%)	Replacement feeding (%)	P -values
Time of testing				
During this pregnancy	104(*83.9*)	54(*53.5*)	47(*46.5*)	0.327
Before this pregnancy	20(*16.1*)	9(*45*)	11(*55*)	
Reaction to status				
Accepted	97(*78.2*)	51(*53.7*)	44(*46.3*)	0.323
Other reactions	27(*21.8*)	12(*46.2*)	14(*53.8*)	
Disclosed to spouse				
Yes	83(*66.9*)	38(*46.9*)	43(*53.1*)	0.193
No	41(*33.1*)	25(*62.5*)	15(*37.5*)	
ARV prophylaxis*				
Started	25(*20.2*)	15(*60*)	10(*40*)	0.158
Not started	99(*79.8*)	48(*50*)	48(*50*)	

*It should noted that all clients in this study received intrapartum nevirapine, so the ARV prophylaxis refers to any other regime received antenatally.

Table 4 shows the timing and outcome of HIV testing in the mothers who responded and how these affected the choice of feeding options.

The HIV status of most (83.9%) of the women was known during this index pregnancy, while only 16.1% knew their status before this pregnancy. Most (85.5%) of them had accepted their status while a few 11.5% were annoyed: 4 % wanted another test. Eighty (64.5%) patients had done CD4 cells counts, of these 16(12.9%) had less than 200, 63(50.8%) had between 200 and 300 and one had more than 300. Only 25(20.2%) were started on treatment, Prophylaxis ARV other than intrapartum niverapine.

The time of testing had no association of statistical significance with the option of feeds (p, 0.327), neither did their reaction to the result (p, 0.323), disclosure to spouse(p, 0.193) nor being on antiretroviral therapy (p, 0.158)

N/B Majority, 81(70%) of the partners were aware of the client's status. Partners who knew of their own status were 44(40%), and of these 25(56%) were HIV seropositive. All the patients were in stage I WHO clinical classification

Table 5. Distribution and Preferred option by knowledge of options

Characteristic	Distribution N (%)	Exclusive breastfeeding (%)	Replacement feeding (%)	P-values
Knows benefit of replacement				
Knows	121(*97.5*)	61(*51.2*)	58(*48.8*)	0.554
Does not know	3(*2.5*)	2(*100*)	0(*0*)	
Knowledge of benefits exclusive breastfeeding				
Knows	120(*96.8*)	60(*51.3*)	57(*48.7*)	0.322
Does not know	4(*3.2*)	3(*75*)	1(*25*)	
Counselled on options				
Yes	115(*92.7*)	55(*47.8*)	60(*52.2*)	0.526
No	9(*7.3*)	3(*42.9*)	4(*57.1*)	
Number of counselling sessions				
One and 2	65(*56.5*)	33(*51.6*)	31(*48.4*)	0.427
Three and more	50(*43.5*)	27(*55.1*)	22(*44.9*)	

Table 5 shows the distribution by knowledge of options and how they affected the choice of the feeding options. Majority (97.5%) of the mothers knew that having replacement feeding would prevent mother to child transmission of HIV, and 96.8% knew the exclusive breast feeding was good for the baby. 92.7% were counseled, of these majority (56.5%) had one or two sessions. Whether one knows the benefit of exclusive or replacement feeds in general did not have any statistical significance in determination of the choice of feeds with (p, 0.322 and 0.554) respectively, neither did being counselled nor the number of counselling sessions with (p, 0.526 and 0.427) respectively.

Table 6: Preferred feeding option by reason of preference, foreseeable difficulties

Characteristics	Distribution N (%)	Exclusive breastfeeding (%)	Replacement feeding (%)	P-values		
Reason of prefference						
PMTCT	71(*58.7*)	18(28.6)	53(91.4)	<0.005		
Others	53(*41.7*)	45(71.4)	5 (8.6)			
Foreseeable difficulty				OR 95% CI	P value	0.009
Financial	16(*12.9*)	1	14	17.195	0.007	
Social	*28(22.6)*	19	8	0.58	0.244	
None	*80(64.5)*	43	36	1	-	

Table 6 demonstrates some of the reasons for preference and foreseeable difficulties and how they affected the choice of feeding of the client.

