GLUCOSE INTOLERANCE AND ASSOCIATED FACTORS AMONG ANTENATAL CLIENTS AT KENYATTA NATIONAL HOSPITAL AT 24-36 WEEKS.

MASTERS OF MEDICINE DISSERTATION IN OBSTETRICS AND GYNECOLOGY, UNIVERSITY OF NAIROBI.

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2010.

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DECLARATION.

I declare that this is my original work and has not to the best of my knowledge been presented for a degree in any other university. I declare that I personally decided to undertake the study as a postgraduate student in the University of Nairobi.

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CERTIFICATION OF SUPERVISORS

This is to certify that Dr Adelaide N Barasa conducted this research as a postgraduate student in the University of Nairobi under my guidance and supervision and the study is being submitted with our approval for a Masters of Medicine degree in Obstetrics and Gynecology.

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CERTIFICATE OF AUTHENTICITY

This is to certify that this dissertation is the original work of Dr Barasa A N; Mmed student registration number H58/7632/06 in Obstetrics and Gynecology department, University of Nairobi (2006-2007). The research was carried out in the department of Obstetrics and Gynecology, School of Medicine, College of Health Sciences. It has not been presented in any other University for award of a degree.

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To you all, I say thank you and may God bless you to continue with the good work.

DEDICATION.

This book is dedicated to my husband, Dr Abner Nasio, my little son Derek and my parents: Marcian and Daniel Barasa who have worked tirelessly to provide the financial, psychosocial and moral support.

LIST OF ABBREVIATIONS:

1. ADA-American Diabetic Association
2. BOH-Bad Obstetric History
3. BMI-Body Mass Index
4. Co2-Carbon dioxide
5. C/S –Caesarian section
6. DM-Diabetes Mellitus
7. FPG-Fasting Plasma Glucose
8. GDM-Gestational Diabetes Mellitus
9. GAD-Glutamate Decarboxylase
10. GLUT-Glutamine
11. H2O-Water
12. HX-History
13. HPL-Human Placental Lactogen
14. IGT-Impaired Glucose Tolerance
15. IFG-Impaired Fasting Glucose
16. IL-Interleukin
17. IRS-Insulin Receptor Substrate
18. KNH-Kenyatta National Hospital
19. OGTT-Oral Glucose Tolerance Test
20. PCOS-Polycystic Ovary Syndrome
21. PPROM-Preterm Premature Rupture of Membranes
22. RDS-Respiratory Distress Syndrome
23. TNF-Tumor Necrosis Factor
24. WHO-World Health Organization

ABSTRACT

Background

The prevalence of glucose intolerance worldwide has been on the increase with the highest incidence being among non-industrialized nations. Data on trends especially in pregnancy and associated risk factors in Kenya is lacking. There is evidence that ante partum screening and early intervention prevents future complications and improves maternal-fetal outcome making it essential to screen at risk mothers during pregnancy. The data from this study will provide the current prevalence rates and guidance on clinical circumstances for which screening will be beneficial.

Objective:

To determine the prevalence of, and associated factors for glucose intolerance among antenatal clients at Kenyatta National Hospital between 24-36 weeks of pregnancy.

Study site:

Kenyatta National Hospital antenatal clinic: Nairobi; Kenya

Study design:

Cross-sectional analytical study.

Materials and methods:

One hundred and two antenatal mothers at a gestational age of 24-36 weeks underwent a 100g OGTT between November 15th 2008 and April 15th 2009 after consenting to participate in the study. Their socio-demographic data, obstetric/ gynecologic history and familial history was obtained through an interviewer administered questionnaire. The results were interpreted according to the Carpenter and Couston/American Diabetic Association Criteria and entered in a results sheet. These were then analyzed using SPSS version 15.

Results

From one hundred and two participants, 37(36%) had glucose intolerance while 65 (64%) had normal glucose tolerance. Among clients with glucose intolerance, 16.7% met the diagnostic criteria for gestational diabetes, 3.9% had impaired glucose tolerance while 15.7% had impaired fasting glycaemia. 22.5% of clients with normal glucose tolerance displayed flat curves.

Factors significantly associated with glucose intolerance were: BMI $\geq$ 25; P value 0.036: OR 0.37 (1.06-6.90), history of and treatment for infertility P value 0.002: OR

8.69(1.74-43.50) and family history of hypertension; P value 0.037: OR 2.66(1.04-6.78).

Conclusion

The prevalence of glucose intolerance was 36 %. The factors associated with glucose intolerance were body mass index ≥ 25, family history of hypertension and history of infertility.

Recommendations

1. Screening - all antenatal mothers should be screened for glucose intolerance regardless of risk status as part of the antenatal profile.
2. Advocacy-there is need to raise awareness about the diabetic epidemic, its public health implications and the need for screening and intervention to reduce complications.

INTRODUCTION

Glucose intolerance is defined as a state of impaired glucose homeostasis characterized by hyperglycemia, abnormalities of lipid and protein metabolism due to the defect in insulin secretion and action. It is currently on the increase with the greatest increase being in developing nations where most of the health resources are spent on treatment and prevention of communicable diseases (1, 2). Currently, diabetes mellitus is the only non-communicable disease with a United Nations Declaration. For its observance, November 14th was declared the world's diabetic day (3).

Gestational diabetes alone is estimated to complicate 1-14% of all pregnancies with mild degrees of glucose intolerance being even higher (4, 5, 6 and 7). It is associated with increased maternal morbidity, fetal/neonatal morbidity and mortality and increased risk of future micro and macrovascular complications (8, 9, 10, 11, 12, 13 and 14). These are potentially preventable through early diagnosis and risk modification (15, 16 and 17). The hormonal milieu of pregnancy is associated with changes in glucose metabolism which may manifest as various degrees of glucose intolerance in individuals with inherited or acquired defects in glucose regulation.

BACKGROUND INFORMATION AND LITERATURE REVIEW

Normal glucose tolerance involves control of plasma glucose within a narrow range despite widely varying input from the gastrointestinal tract. This is due to a balance between glucose entering the cells and that leaving to the extra cellular compartment. The hormone insulin facilitates conversion of glucose to glucose 6 phosphate and then to glycogen through the action of glucokinase in the liver and hexokinase in other tissues (18). Some of the excess glucose is converted to fatty acids and triglycerides stored in adipose tissue. Glucose entry into cells is by facilitated diffusion through GLUT 4 transporters (19)

In glucose deficiency, the liver synthesizes glucose from other metabolites such as glycerol, lactate, amino-acids or breakdown of glycogen by glucose 6 phosphatase; stimulated by the hormone glucagon.

Insulin also inhibits lipolysis and proteolysis. In insulin deficiency or resistance, these actions are impaired resulting in increased plasma glucose levels, lipolysis and proteolysis with subsequent accumulation of fatty acids and ketone bodies.

Several disorders of glucose tolerance characterized by elevated blood sugar beyond normal values have been recognized. These are caused by insulin deficiency, receptor abnormality or post receptor abnormality resulting in reduced glucose entry into cells with stimulation of lipid and protein metabolism (20).They are classified depending on etiology and clinical presentation.

Classification:

1. Diabetes Mellitus

Defined as a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbance of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, action or both. Further sub classified into:

a. Type 1 Diabetes Mellitus

Due to idiopathic pancreatic beta cell destruction or cell mediated autoimmune destruction leading to absolute insulin deficiency.

b.Type 2 Diabetes Mellitus

Most common type. Either due to insulin resistance with relative insulin deficiency, or predominant insulin secretory defect with or without insulin resistance. Mostly associated with obesity.

C.Specific types:

a. Genetic defects of beta cells
b. Genetic defects in insulin action
c. Diseases of exocrine pancreas
d. Endocrinopathies
e. Drug/chemical related
f. Infections

D. Gestational Diabètes Mellitus (GDM)

Defined as glucose intolerance of variable severity, with onset or first recognition during pregnancy.

There are stages of impaired glucose homeostasis which don't meet the criteria for Diabetes Mellitus and which could precede the development of Diabetes Mellitus. These include:

2. Impaired Glucose Tolerance (IGT)

State of impaired glucose regulation characterized by hyperglycemia with blood sugar levels below the threshold required to diagnose Diabetes Mellitus.