Prevention of MTCT of HIV was the most, 71(58.7%) cited answer for reason of prefered infant feeding method. Other reasons given were social acceptability 22(17.7%), breast milk is best 7(5.6%) and bonding at 24(19.4%). Among the mothers who mentioned PMTCT as a reason for their preferred method, a big majority (53) 91.4% chose replacement feeding method. There was a statistical association between those who exclusively breast fed ($p < 0.005$).

The foreseeable difficulties mentioned were financial (12.9%), social reasons (22.6) and 64.5% mothers had no foreseeable difficulties. Taking the later to be the reffering point, the OR for chosing replacement were 17.0 for financial reasons and 0.538 for social reasons with p values of 0.007 and 0.244 respectively , the general p value for the group was 0.009 showing that the forseable difficulty had an influence of statistical significance with the infant feeding method the mother opted for. Mothers who chose replacement feeding were 17 times more anticipating financial difficulties as compared with those who opted for exclusive breast feeding.

DISCUSION

HIV infection is an important public health problem in Kenya as outlined in the session paper number 4 of 1997(2,3). The transmission rates are as high as 35% where there is no intervention and below 5% when appropriate care is available (4, 6). In Kenya 10% of reported AIDS cases are in children under 5 years, out of which 90% was due to mother to child transmission. The estimated number of children infected with HIV per annum is 100,000 in Kenya (2).

Transmission from an infected mother to their babies can occur during antenatal period (10-20%), labour and delivery (35-50%), and breast feeding (40-50%). Mother to child transmission can be greatly reduced by expanding high quality antenatal and obstetric care, voluntary counseling and testing (VCT), elective caesarean delivery, and antiretroviral drug therapy, use of replacement feeding or exclusive breastfeeding and modification of routine care during labour and delivery(1, 2,4).

According to KDHS (2003) 88% of pregnant women attend antenatal care in Kenya. But only 50% deliver in hospital (2). The national sero prevalence rate among antenatal mothers is 8.7%. In this study, acceptance rate to HIV testing was 98% while the sero-prevalence rates among the antenatal mothers was 8.9% which compares with the above national figures.The seropravalence rate among postnatal mothers in this study was much lower (3.5%) compared to the general population.This is due to having some unbooked mothers delivering at the institution and the lack of routine intrapartum HIV testing during the study period.

The study population was self selected and not a community sample thus the results may not be generalized. In this study 79.8% were in stable marriages and 76% had education upto secondary years or above.This compares well with Muliro found: 90% of antenatal mothers were married.Though according to KDHS 2003; married women were of increased risk of being HIV positive compared to the single counterparts. Majority were not in formal employment 67.7% and they could be classified as upper low class, majority of the families living on 5,000 Ksh per month (76%) and living in permanent housing (74.2% with 48% being in one bed roomed house). The mean age of our respondants was 28.17 (SD 4.2) years, the peak prevalenceof HIV among Kenyan women was at age 25- 29(KDHS 2003), the findings in our study lie within that age group.

Majority 104(83.9%) learnt of their HIV status during this index pregnancy. This compares well with 89.9% of women who knew their HIV status in index pregnancy (Waweru KNH 2004). In 83(66.9%) of the patients their spouses were aware of their status, which is higher than what Waweru found at KNH (53.2%) (27).

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Correct infant feeding choice in the first postnatal day was done by 97.6% of the women (correct infant feeding to all methods other than mixed feeding) . Only 2.4 % practised mixed feeding. This compares well with what was found by Osoti in Kijabe (2004) where 98% of the postnatal mothers had correct practice on infant feeding(There seems to be an upward trend as far as knowledge attitude and practice on prevention of mother to child transmission of HIV. Earlier studies showed lower figures on correct infant feeding knowledge, Ongech KNH 2002 (88%), Amoth Aga-Khan 2000 (75%) (27, 28).

Most (91.4%) mothers chose replacement feeding for PMTCT. All the mothers who cited social acceptability as the reason for their choice preferred exclusive breast feeding. This may be related to the way the community at large views people living with HIV. This suggests that if stigma is dealt with at the community level more mothers would accept replacement feeding taking account of other factors. Mothers who chose replacement feeding more often anticipated financial difficulties.according to WHO infant feeding recommendation replacement feeding should be feasible , accessible , available and sustainable. In this regard, this study shows the concern of the mothers that replacement feeding may not be sustainable within their means.