3. Impaired Fasting Glycaemia

Fasting glucose levels above normal, but below that diagnostic for Diabetes Mellitus.

People with Impaired Glucose tolerance, Impaired Fasting Glycaemia and Gestational Diabetes Mellitus are at a greater risk of future micro vascular, macro vascular disease and Type 2 Diabetes Mellitus (12, 21).

Epidemiology

Diabetes mellitus is the only non-infectious disease with a UN Resolution where November 14th was declared the world's diabetic day following the recognition of diabetes as a chronic, debilitating and costly illness. The UN called upon all governments in its member countries to develop policies aimed at increasing public awareness and promoting prevention of diabetes and its complications (3). The American Diabetic Association estimates an increase in annual prevalence of 5% per annum since 1990 (22).It is currently the commonest medical disorder complicating pregnancy in the developed world.

Data on prevalence rates for glucose intolerance in pregnancy and the general population in our setup is limited. The overall prevalence rate for glucose intolerance in pregnancy in Kenya is not available but estimates of up to 7 % prevalence of gestational diabetes in some east African countries have been cited (23)

A study done by Githaiga in Kenyatta National Hospital in 1991 reported an incidence rate of 0.15% with ethnic variation (24). Previous studies in Nigeria showed a rate of 0.1% in rural and 3.9% in urban centers for gestational diabetes (25).More recent studies showed a prevalence rate of 2.98 per 1000 (26).

Prevalence rates for gestational diabetes alone in the world range from 1-14 % depending on the race, ethnic group and screening criteria used (4, 5, 6 and 27).

Pathophysiology of glucose intolerance during pregnancy

The first trimester of normal pregnancy is characterized by facilitated anabolism and accelerated starvation. The placental hormones; human placental lactogen (hpl),cortisol, estrogen, progesterone lower blood glucose, promote fat deposition and stimulate appetite. They also increase insulin production and secretion while increasing tissue sensitivity to insulin. The overall effect is lowering of fasting and postprandial glucose levels with increased adiposity (28).

In the 2nd trimester, hpl stimulates lypolysis, reduces hunger sensation and impairs glucose uptake. These together with increased hepatic glucose production results in high fasting and postprandial glucose levels to facilitate transport across the placenta. The increase in free fatty acids and triglycerides further increases insu29).

Late in the 3rd trimester, plateau in human Placental Lactogen with reduced glucose absorption reduces nutrients delivery across the placenta with resultant reduction in placental hormone production. This acts as a signal to the fetus to secrete cortisol and thyroxin which in turn stimulate development of enzymes for maturation of fetal vital organs. Individuals predisposed to glucose intolerance have insufficient compensatory insulin production to counter the effects of the diabetogenic hormones as demonstrated by a lag in the 1st phase of insulin response; an indicator of beta cell dysfunction (20) which is either autoimmune or monogenic in origin (9, 21). The persistently high glucose levels cross the placenta to stimulate fetal pancreatic islet cells causing hyperplasia with increased insulin production. This leads to delays in

fetal organ maturation (10, 20) and promotes fat deposition in the trunk and cardiac muscles.

The mechanisms of glucose intolerance at the cellular level include; low levels of adiponectin (30, 31), increased Tumor Necrosis Factor alpha (TNF-alpha), Interleukin 6 and Leptin mRNA in placental tissue which promote glucose intolerance (10, 28, and 31). Abnormalities in the insulin receptor and insulin signaling cascade, abnormal localization of the glucose transport protein GLUT 4 as well as inherited mitochondrial dysfunction with subsequent reduction in ATP production have been implicated (20, 28, 30, 32,33).

Risk factors for glucose intolerance

Glucose intolerance has been described as a disease of old, multiparous and obese women (32, 34).The lowest incidence is in mothers < 25 years old (36, 37, 40).Nutrient deprivation early in life manifested as birth weight less than 2.7 kg has also been cited as an important risk factor (22).

The most important determinants of GDM are: advanced maternal age especially more than 25 years, high body mass index and family history in a first degree relative (35). Pre-pregnancy weight of 110 % or more of the ideal body weight or significant weight gain in early adulthood is a significant risk factor (28).

The risk of macrosomia in women with moderate to severe degrees of hyperglycemia is estimated to be about 17-29% compared with about 10% in the general population (10,36).The risk of recurrence of GDM in subsequent pregnancy is high averaging about 30-66% (35,37).Other factors include maternal birth weight more than 9 pounds (4.1kg) or less than 2.7 kg (35, 37), history of polycystic ovary disease (35,36), pre-eclampsia and chronic hypertension (35, 37).

Ethnicity is also a risk factor. The WHO and ADA considers the high risk ethnic groups to be the Asian, Hispanic, Africans, Native Americans and Pacific Islanders (27).

Glycosuria may be a normal finding in pregnancy due to increase in glomerular filtration rate with reduction in the renal reabsorptive capacity. However, persistent glycosuria on 2 or more occasions needs to be investigated to rule out glucose intolerance (10).

Patients are categorized into 3 groups depending on the risk status (37, 38 and 39):

1. High risk;

These include:

Obesity BMI ≥25kg/m2; 1st degree relative with DM; history of glucose intolerance; history of delivery of a macrosomia baby ≥4000g; Glycosuria on 2 or more occasions; age more than 25 years; high risk ethnic group/race; bad obstetric history/stillbirth/congenital malformations

2. Intermediate risk -neither high nor low risk
3. Low risk:

Age <25 year; low risk race/ethnic group; no DM in 1st degree relative; no history of abnormal glucose; no poor obstetric outcome; normal weight/pregnancy weight gain.

However, it has been demonstrated that majority of mothers who develop glucose intolerance lack the above mentioned risk factors with only about 30% having the risks(15, 40).

Screening for glucose intolerance.

Glucose intolerance is mostly asymptomatic hence the need for screening. Two screening approaches are available; universal screening or selective screening. American Diabetic Association recommends selective screening (41) but other schools advocate for universal screening (15, 16). The diagnostic approach involves either a 2 step approach involving initial screening with a 50g glucose challenge followed by either a 100g OGTT or a 75g OGTT (38, 41,43), or a one step approach with a 75g or 100g OGTT in populations with a high prevalence. The use of a 3 hour 100g OGTT though associated with poor reproducibility is the gold standard for screening in pregnancy since it has been validated against neonatal outcome and has a high sensitivity and specificity (39, 41, 42).However use of a modified 2 hour 100g OGTT has been shown to have the same diagnostic value as 3 hour OGTT, is less time consuming and causes less patient discomfort (44, 45).

The recommended time for screening is at the time of first contact in high risk groups and between 24-28 weeks in low risk groups. Extending the screening time beyond 28 weeks is associated with increased detection rates but little improvement in neonatal outcome (35). Patients at high risk who have a negative screen early in pregnancy should have a repeat screen after 24 weeks.

The sensitivity and specificity of the 50g glucose challenge test has been shown to vary depending on the population and the cut off points used(46,47, 48).Fasting

blood sugar has been recommended by the WHO as an alternative cheap and less time consuming screening and diagnostic method though its sensitivity is low(49,50).

The use of portable glucose meters is not recommended for diagnosis due to inaccuracies, technical errors, sensitivity to climatic changes and cost(51, 52).However, if carried out by trained technician in presence of good quality control, they offer reasonable quantitative results (53, 54).

There are various method of interpreting the 100g OGTT and 75g OGTT which include:

1. The 100G OGTT according to the National Diabetic Data Group (5, 37, 38, 39): Abnormal values:

Fasting blood glucose $\geq$ 105 mg/dl (5.8mmol/l)

1 hour post prandial $\geq$ 190mg/dl (10.6 mmol/l)

2 hour post prandial $\geq$ 165 mg/dl (9.2 mmol/l)

3 hour post prandial $\geq$ 145 mg/dl (8.0 mmol/l)

2. The 100g OGTT according to the ADA/Carpenter Couston criteria (5, 37, 38, 39). Abnormal values

Fasting $\geq$ 95mg/dl (5.3mmol/dl)

1 hour post prandial $\geq$180mg/dl (10.0mmol/dl)

2 hour post prandial $\geq$155mg/dl (8.6mmol/dl)

3 hour post prandial $\geq$140mg/dl (7.8mmol/dl)

At least 2 abnormal values required to make a diagnosis of GDM. However one abnormal value has been shown to increase the risk of fetal macrosomia and future cardiovascular death (56). The 4^{th} and 5^{th} international conferences on GDM have recommended use of the 100g OGTT according to the Carpenter Couston criteria.