In this study the practise of replacement feeding was 46.8%, which is higher than that found by UNICEF/UNAIDS pilot project for Africa in Kenya (29) which found 33% acceptance of replacement feeding. The figures on acceptance and practise of correct feeding methods are higher but question on adherence to the method chosen are beyond the scope of this study. This may be related to the level of education being of the population in the project being lower than that of this study

Just over half of the study population had vaginal deliveries 70(56.5%), while 54(43.5%) had a caesarean delivery this includes both elective and emergency caesarean sections. A study done in the same institution showed that 34% of the HIV positive mothers deliver by elective caesarean section for PMTCT purposes. A study by Waweru KNH 2004 found the acceptability of elective caesarean delivery among sero-positive mothers to be (52%) which could be much higher than the real figures in this study population. In that study Waweru also found that of all those who did replacement feeding (55.7%) delivered by caesarean section. This compares with this study, where by women who delivered by caesarean section were 3.05 times more likely to do replacement feeding than those whop delivered vaginally (OR=3.05)

Most, 80 (64.5%), of the women had CD4 cell count levels done, with 63 (50.8%) having levels 200-300. A great majority 99(80%) were not on prophylactic ARVs. All the mothers received intrapartum Nevirapine except those on HAART who continued with their usual treatment. Antenatal HIV testing is funded therefore free to the mother. Whereas they have to pay 500/= Ksh to have a CD4 cell count. This could explain why some of the mother did not take the test.

There was no significant statistical association between the feeding method chosen and duration of mother knowing her status, whether she has accepted the seropositive status and whether she is on ARVs or not ($p<0.05$). While it is obvious that human beings are more likely to be receptive to change from the norm after being given time to accept their status this study did not find this.

CONCLUSIONS.

1. Women with tertiary education had three fold increase in opting for replacement feeding compared to those with primary scholl education.
2. Women who chose replacement feeding were more likely to have been delivered by caeserian section.
3. There was high level of acceptance for HIV testing among the population in this study as well as high level of disclosure to spouse.
4. Majority of the patients (83%) learnt of there HIV status in this index pregnancy and had come to accept their status.

RECOMMENDATIONS

- There should be increased access to universal HIV testing in pregnancy because the uptake is high and most women are first tested in pregnancy.

- St Mary's Hospital should increase access to ARV for those with low CD4 counts.

- Kenya should aim at improving socio-economic and education status of women which is likely to have profound positive impact in prevention of mother to child transmission of HIV.

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APPENDIX I
Common toxicity criteria proposed by National Cancer Institute (Britain).

Grade					
	0	1	2	3	4
Acute adverse event					
Nausea anorexia	None	Able to eat	Oral intake significantly reduced	No significant intake, requiring i.v fluids	Requiring feeding tube
Diarrhea	None	Increase of <4 stools/day over pretreatment	Increase of 4-6 stools/day or nocturnal stools	Increase of >7 stools/day or incontinence, or need for parenteral support for dehydration	Physiologic consequences requiring intensive care haemodynamic collapse
Dysuria	None	Mild symptoms requiring no intervention	Symptoms relieved with therapy	Symptoms not relieved despite therapy	-
Urinary urgency	Normal	Increase in frequency or nocturia up to 2x normal	Increase >2x normal bur <hourly	Hourly or more with urgency or requiring catheter	-
Leucocytosis(total WBC)	WNL	<LLN-3.0 X IO9/L	>2.0-<3.0x109/L	>1.0 -<2.0 x109/L	Grade
Late adverse events					
Lymphatic	Normal	Mild lymphedema	Moderate lymphedema requiring compression; lymphocyst	Severe lymphedema limiting function: lymphocyst requiring surgery	Severe lymphedema limiting function and ulceration
Diarrhea (periodic)	None	Increase of <4 stools/day over pretreatment	Increase of 4-6 stools/day or nocturnal stools	Increase of >7 stools/day or incontinence or need for parenteral support	Physiologic consequences requiring intensive care: haemodynamic collapse

				for dehydration	
leus	None	-	Intermittent not requiring intervention	Requiring non-surgical intervention	Requiring surgery
Urinary retention	Normal	Hesitancy or dribbling but not significant residual urine retention occurring during immediate post operative	Hesitancy requiring medication or occasional in/ put catherization (<4 x per week or operative bladder atony requiring indwelling catheter beyond immediate post operative period	Requiring frequent in/out catheterization (>4x per week) or urologic intervention	Bladder rupture
Urinary incontinence	None	With coughing or sneezing	Spontaneous some control		

WNL Within normal limit, LNL Lower normal limit, WBC White blood cell count.