3. The ADA 75G OGTT (37, 38, 39, 55)

Fasting 95 mg/dl (5.3 mmol/l)

1 hour post prandial 180 mg/dl (10.0 mmol/l)

2 hour post prandial 155 mg/dl (8.6 mmol/l).

4. The WHO 75 G OGTT (36, 43, 53)

Fasting >126(7.0mmol/dl)

2 hour post prandial >200mg/dl (11.1mmol/dl):

Some pregnant mothers demonstrate a persistent flat OGTT curve which is defined as blood sugars peak less than 7.0mmo/dl and which does not show a normal trend. This is due to reduced absorption and lower insulin response and is not associated with adverse pregnancy outcome (57) hence a variant of normal glucose tolerance. Use of intravenous glucose tolerance test overcomes the incidence of flat curves though its diagnostic value is not universally accepted (58).

Management of glucose intolerance.

Several management principles are aimed at preventing progression and therefore future macrovascular and microvascular complications and reducing maternal and fetal morbidity and fetal mortality (59, 60).

These include:

1. Lifestyle modification

Exercise, reduction in obesity and dietary modification with consumption of complex carbohydrate is encouraged. Restriction of calories to 25 Kcal/kg in obese and 35 Kcal/kg in normal weight where carbohydrates constitute 35-45%, proteins 20-25 % and 35-40 % being fats. Weight loss reduces the risk of future glucose intolerance by 50% with every kilogram of weight lost reducing the risk by 16 % (17, 59 and 60).

2. Pharmacologic measures;

Insulin is recommended in patients whose sugars can't be controlled on diet and exercise alone. Oral hypoglycemic agents are not approved for use in pregnancy due to potential fetal risk (11).

Patients with severe glucose intolerance require home glucose monitoring or where this is not feasible 1-2 weekly fasting, 1 and 2 hour postprandial glucose monitoring with the goal of maintaining fasting glucose at 5.0-5.3mmol/dl and 1 and 2 hour post prandial at 7.8 and 6.7mmol/dl respectively (38) Fetal surveillance is also recommended in mothers with poor control, those requiring insulin, history of adverse pregnancy outcome or hypertensive disorder. This involves; monitoring of fetal movements, non stress test from 32 weeks, Ultrasound for fetal size and amniotic fluid levels from 32 weeks, Biophysical profile and Doppler studies incase of poor fetal growth or associated hypertensive disease, amniocentesis for fetal lung maturity

if delivery is to be effected before 38 weeks and Cardiotocography if pregnancy should go beyond 40 weeks (37, 38, 39). Delivery should be at term and not more than 40 weeks though this is not universally accepted (55).

3. During the post partum period (37, 38)
(a). A 75 g OGTT should be done after 6 weeks, then every 1-3 years.
(b). Exercise
(c). Weight reduction
(d). Avoidance of diabetogenic drugs including progesterone contraceptives, thiazide diuretics and steroids.
(e). Contraception to avoid conception in presence of hyperglycemia.

Complications

The complications of glucose intolerance are well known (11, 12). Fetal macrosomia is thought to be the commonest complication occurring in 1 in 8 patients (11).This predisposes to birth trauma and increased need for operative delivery.

The risk of major congenital abnormality is 3-8 times that of the general population especially in patients with a fasting hyperglycemia of 6.1 mmo/l or more, while patients with a fasting hyperglycemia $\geq$ 5.8 mmo/l are prone to stillbirth and prematurity (14).

The major cause of prematurity is thought to be pre-eclampsia (59).However the severity of pre-eclampsia is not related to the level of hyperglycemia (21, 28).

Offspring's of mothers with abnormal glucose tolerance are predisposed to early childhood obesity and future glucose intolerance (62). The risk of type 2 DM in the mother is estimated at 50% after 10-15 years while 5% develop type 1 DM (8, 20, 28 and 63).Early gestation age at diagnosis, severe glucose intolerance at the time of 1st diagnosis, obesity, impaired beta cell function and use of progesterone containing oral contraceptives increase the risk of development of future diabetes mellitus (63).

Other complications include preterm premature rapture of membranes, breech delivery and preterm birth (12) .The neonates are predisposed to hypoglycemia, respiratory distress syndrome, hypocalcaemia, polycythemia with resultant hyperbilirubinaemia and hypertrophic cardiomyopathy.The complications occur at low blood sugars than previously thought(64).

RATIONALE

A world wide increase in the prevalence of glucose intolerance has been demonstrated with some regions reporting doubling of prevalence in various racial and ethnic groups between 1990's and the year 2000 (65). This is associated with adverse maternal-fetal outcomes as previously cited, up to 60 % risk of recurrence in subsequent pregnancies, development of type 2 Diabetes Mellitus and future macro and microvascular complications (8, 33, and 37). There is evidence that early intervention prevents these complications (59, 60).The antenatal period provides an opportunity to identify at risk clients and offer primary prevention in terms of lifestyle and pharmacologic measures as well as management of those already affected to prevent maternal complications and optimize fetal outcome.

There is currently limited data on prevalence rates in our set up yet a general increase in glucose intolerance has been observed (66). This study therefore aims at providing current prevalence rates and identifying associated factors in our antenatal population.

RESEARCH QUESTION

What is the prevalence of, and associated factors for glucose intolerance among antenatal clients at Kenyatta National Hospital at 24-36 weeks gestation?

OBJECTIVES

Broad objective

To determine the prevalence of glucose intolerance and associated factors among antenatal clinic attendants between 24 and 36 weeks of gestation at Kenyatta National Hospital.

Specific objectives

1. To determine the socio-demographic characteristics of clients attending antenatal clinic at Kenyatta National Hospital.
2. To determine the prevalence of glucose intolerance among antenatal clinic attendees at Kenyatta National Hospital at 24-36 weeks.
3. To determine the socio-demographic characteristics associated with glucose intolerance among these clients.
4. To determine the obstetric characteristics associated with glucose intolerance among antenatal clients at Kenyatta National Hospital.
5. To determine the familial characteristics associated with glucose intolerance among antenatal clients at Kenyatta National hospital.

METHODOLOGY

Study design:

This was a cross sectional analytical study in which 102 antenatal mothers at 24-36 weeks of gestation was sampled and underwent a 100g 3 hour OGTT. The study was conducted between November 15th 2008 and April 15th 2009 to determine the prevalence of glucose intolerance and associated factors.

Study site:

The study was carried out at Kenyatta National Hospital in Nairobi Kenya. This is a major teaching and referral health facility with a bed capacity of 1800 that provides general and specialized services to both inpatients and outpatients. It is situated abou3 kilometers from the city

centre. It has an Obstetrics Unit that provides services to high risk and regular clients. The in patient Unit comprises of 3 antenatal wards, an acute and a cold gynecology ward. The antenatal outpatient wing attends to about 300 mothers per week from Monday to Thursday.

Patient recruitment was done in the antenatal clinic while the oral glucose tolerance test was done in the biochemistry laboratory number 16 by trained and qualified laboratory technicians.

Study population gestation

This consisted of expectant mothers at 24-36 weeks of gestation who were attending the antenatal clinic between November 15th 2008 and 15th April 2009 who met the inclusion criteria and consented to the study.

Inclusion criteria

1. All clients attending the antenatal clinic within the study period and who were between 24-36 weeks of gestational without pre-gestational or gestational diabetes.
2. All clients who gave an informed consent.

Exclusion criteria

1. Clients who had pre-gestational diabetes mellitus or those already diagnosed with glucose intolerance in current pregnancy or in previous pregnancy which had not resolved by 6 weeks postpartum.
2. All clients who did not consent.
3. Clients outside the defined gestation within the study period
4. Clients who were receiving steroids, Thiazide diuretics, phenytoin or protease inhibitors during the study period

Sampling procedure and sample size estimation

Sampling frame:

This consisted of antenatal mothers attending the antenatal clinic between November 15th 2008 and 15th April 2009.