APPENDIX II

questionnaires for long gynaecologic commentary

1. Serial number .....
2. Patients' number. .....
3. Age of patient at opening file ..... (In years.) Date of birth ☐☐

   Date file opened (In radiotherapy department) ☐☐
4. Marital status

   a) Married ☐ b) Single ☐ c) Divorced/ Separated ☐ d) unknown ☐
5. Level of education

   a) No formal education ☐ b)Primary level ☐ c)Secondary level ☐

   d) Collage/ University ☐ e)Unknown ☐
6. Occupation of patient partner

   a) Unemployed ☐ ☐

   b) Casual worker ☐ ☐

   c) Employed professional ☐ ☐

   d) Businesswoman ☐ ☐

   e) Farmer ☐ ☐

   f) Unknown ☐ ☐
7. Resident of patient.....
8. Home district. .....
9. Referred to KNH a) Yes ☐ b) No ☐ c) Unknown ☐

   If answer to above is yes a) Referring institution. .....

   b) Referring personnel 1) doctor ☐ 2) Clinical officer ☐

   3) Nurse ☐ 4) Unknown ☐

Risk Factors

10. Type of marriage. a) Polygamous ☐ b) Monogamous ☐
11. Alcohol intake cigarette smoking

    a) Yes ☐ a) Yes ☐

    b) No ☐ b)No ☐

12. History of contraceptive use

a) Injectable ☐ b) Oral contraceptive ☐ c) Norplant/Jadelle ☐

d) IUCD(Intrauterine contraceptive devise) ☐

e) No history of contraceptive use ☐ f) Unknown ☐

13. Age at menarche. .....If unknown tick here ☐

14. Postmenopausal A) Yes ☐ b) No ☐

If yes, number of years postmenopausal ☐

15. Age at first sexual contact.....If missing tick here ☐

16. Parity. ☐ + ☐

Still births. ☐

Abortions. ☐

Term live babies. ☐

17. Age of first child. ☐

18. Number of years since last delivery or pregnancy ☐

19. History of STD a) Yes ☐ b) No ☐ c) unknown ☐

20. Lifetime sexual partners ☐ ☐

21. Ever used of condom a) Yes ☐ b) No ☐

22. Pap smear ever done a) Yes ☐ b) No ☐

23. HIV testing done a) Positive ☐ b) Negative ☐ c) Unknown ☐

Treatment and related toxicity

24. Time lapse between the various stages of management.

Duration between		**Indicate duration in months or date**	**Tick here if unknown**	**Time difference (fill at analysis)**
a	Duration of symptoms at first presentation			
b	Date at presentation to KNH			
c	EUA date			
d	Histological diagnosis date			

	when written by pathology department			
e	Date of treatment initiation 1)Surgery 2) Chemotherapy 3) Radiotherapy			

25. Symptom at presentation and there duration at time of presentation in KNH.

Symptoms		**Indicate duration in months**
a	Smelly vaginal discharge	
b	Abnormal vaginal bleeding	
c	Pelvic pain	

26. Earliest taken HB (Hemoglobin level) ☐ Date when done ☐

27. Histological types and grade of differentiation. (tick where it applies)

stage	**tick**	**Histological type**	**tick**	**Grade of differentiation**	**tick**
IA		Squamous cell carcinoma		Well differentiated	
IB		Adenocarcinoma		Moderately differentiated	
IIA		Others specify……..		Poorly differentiated	
IIB				Not indicated	
IIIA					
IIIB					
IVA					
IVB					

28. Date of therapy initiation. (Indicate on which it applies to the patient)

a) Radiotherapy ☐ b) date of surgery ☐ ☐

29. Radiotherapy

No of session prescribed	No of sessions completed	Date of last session

c). surgery

i) TAH ☐ ii) Extended TAH ☐ iii) Wertheim's hysterectomy ☐

d). Incomplete treatment a) Yes ☐ b) No ☐

If yes reason for incomplete treatment .....if not indicated tick here ☐

30. Complains on follow-up (period after treatment)

	3 months	6 months	1 year	18 months	2 years
Skin burns					
Sin fibrosis					
Diarrhea/ colitis					
Stool incontinence/ RVF					
Cystitis, dysuria, urgency					
Urinary incontinence/ VVF					
Hematological complication leucocytosis					
Lymphedema					
Evidence of residual tumor					
Evidence of recurrent tumor					
Death of patient					

31. Blood transfusion

a) Before treatment initiation ☐ b) During treatment period ☐

☐

c) After completion of treatment ☐ d) No transfusion done.