Sample size

The minimum sample size was calculated using the formula for prevalence rates:

$n = \frac{Z^2Pq}{d^2}$: Where

Z=Standard score 1.96, assuming a 95% Confidence Interval.

P=Prevalence (7%); the average of the estimated total prevalence of GDM of 1-14 %(13)

d=level of precision (0.05)

q= 1-p

Hence ;$[\frac{1.96)^2*0.07*0.93]}{(0.05)^2}$

n=100

Sampling method.

Systematic sampling of every 4th patient reporting in the observation room and who met the study criteria was done. This was derived at by estimating the number of clients who were eligible (20) on each antenatal day divided by the number of clients recruited per day (5). The number of clients recruited per day was limited by the laboratory capacity to a maximum of 5 per day.

Data collection materials:

These included:

1. A pre-tested questionnaire. (enclosed)
2. Glucometer and Glucostics.
3. 100 grams glucose solution (prepared in biochemistry lab 16 at Kenyatta National Hospital)
4. Accessories like: Cotton Wool/spirit swabs, Lancet.

Research personnel

Three research assistants were recruited. A trained nursing officer working within the antenatal clinic who could establish rapport with the clients was trained by the principal investigator on client recruitment and filling of questionnaires. Two laboratory assistants working within the biochemistry laboratory and who administered OGTT on the day to day basis were recruited to conduct the test according to the usual practice except for some modification where the blood sugars were taken at 1 hourly interval instead of the usual half hourly intervals. Their assistance in ensuring that clients were served without delay and that quality control was strictly adhered to was also sought.

Pre-testing of research tools.

A questionnaire and a laboratory request form were designed by the principal investigator and pre-tested one week prior to initiation of data collection on 3 clients to ensure applicability and ease of use. These were then modified by re- structuring and deletion of irrelevant questions before data collection was initiated.

Data collection methods:

After obtaining permission from Kenyatta National Hospital/University of Nairobi Ethics and Research Committee, the nursing assistant sensitized the mothers about the study every morning during the health education talk.

Sampling was then done where every 4th client who reported in the observation room and met the inclusion criteria was approached by the Research assistant and the Principal Supervisor and an informed consent sought and signed after explanation on the nature of the study, procedures involved (obtaining a fasting blood sugar through a needle puncture followed by consumption of a sugar solution and thereafter hourly blood sugars for 3 hours), its importance, assurance of safety and side effects (vomiting). Those who did not consent were assured that their decision would not compromise their quality of care received. Mothers who requested for the test but were not among those sampled were offered the test but were not included in the study.

A questionnaire was then administered to all participants who consented. This captured the socio-demographic characteristics, past and current

obstetrics/Gynecologic history and any personal/familial risk factors for glucose intolerance e.g. pre-pregnancy weight, family history of Diabetes Mellitus, hypertension, previous history of glucose intolerance, sudden deaths in a first degree relative, family history of cerebrovascular accidents and previous deliveries of infants weighing more than 4.0 kg. Information on height, trends in weight gain, correspondence of fundal height to gestational age, presence of glycosuria in current pregnancy and current blood pressure were obtained from the clinic records.

They were then advised to have a non-restricted high carbohydrate diet (all the available carbohydrates they can access) for 3 days and come back on the 4th day before a meal for a 3 hour 100g OGTT. Most of the clients were on an adequate carbohydrate diet and 3 days were not required but were booked to come for the test on the day that was convenient for them. They were advised to eat up to midnight on the night before the test and then come to laboratory 16 before breakfast between 8.00 and 9.00 am the following day for the test. The clients were advised to carry a snack to take after the test.

Laboratory method:

A fasting capillary blood sample was taken 30 minutes after arrival in the laboratory (finger puncture after swabbing the finger with methylated spirit) and tested for sugars. A 300 ml solution containing 100g of glucose was then administered orally over 5 minutes and capillary blood drawn and tested for sugars hourly for 3 hours. Clients were advised to minimize physical activity during the test. Reading materials were provided during the waiting period. The blood drop obtained after a needle prick was analyzed using reflectance meters with accompanying strips.

After the test, they were allowed to take the snack, informed about the results and a copy of the results given to them to take to the attending obstetrician. Those who met the diagnostic criteria for GDM were in addition given a consultation by the principal investigator to take to the diabetic clinic. The clients who vomited on taking the glucose solution were booked to come on a different day for a glucose solution containing citric acid.

Quality control

The Glucometer was run through a control strip whenever any set of glucose strips was opened and then subjected to weekly calibration. The glucose testing was

periodically supervised by a qualified pathologist at regular intervals and standards maintained in accordance with preset standard in a reference laboratory in the U.K.

Interpretation of the test was done according to the Carpenter Couston criterion which is recommended by the ADA and which has been shown to correlate to neonatal outcome (29, 37, 38 and 39) as follows:
Normal values:

1. Fasting blood sugar < 5.3mmol/dl
2. 1 hour post prandial <10.0mmol/dl
3. 2 hour post prandial < 8.6mmol/dl
4. 3 hour post prandial < 7.8 mmol/dl

Abnormal values
Any 2 or more values equal to or greater than the above or a fasting blood glucose ≥ 7.0mmol/dl were classified as GDM. Presence of one abnormal 1, 2 or 3 hour value was classified as impaired glucose tolerance. A fasting blood sugar more than 5.3mmol/dl but less than 7.0 mmol/dl was classified as Impaired Fasting Glycaemia (29, 38 and 39). A test result where the peak glucose level was less then 7.0 mmo/dl without the normal peak at 30-60 minutes was classified as flat glucose tolerance curve.

For analysis, normal glucose tolerance(all 3 values within normal limits)and Flat curves with fasting glucose below 5.3 mmol/dl were analyzed as normal glucose tolerance while GDM, impaired fasting glycaemia and impaired glucose tolerance as `Glucose intolerance'.

Those with abnormal results were given advice on importance of exercise, dietary modification (consumption of high fiber diet, complex carbohydrates and less fat), exercise and post pregnancy weight loss. They were also counseled on the need to undergo a 75g OGTT at 6 weeks postpartum and annually thereafter. A copy of the result was given to the participants to take to the attending clinician.

STUDY LIMITATIONS:

1. There was a possibility of bias with participants with family history of diabetes being more likely to consent and yet not disclose the information.
2. Some patients may not have fasted before coming for the OGTT.
3. Not all the clients who consented turned up for the test. This was addressed by extending the recruitment period till the desired sample size was attained.
4. Credibility of the data. This was addressed by counter checking with the clinic records to ensure that the information given was similar with that in antenatal records and those with conflicting information were contacted for clarification.

ETHICAL ISSUES;

1. The proposal was submitted to the Kenyatta National Hospital/University of Nairobi Ethics and Research committee (KNH/UON,ERC) and was approved before the study was undertaken. A copy of the approval letter is attached at the appendix section.
2. No patient was forced or coerced and no financial inducements were used to influence participation.
3. All patients participating in the study signed an informed consent.
4. The study was not harmful. No major adverse effects were experienced
5. All patients diagnosed with glucose intolerance were counseled and copies of the results sent to the clients' files to aid in their antenatal management and follow up.
6. Confidentiality was maintained during data collection and revelation of results.

DATA MANAGEMENT.

Data was collected using pre-tested questionnaires while laboratory results were recorded on request forms. These were checked for completeness before entry into excel. Data cleaning and entry was done followed by analysis using statistical scientific package version 15 (SPSS 15).

RESULTS

The study period extended from 15th November 2008 to 15th April 2009. A total of 102 clients were recruited and underwent a 3 hour 100g OGTT. The results are presented below in tables and charts.

TABLE 1:

THE SOCIO-DEMOGRAPHIC CHARACTERISTICS OF THE STUDY CLIENTS.