32. Evidence of recurrence (9 months after therapy) a) Yes ☐ b) No ☐

c) Has residual tumour (evidence of tumour immediate after therapy) ☐

33. Specify mode of diagnosis of recurrence. a) CT Scan ☐ b) Pelvic ultrasound ☐

Clinical examination ☐

34. Is the patient dead a) Yes ☐ b) No ☐ c) Unknown ☐

35. If yes to above date of death ☐☐☐

36. If answer question 34 is no or unknown when was the date of last visit ☐☐☐

APPENDIX III

QUESTIONAIRE FOR LONG OBSTETRIC COMMENTARY
SECTION 1

1. STUDY NO ☐

2. HOSPITAL NO ☐

3. AGE OF CLIENT ☐

4. CLIENTS MARITAL STATUS :

   A) SINGLE ☐

   B) MARRIED ☐

   C) WIDOWED ☐

   D) SEPARATED ☐

5. WHAT KIND OF A MARRIAGE ARE YOU IN.

   A) MONOGAMOUS ☐

   B) POLYGAMOUS ☐

6. PARITY ☐ ☐

   SPECIFY THE NUMBER OF A) LIVING CHILDREN ☐

   B) ECTOPIC PREGNANCIES ☐

   C) ABORTIONS ☐

   D) NND (NEONATAL DEATHS) ☐

   E) EARLY INFANT DEATHS ☐

7. EVER USED FAMILY PLANNING METHOD

   A) ORAL CONTRACEPTION ☐

B) DMPA (DEPOT) ☐

C) IUCD ☐

E) NORPLANT/ JADELLE ☐

F) BARRIER (CONDOM) ☐

G) NATURAL FAMILY PLANNING ☐

F) OTHERS SPECIFY. .....

8. AT WHAT GESTATION WERE YOU WHEN YOU HAD THIS BABY?

.....

9. WHEN WAS YOUR LAST MENSTRUAL PERIOD (LMP?) ☐

10. WHAT IS YOUR BABY'S BIRTH WEIGHT? ☐

11. WHAT WAS THE MODE OF DELIVERY?

A) CAESARIAN SECTION ☐

B) VACUUM ☐

C) VAGINAL ☐

D) EPISIOTOMY ☐

12. DID YOU HAVE ANY COMPLICATION DURING ANTENATAL?

PERIOD OR AT DELIVERY? YES ☐ NO ☐

SPECIFY (IF YES).....

13. CLIENT PROFFERED METHOD OF INFANT FEEDING

a) EXCLUSIVE BREASTFEEDING FOR 3 MONTHS OR LESS ☐

b) EXCLUSIVE BREASTFEEDING FOR 3 TO 6 MONTHS ☐

c) REPLACEMENT FEEDING (FORMULA) ☐

☐

d) MIXED FEEDING

e) OTHERS (SPECIFY)

14. WHAT IS/ARE YOUR REASONS FOR OPTING FOR THIS?

METHOD? A).....

B).....

15. ANY FORESEEABLE DIFFICULTIES. .....

16. WHAT WAS YOUR GREATEST CHALLENGE TOWARDS ACCOMPLISHING THE METHOD OF CHOICE YOU HAVE OPTED FOR.?

.....

.....

17. DID YOU RECEIVE ANY FORM OF COUNSELING BEFORE CHOOSING THE ABOVE METHOD?

YES

NO

18. IF YES TO 39 HOW MANY SESSIONS DID YOU HAVE ?

A) TWO AND LESS

B) TWO TO FIVE

19. ARE YOU AWARE OF THE BENEFITS OF REPLACEMENT FEEDING?

AND COULD YOU MENTION TWO IF YES

A).....

B).....

20. ARE YOU AWARE OF THE BENEFITS OF EXCLUSIVE BREAST FEEDING? IF YES MENTION TWO.

A).....

B).....

21. WHAT MADE YOU CHOOSE THE METHOD YOU ARE PRACTICING?

.....

.....