Socio-demographic characteristics		Frequency N=102	Percentage (%)
Age			
	< 25 years	21	20.6
	≥ 25 years	81	79.4
Marital status			
	Single	11	11
	Married	89	87
	Non response	02	2.0
Residence in the past 10 years			
	Rural	13	12.7
	Urban/rural then urban	89	87.3
Level of education			
	Primary and below	13	13.0
	Secondary and above	87	85.0
	Non response	02	2.0
Employment status			
	Self	30	29.4
	Salaried	28	27.5
	Unemployed	44	43.1
Income per month			
	< 15,000ksh	36	35.3
	15,000-30,000ksh	22	21.6
	Non response	44	43.1
Pre-pregnancy BMI	≥ 25	36	35.3
	< 25	41	40.2
	Unknown	25	24.5

79% of the participants were aged 25 years and above with a mean age of 29.3 years. 11% were single while 89% were married. Most had resided in urban centers within the past 10 years. 87 % had received at least secondary education with just slightly more than 50% being employed.

TABLE 2:

OBSTETRICS AND FAMILIAL CHARACTERISTICS OF THE STUDY CLIENTS

1. Obstetric characteristics	**Frequency**	**(%)**
Gravidity (102)		
Primigravid	36	35.3
Multigravid	66	64.7
Glycosuria in current pregnancy	4	3.9
Polyhydramnous in current/past pregnancy	1	1
Pregnancy larger than dates	15	14.7
Previous neonatal weight ≥ 4000g	8	7.8
Previous pregnancy wastage	16	15.6
Hypertension in previous pregnancy	11	10.8
History of Gestational diabetes	1	1
History of infertility	10	9.8
Medical treatment for infertility	6	5.9
2. Familial characteristics	**frequency**	**(%)**
Family Hx of diabetes	20	19.6
Family Hx of hypertension	24	23.5
Family Hx of sudden death	6	5.9
Family Hx of CVA	9	8.8
Family member with obesity	4	3.9

Only 1 % had been diagnosed with glucose intolerance in previous pregnancy which had subsided after delivery. About 16 of the participants reported an adverse pregnancy outcome; an abortion, stillbirth or preterm delivery. Only 7.8 % had had a previous delivery to a neonate ≥ 4000g, 9 reported having had difficulty with conception, out of which 6 had received some form of treatment for the infertility. The mean birth weights for previous deliveries were 3.19 kg (SD 0.69) for first; 3.22 kg (SD 0.73) for second and 3.86 kg (SD 0.30) for third deliveries.

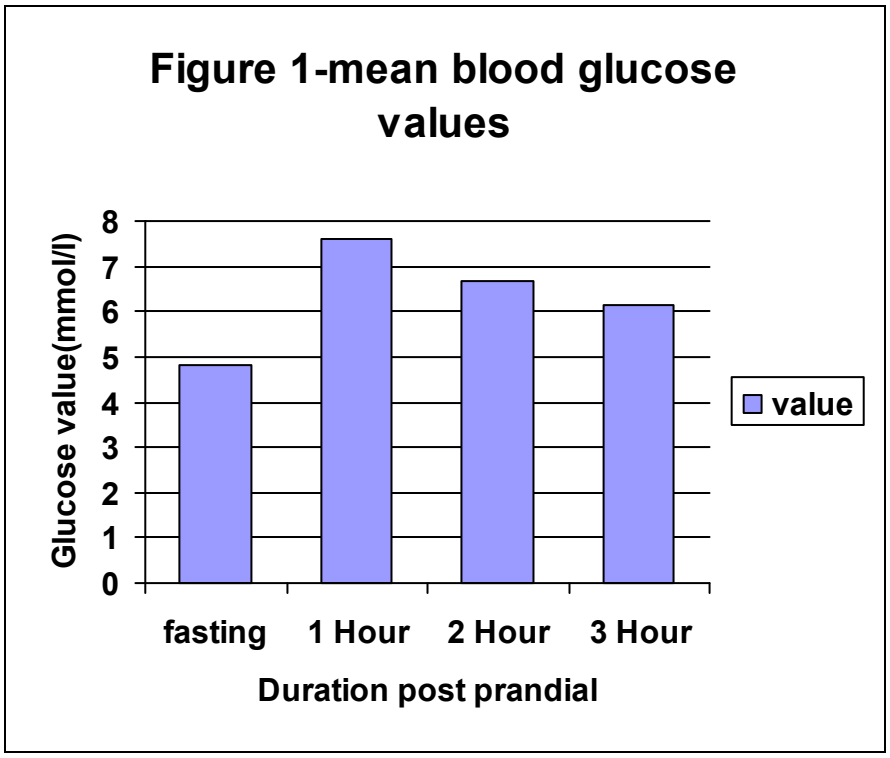
About 19 % of participants gave a positive family history of diabetes while 23% had a family history of hypertension. A small proportion had a family history of other indicators of vascular disease.

2. LABORATORY RESULTS

Mean blood glucose values.

The mean blood glucose values were 4.82mmol/dl fasting (1.00), 1 hour post prandial of 7.59mmol/dl (1.55), 2 hour post prandial of 6.68 mmol/dl (1.35) and 3 hour post prandial of 6.15 mmol/dl (1.29) with the ranges of 3.1-12.1mmol/l, 3.4-11.9 mmol/l, 4.0-9.8 mmol/l and 3.8-12.2 mmol/dl for fasting, 1, 2 and 3 hour post prandial respectively.

Figure 1:

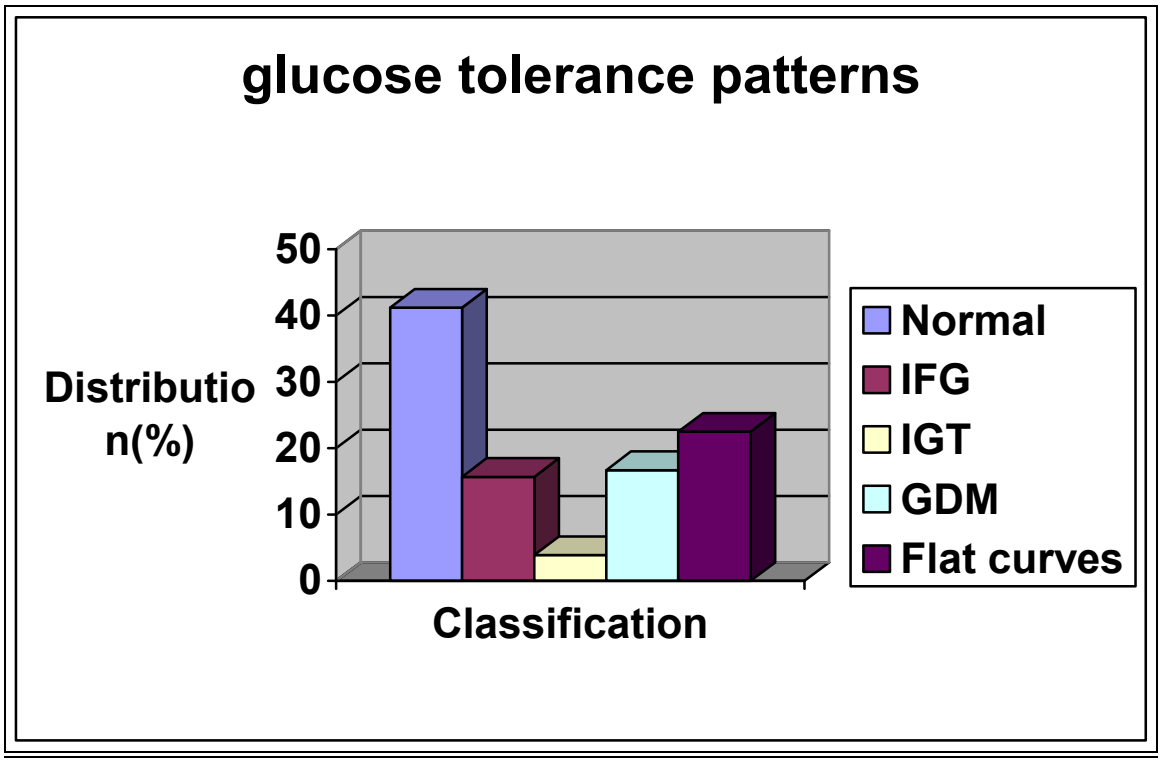


KEY: **PP**-Post prandial

Glucose tolerance patterns

Out of the 102 participants, 42 (41.2) had normal glucose tolerance, 23 (22.5%) met the diagnostic criteria of flat curves, while 19.6 had mild degrees of glucose intolerance (one abnormal value). 16.7% met the diagnostic criteria of gestational diabetes mellitus as shown in figure 2 and 3 below.