SECTION II

22. OCCUPATION .....

23. YEARS OF EDUCATION ☐

24. TYPE OF HOUSING:

A) SEMI- PERMANENT ☐

B) PERMANENT ☐

I) SINGLE ROOM ☐

II) ONE BEDROOM ☐

III) ONE BED ROOMED ☐

25. SOURCE OF WATER FOR HOME USE:

A) RELIABLE TAP WATER ☐

B) FROM WATER VENDORS ☐

c) OTHERS SPECIFY ☐

26. WHAT IS THE COST WATER (IN SHILLINGS PER MONTH?) ☐

27. SOURCE OF ENERGY (FOR COOKING):

a. GAS ☐
b. CHARCOAL ☐
c. PARAFFIN ☐

d. OTHERS ☐

28. INCOMES PER MONTH ☐

29. COST OF ENERGY ☐

30. NUMBER OF DEPENDANTS ☐

SECTION III

31. DATE FIRST TESTED POSITIVE FOR HIV ☐

32. HAVE YOU EXPERIENCED ANY DIFFICULTIES SINCE LEARNING OF YOUR SERA STATE?

YES ☐ NO ☐

IF YES SPECIFY.....

33. WHAT DO YOU THINK ABOUT IT .....

.....

34. HOW LONG HAVE YOU BEEN MARRIED/ PARTNERSHIP (IF FOR MORE THAN ONE YEAR MONTHS NOT APPLICABLE?)

YEAR'S ☐ MONTHS ☐

35. DO YOU SHARE A HOUSE WITH YOUR PARTNER?

YES ☐ NO ☐

36. DOES YOUR PARTNER KNOW OF YOUR SERA STATUS?

YES ☐ NO ☐

37. YES BEING THE ANSWER TO THE ABOVE QUESTION, HOW DID HE COME TO KNOW OF YOUR STATUS

.....

38. WHAT WAS HIS REACTION ON LEARNING OF YOUR SEROSTATUS?

.....

39. DOES YOUR PARTNER KNOW HIS SEROSTATUS?

YES ☐

NO ☐

YES

NO ☐

40. IF THE ANSWER TO THE ABOVE QUESTION IS YES, WHAT IS HIS SEROSTATUS NEGATIVE? ☐

POSITIVE ☐

41. WHAT IS THE OCCUPATION OF YOUR PARTNER……..

……

42. DOES YOUR PARTNER

A) TAKE ALCOHOL

YES ☐ NO ☐

B) SMOKE CIGARETTE

YES.☐ . NO ☐

43. DO YOU BELONG TO ANY COMMUNITY SUPPORT PROGRAM (ANY RED RIBBON SUPPORT GROUP) YES ☐

NO ☐

44. HAVE YOU DISCLOSED YOUR STATUS TO ANY OF YOUR NEIGHBOR, FRIEND OR RELATIVE?

YES ☐

NO ☐

IF YES SPECIFY…….

SECTION IV

TO BE FILLED IN BY THE COUNSELOR

1. DOES THE MOTHER HAVE ANY OF THE HIV RELATED ILLNESS? (TICK THE ONES THE CLIENT HAS)

A) CANDIDIASIS ☐

B) INFECTIVE DIARRHOEA ☐

C) HERPIS ZOSTER ☐

☐

G) PNEUMOCYSTIS CARINII PNEUMONIA

H) CRYPTICOCAL MENENGITIS ☐

I) UNEXPLAINED FEVERS ☐

J) RECURRENT UPPER RESPIRATORY TRACT INFECTION. ☐

K) NONE OF THE ABOVE ☐

L) OTHERS SPECIFY……..

2. HOW WOULD THE PATIENT BE RATED ON THE PERFORMANCE SCALE

A) ASYMPTOMATIC NORMAL ACTIVITY. ☐

B) SYMPTOMATIC WITH NORMAL ACTIVI… ☐

C) BED-RIDDEN BUT FOR <50% OF THE DAYS OF THE LAST MONTH. ☐

D) BED-RIDDEN BUT FOR >50% OF THE DAYS OF THE LAST MONTH ☐

3. HAS THE MOTHER ACCEPTED HER HIV STATUS

YES ☐

NO ☐

4. WHAT IS HER CD4 CELL COUNT? ☐

5. WHAT DRUGS IS THE MOTHER ON (SPECIFY THE ARVS OR OTHER TREATMENT FOR OPPORTUNISTIC INFECTION). ……..

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.
Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi.
Email: KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/ 01/ 3793 **29th September 2006**

Dr. Lydia Okutoyi
Dept. of Obstetrics & Gynaecology
Faculty of Medicine
University of Nairobi

Dear Dr. Okutoyi

RESEARCH PROPOSAL: "TREATMENT OUTCOME OF INVASIVE CANCER OF THE CERVIX AT KENYATTA N. HO SPITAL" (P131/06/2006)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** revised version of your above cited research proposal for the period 29th September 2006 – 28th September 2007.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely

PROF A N GUANTAI
SECRETARY, KNH-ERC

c.c. Prof. K.M.Bhatt, Chairperson, KNH-ERC
The Deputy Director CS, KNH
The Dean, Faculty of Medicine, UON
The Chairman, Dept. of Obs/Gynae, UON
The Head, Medical Records, KNH
The Chairman, Dept. of Diagnostic Radiology, UON
Head of Department, Cancer Treatment Centre
Supervisors: Dr.F.X.O Odawa, Dept. of Obs/Gynae, UON
Dr. Nelly Mugo, Obs/Gynae, KNH
Dr. Catherine N. Nyongesa, Radiotherapy Dept, KNH

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Fax: 725272
Telegrams: "MEDSUP", Nairobi.
Email: **KNHplan@Ken.Healthnet.org**

Ref: KNH-ERC/ 01/ 3397 **Date: 29th March 2006**

Dr. Lydia Okutoyi
Dept. of Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr. Okutoyi

RESEARCH PROPOSAL: "THE PRACTICE OF INFANT FEEDING OPTIONS AMONG SEROPOSITIVE MOTHERS AT ST. MARY'S MISSION HOSPITAL(LANGATA) " (P2/01/2006)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** revised version of your above cited research proposal for the period 29th March 2006 – 28th March 2007.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

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The HOD, Medical Records, KNH
Supervisors: Dr. Nelly Mugo, Obs/Gynae, KNH
Dr. F.X.O. Odawa, Obs/Gynae, UON

CONSENT FORM

Title of the study;

The practice of infant feeding among seropositive mothers.

Investigators. *Principal;* **Dr. Lydia Okutoyi, university of Nairobi,**
Department of Obstetrics and gynaecology.
P.O. Box 19676 Nairobi Tel +254721814381
Supervisors; 1. Dr. Nelly Mugo. 2. Dr. Odawa F. X. O.
Department of obst. and Gyn. University of Nairobi

Background and Purpose of study

Mother to child transmission of HIV infection remains a major public health problem worldwide. The transmission rate is as high as 35% when there is no intervention and below 2% where anti retroviral treatment; elective C/S and abstinence from breast milk is offered to the infected mothers and their infants. We know a lot about the best feeding option to prevent HIV transmission from mother to child. Mixing breast milk and formula is high risk, however this practice is still common. With safer infant feeding methods up to 40% of postnatal transmission can be prevented.

Procedure. In this study, we will get information from mothers using a formulated questioner. This will be done in the post-natal wards and a later follow-up in the post-natal clinics. We will be able to find out factors affecting the choice of practice (infant feeding), and whether mothers stick to the method they choose. All mothers who accept will be recruited into the study.

Voluntary participation.

Participation in the study is on voluntary basis. You can terminate your participation at any time with no consequences whatsoever. Participation in the study does not entail any financial benefits.

Confidentiality

Information obtained will be treated with care to maintain utmost confidentiality. Patients' names will not be entered on the questionnaires; only code numbers will be used for purposes of confidentiality. Consent forms will be kept separate from the questioners. The information will be used only for the intended purpose of the study. The information will not jeopardize the patients' treatment and follow up in any way.

Benefits. There are no financial benefits in participating in this study. However mothers will benefit by receiving counseling on the prevention of transmission of HIV from mother to child and also counseling on how to cope with the HIV status.

Risks or disadvantages

We will do everything we can to maintain confidentiality, however there is a risk of some else knowing your status. Knowing your status can be upsetting.

I confirm that I have been informed about the study and fully understand my rights of withdrawing at any time. I give my informed consent of my free will.

Patient's signature. ..... Date. .....

Patients name.....

Witness signature. (Counselor) ..... Date. .....

CONSENT FORM

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The practice of infant feeding among seropositive mothers.

Investigators. *Principal;* **Dr. Lydia Okutoyi, university of Nairobi,**
Department of Obstetrics and gynaecology.
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Patients name.....
Witness signature. (Counselor) ..... Date. .....