Figure 2-Glucose tolerance pattern.



Key: **IFG**-Impaired fasting glycaemia

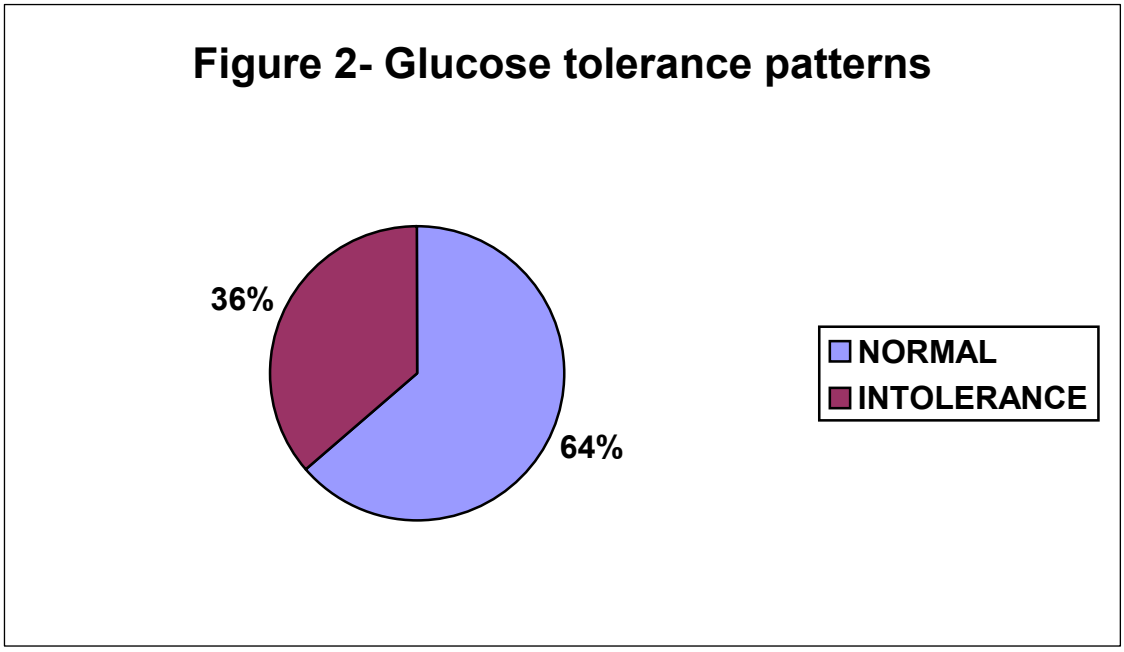
IGT-Impaired Glucose Tolerance

GDM-Gestational Diabetes Mellitus

Normal-Normal glucose tolerance.

Figure 3 –glucose tolerance pattern.

Overall, the total number of clients with normal glucose tolerance was 65(63.7%) i.e. normal plus flat curves while 37 (36.3%) had glucose intolerance (impaired fasting glycaemia, impaired glucose tolerance and Gestational Diabetes Mellitus) as depicted in figure 2 below. Of the clients with glucose intolerance, 48 % were at a gestational age of 28 weeks and below while 52 % were more than 28 weeks gestational age.



FACTORS ASSOCIATED WITH GLUCOSE INTOLERANCE.

TABLE 3: SOCIO-DEMOGRAPHIC CHARACTERISTICS AND GLUCOSE INTOLERANCE.

	Glucose intolerance		**O.R (95% CI)**	**P Value**
	Yes	**No**		
Age in years				
<25	4 (19.0 %)	17 (81.0 %)	0.34 (0.11-1.11)	0.07
≥ 25	33 (40.7 %)	48 (59.3 %)		
Marital status				
Single	4 (36.0 %)	7 (64.0 %)	1.02 (0.27-3.6)	0.98
Married	32 (36.0 %)	57 (64.0 %)		
Residence in the past 10 years				
Rural	5 (38.0 %)	8 (62.0 %)	1.11(0.33-3.69)	0.86
Urban /Rural then urban	32 (36.0 %)	57 (64.0 %)		
Level of Education				
Primary	4 (31.0 %)	9 (69.0 %)	0.76(0.22-2.68)	0.67
Secondary/tertiary	32 (37.0 %)	55 (63.0 %)		
Employment status				
Employed	21 (36.0 %)	37 (64.0%)	0.99 (0.44-2.25)	0.99
Unemployed	16 (36.0 %)	28 (64.0%)		
Income per month				
< 15,000	10 (28.0%)	26 (72.0%)	0.38(0.13-1.17)	0.09
15000-30,000	11 (50.0%)	11 (50.0%)		
Body mass index				
< 25	12(29.0)	29 (71.0)	0.37(1.06-6.90)	0.036
≥ 25	19(53.0)	17 (47.0)		

There was no association between age, marital status, residence, level of education and income with glucose intolerance. Body mass index ≥ 25 was positively associated with glucose intolerance.

TABLE 4:

OBSTETRIC CHARACTERISTICS AND GLUCOSE INTOLERANCE.

	Glucose intolerance		O.R(95 % CI)	P Value
	Yes	No		
Gravidity Primigravid Multigravid	 14 (39.0%) 23 (34.8%)	 22 (61.0%) 43 (65.2%)	 1.19(0.51-2.76)	 0.69
Glucosuria in current pregnancy	3 (75.0%)	1(25.0%)	5.65 (0.57-56.39)	0.10
Excess liquor in this pregnancy	1 (100.0)	0 (0)	-	0.18
Current pg larger than dates	6 (40.0%)	9 (60.0%)	1.21(0.39-3.71)	0.75
Neonatal weight ≥4000g	5 (62.5%)	3 (37.5%)	3.2 (0.73-14.38)	0.11
Neonatal NBU admission	2 (22.2%)	7 (77.8%)	0.47 (0.09-2.41)	0.36
Previous pregnancy loss	6 (37.5%)	10 (62.5%)	0.58(0.23-1.48)	0.25
Hypertension in pregnancy	4 (36.4%)	7 (63.6%)	1.00(0.27-3.69	1.0
Hx of gestational D.M	1 (100.0%)	0(0)	-	0.18
Difficulty in conception	8 (80.0%)	2 (20.0%)	8.69(1.74-43.50)	0.002
Rx for infertility	5 (83.3%)	1 (16.7%)	10 (1.12-89.22)	0.013

No association was demonstrated between parity, larger for gestational age pregnancy, prior pregnancy loss, hypertension, polyhydramnous, Glycosuria and neonatal NBU admission and glucose intolerance. Positive history of difficulty in conception and treatment for infertility were associated with statistically significant risk as shown in table 4 above.

TABLE 5:

FAMILIAL CHARACTERISTICS AND GLUCOSE INTOLERANCE.

	Glucose intolerance		**O. R(95% CI)**	**P Value**
	Yes	No		
1st degree relative with hypertension	13 (54.2%)	11 (45.8%)	2.66 (1.04-6.78)	0.037
1st degree relative with diabetes	11 (55.0%)	9 (45.0%)	2.63 (0.97-7.13)	0.05
Family Hx of sudden death	3 (50.0%)	3 (50.0%)	1.82 (0.35-9.53)	0.471
CVA in a 1st degree relative	1 (11.1%)	8 (88.9%)	0.20 (0.02-1.65)	0.10
Family member with obesity	3 (75.0%)	1 (25.0%)	5.65(0.57-56.39)	0.10

No association was found between a family history of sudden death, cerebrovascular accident in a first degree relative and a first degree relative with diabetes/obesity with glucose intolerance. A positive family history of hypertension was associated with statistically significant risks as shown in table 5 above.

DISCUSSION

The study comprised of 102 participants with a mean age of 29.3 years and a median age of 28.0 years (SD 5.40). 79% of clients in this study were aged more than 25 years. Githaiga in 1986 demonstrated that 75 % of mothers with diabetes mellitus were aged more than 25 years (24) while in this study, only 41% of mothers with glucose intolerance were aged more than 25 years. From Githaiga's study 85% of the mothers were married, a similar finding to our antenatal population where 89 % of mothers were married while only 11% were single; perhaps a good indicator of the important role played by the family unit in child upbringing. However, only 41 % of mothers with glucose intolerance were married. 66 of mothers in the study were parous; 35 % of these had glucose intolerance, a figure different from Githaiga et al where 91 % of his antenatal population was composed of multiparous clients all of whom had diabetes mellitus. Other similar studies have associated glucose intolerance to high parity (32, 24).

The mean gestational age for the participants was 28.8(SD 2.78) with a median of 29 weeks and a range of 24-36 weeks. The mean birth weights for previous deliveries for the study population were 3.19 kg (SD 0.69) for first; 3.22 kg (SD 0.73) for second and 3.86 kg (SD 0.30) for third deliveries. Glucose intolerance is associated with higher birth weight due to glucose deposition and increased adiposity (10, 20). In the study, the mean birth weights for clients with glucose intolerance was similar to the study population with regard to the first delivery but high for the second and third deliveries thus; 3.1, 3.4 and 3.9 kg for 1^{st}, 2^{nd} and 3^{rd} deliveries respectively. Fetal macrosomia is estimated to complicate one in every 8 mothers with glucose intolerance (11). Previous findings in KNH have found a prevalence of fetal macrosomia in diabetic mothers to be 24.1 % (24).In our current antenatal population; only 7.8 % mothers gave a history of previous infant birth weight $\geq$ 4000g. 63 % of these displayed glucose intolerance though the association was not statistically significant. This is higher than the findings of the Nairobi birth survey IV which showed an incidence rate of fetal macrosomia of 4.2 % in the general population and 1.3 % in teenagers (68).

The prevalence of familial risk factors for microvascular disease ranged from 19 % for diabetes which is 5 times higher than our estimated national prevalence of type 2 diabetes in the general population which is estimated at 3.3 % (69). This was not

statistically significant, P value 0.05: OR 2.66(1.O4-6.78), perhaps due to the small sample size. 24 % reported a family history of hypertension which was statistically significant, P value 0.037; OR 2.66 (1.04-6.78); an alarming figure considering that this was a non selected low risk population. This is thought to be due to increased vascular reactivity in individuals with genetic defects in beta cell function which predispose to development of hypertension and glucose intolerance (20, 28 and 31). Only a small proportion reported a family history of obesity (3.9) which is unreliable since it is a subjective assessment though it was not a statistically significant association.

The results showed a prevalence of glucose intolerance of 36 % with gestational diabetes comprising 16.7%, mild degrees of glucose intolerance was 19.6% while flat curves comprised 22.5%. This depicts high rates of glucose intolerance as compared to previous estimations of less than 1% incidence of gestational diabetes by Githaiga in 1986 (24). Rates of 7 % for East Africa have been estimated in the year 2002 though this was based on fasting blood glucose (23). This is in line with the current observations that have demonstrated similar increase especially in third world Nations (1, 2). However, most studies have restricted screening to a gestation of 24-28 weeks hence this could lend support to the benefit of extending the screening time to beyond 28 weeks to increase detection rates (35) as 52 % of mothers with glucose intolerance in this study were between 29 to 36 weeks. Recent estimates in Kenya have cited rates of up to 11% prevalence in some high risk rural communities in central Kenya and up to 20% prevalence rates in high socio-economic urban areas (66). The prevalence of flat curves which is thought to be due to intestinal stasis with reduced absorption is slightly more (22 %) than that observed in previous studies by Thuo in 1980 which showed figures of 18 %(57).

The ADA recommends a two stage screening approach starting with a 1 hour 50g glucose challenge followed by a 3 hour OGTT in populations with low prevalence rates. In populations with rates more than 2 %, a one step screening approach using an OGTT is recommended (37). Our antenatal clients meet the criteria for routine 3 hour OGTT beginning at 24 weeks gestation.

Several socio-demographic characteristics have been associated with glucose intolerance among them; age more than 25 years, history of previous adverse pregnancy outcome, high socio-economic status/recent exposure to affluence, certain ethnic/racial groups among them the African race and pre-pregnancy weight gain

more than 110 % of the ideal body weight (37, 40).Wagaarachchi and others have demonstrated lower maternal age to be associated with a 50% lower incidence of GDM as compared to mothers of advanced age (40). Among our antenatal population, mothers aged less than 25 years had a three fold lower risk of glucose intolerance compared to mothers aged over 25 years: 19 % verses 41 % respectively. Only 28 % of clients who earned less than 15,000 ksh had glucose intolerance as compared to 50 % with income more than 15,000 ksh; an indicator of the effect of socio-economic status though the difference was not statistically significant. There was no difference in residence in the past 10 years and employment status with glucose intolerance contrary to expectation as urban residence or migration from rural to urban centre has consistently been associated with a higher risk of glucose intolerance (2, 22). There was a slight insignificant difference in glucose intolerance between clients who attained primary education (31 %) and those with secondary/ tertiary education (37 %), perhaps an indicator of socio-economic status. A pre-pregnancy BMI $\geq$ 25 was positively associated with glucose intolerance; a similar finding to other studies (34, 37). This could be a risk factor per se but could also be influenced by maternal age, parity and socio-economic status. Of note is that 35% of the study population had a pre-pregnancy BMI of $\geq$ 25, a figure far above the estimation given by the 2003 Kenya Demographic and Health Survey of 23 percent (67). This raises concern considering that up to 24.5 % of the mothers did not know their pre-pregnancy weight and therefore their BMI. It looks obvious that our clients don't have knowledge on the role that weight plays in contributing to future cardiovascular disease.

The ADA and the American College of Obstetricians and Gynecologists recommends universal screening in mothers with certain obstetric characteristics among them Glycosuria at the first prenatal visit, Polycystic Ovary syndrome, previous delivery of a baby more than 4.0 kg, personal history of abnormal glucose tolerance, previous unexplained perinatal loss or congenital malformation, maternal birth weight greater than 4.0 kgs or less than 2.7 kgs and multiparity (35, 37). From the study up to 75 % of mothers with glycosuria, 100 %(1) of mothers with history of gestational diabetes and polyhydramnous had glucose intolerance though the numbers were small and statistically insignificant. On the other hand, only 40 % of clients with uterine fundus more than dates, 22 % with history of neonatal newborn admission and 37 % with history of prior pregnancy loss had glucose intolerance which was contrary to expectation (24, 34, 35 and 37). Similarly, hypertensive disorder in previous or

current pregnancy was not an association with only 36 % of mothers with hypertension demonstrating glucose intolerance. This could be due to the fact that mothers with hypertension are likely to be young primigravidas with a low risk for glucose intolerance even though age and parity were not significant associations in this study. The screening was also a single point investigation which could have missed mothers who could have developed glucose intolerance at a later stage. On the contrary, nulliparity was associated with a higher prevalence of glucose intolerance (39 %) compared to multiparity (35 %).

Only one mother with glucose intolerance had a history of glucose intolerance in prior pregnancy which had subsided post delivery. This low prevalence could be due to lack of routine screening among our antenatal population hence most asymptomatic mothers are not diagnosed.

History of infertility was associated with glucose intolerance, P value 0.002; OR 8.69 (1.74-43.50).The causes and treatment modalities for the infertility were not documented. Polycystic Ovary syndrome is a known risk factor for infertility and could account for this association (17).

Most of the cited risk factors for glucose intolerance were not significant associations in this study. The only positive associations were a BMI $\geq$ 25 (p value 0.036, OR 0.37 (0.15-0.95)), history of and treatment for infertility and a 1st degree relative with hypertension. This lends support to the advocates of universal testing as opposed to selective testing based on risk factors as it is estimated that only about 30% of mothers who develop glucose intolerance have risk factors (15, 40). However, the study did not have sufficient power to determine these associations. It was also a single point investigation which could have missed some mothers who could have developed glucose intolerance at a later stage.

Glucose intolerance is a preventable illness that is associated with high morbidity and mortality due to associated macrovascular and microvascular complications including blindness, nephropathy, coronary artery disease, diabetic feet and neuropathy. It is estimated that the US spends up to 142 billion dollars per year on management of diabetic complications alone (36). From this study, it is obvious that we are at a high risk like most other third world nations (1, 2).The cost of screening is only about 3 dollars (220 ksh) per person. This should be a wake up call to divert our efforts not only on prevention and treatment of HIV AIDS, TB and Malaria but also

on screening and prevention of glucose intolerance which has previously been regarded as a disease of the affluent

CONCLUSION.

1. The prevalence of glucose intolerance was 37 %. Gestational diabetes comprised 16.7% which was high compared with previous estimations of less than 1% in 1986 and 7 % in 2002 in East Africa.

2. Most of the internationally recognized factors such as age more than 25 years, family history of diabetes, history of macrosomia, persistent Glycosuria and previous adverse pregnancy outcome were not significantly associated with glucose intolerance.

3. Only three recognized risk factors i.e. BMI $\geq$ 25, family history of hypertension and history of infertility were associated with glucose intolerance.

4. Risk factor stratification may not be a reliable method in identifying mothers at risk for GDM in our set up.

5. Extending the screening time beyond 28 weeks results in increase in detection rates.

RECOMMENDATIONS.

1. There is need for routine screening of antenatal mothers using a glucose tolerance test which should be extended beyond 28 weeks regardless of risk to optimize fetal outcome and offer preventive measures to prevent future risk of developing diabetes mellitus. This is particularly important in mothers with a history of infertility or polycystic ovary disease.
2. A follow up study is needed to asses the impact of various degree of glucose intolerance on fetal and maternal outcome in our set up.
3. There is need to educate the wider public on the increasing prevalence of diabetes, its associated complications and risk factors so as to ensure acceptance and widespread screening.
4. There is need for a large nationwide study to establish the true prevalence of glucose intolerance in pregnancy in the country which is currently unavailable.

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APPENDICES:

1. CONSENT FORM:

A.*ENGLISH*:

I, _____

of Study number_____

hereby agree to participate in the study of 'glucose tolerance'. I understand that I will be required to come for a glucose test after fasting for at least 8 hours and serial blood sugars will be taken after consumption of a glucose meal.

I have been informed about the benefits of participating in this study which include identifying my future risk of developing diabetes and birth complications which can be prevented if detected. I also understand that i will have to remain seated for 3 hours and there is a possibility of being unable to tolerate the glucose meal.

No coercion has been used to influence my decision to participate in the study whose nature, benefits and risks have been explained to me by Dr/Mr/Mrs_____of postal address _____and Telephone number_____ .

Signed: client: _____Tel No._____

Health care provider: _____

Researcher's Tel number_____0723-875522

Hospital's Tel Number (KHN) __726300-9

B .*KISWAHILI:*

Mimi_____, wa Nambari ya utafiti_____nakubali kushiriki Kwa utafiti wa kuchunguzwa jinzi mwili unavyotumia sukari.

Ninaelewa kuwa nitahitajika kurudi kupimwa sukari baada ya kutokula chakula Kwa muda usiopungua masaa nane. Ninaelewa kuwa nitapewa maji ya sukari kisha kiwango cha sukari mwilini kipimwe kila baada ya saa moja kwa muda wa saa tatu.

Nimefahamishwa umuhimu wa kuhusika kwa uchunguzi huu ambao ni kujua kama niko hatarini ya kupata ugonjwa wa sukari maishani au kupata matatizo wakati wa uzazi. Ninaelewa ya kwamba nitahitajika kungojea kwa muda wa masaa tatu na kuna uwezekano sitaweza kuvumilia kunywa maji ya sukari.

Nimekubali kushiriki Kwa huu uchunguzi kwa hisani yangu bila kushurutishwa na yeyote.Namna ya uchunguzi, faida na madhara yake yameelezwa na Bwana/Bi/Daktari_____wa nambari ya Posta_____ nambari ya simu_____

Sahihi ya muhusika: _____

Anwani_____

Nambari ya simu_____

Sahihi ya mhudumu wa afya_____

Nambari ya simu ya mtafiti_____0723-875522

Nambari ya simu ya hospitali___ 726300-9

2. DATA SHEET:

1. SOCIO-DEMOGRAPHIC DATA:

a. What is your name? _____

b. Study number_____

c. When were you born? Date_____month_____year_____

d. What is your marital status? (a) Married (b) single (c) Divorced/separated

e. Where have you been staying in the past 10 years? (a) Rural (b) urban (c) Rural then urban (d) Urban then rural

f. What is your level of education? (a) Primary (b) secondary (c) tertiary (d) none

g. Are you currently employed? (a) Yes (b) no

If yes, move to h and i.

h. What is the nature of your employment? (a) Self employed (b) salaried

i. What is your level of income per month? (a) <15,000ksh (b) 15-30,000ksh (c) >30,000ksh

2. PAST OBSTETRIC/GYNECOLOGIC HISTORY:

a). Is this your first pregnancy? (a) Yes (b) No

If No to (a) above, answer questions (b) to (p)

b). How many pregnancies have you carried beyond 37 weeks?

_____

c). How children have you delivered while dead (stillbirths) after 37 weeks ?

_____

d). How many pregnancies have you lost before 28 weeks?

_____

e). How many pregnancies have resulted in preterm births between 28-37 weeks? (explain)

_____

f).How many pregnancies have resulted in stillbirths (explain) between 28-37 weeks?

_____

i). Have you delivered an infant with an abnormality

(a) Yes (b) No

j). Have you had a caesarian delivery?

(a) Yes (b) No

k). what were the birth weights of your previous deliveries?

1._____

2._____

3._____

4._____

5._____

Others

l). Have you had elevated blood pressure in any of the previous pregnancies? (a) Yes (b) No

m). Have you ever been told that you have excess amniotic fluid in any of the previous pregnancies?(explain) (a) Yes (b) No

n). Have you ever been told that you have glucose in urine in any of the previous pregnancies?

(a) Yes (b) No

o). Have you been diagnosed with diabetes in any of the previous pregnancies?

(a) Yes (b) No

p). Has any of your previous infants been admitted to nursery?

(a) Yes (b) No

q).Have you had any difficulty in conception?

(a) Yes (b) No

i).If yes to q above, were you given any treatment?

(a) Yes (b) No

3. CURRENT PREGNANCY HISTORY:

a). what was your last normal menstral period?

Date_____month_____year_____. Gestation in weeks_____

If unknown, what is the estimated gestation by ultrasound? _____

b). what is your current weight in Kilograms (confirm from clinic notes_____

c).What is your current height (measure/confirm from clinic Notes)._____ meters.

d). BMI_____

e). Have you had glucose in urine in the course of this pregnancy? (Confirm form the file) (a) Yes (b) No

f). what was your booking blood pressure? (Check in the file)

Systolic_____ Diastolic_____

g) What is your current blood pressure? (Check in the file)

Systolic_____Diastolic_____

h). Have you been diagnosed with excessive liquor in the course of this Pregnancy? (Check with clinic records) (a) Yes (b) no

h).Is your current pregnancy larger than dates? Confirm from clinic records and ultrasound scans if available. (By>2wks)

(a) Yes (b) no

4. FAMILY/PERSONAL HISTORY:

a. What was your weight before this pregnancy (a) _____ (b) unknown

b. Do you have/have you had a first degree relative with diabetes mellitus (explain) (a) yes (b) no

c. Do you/have you had a 1st degree relative with high blood pressure (Explain) (a) Yes (b) no

d. Has any of your close family member/relative died suddenly?
(a) Yes (b) no

e). Has any of your family member/close relative suffered a stroke?
(Explain) (a) Yes (b) no

f).Do you/have you had a close relative who is overweight? (Subjective)
(a) Yes (b) no;

RESULTS

a. Date of results: Date_____month_____Year_____

b). FASTING BLOOD SUGAR_____ (N <5.3 mmol/dl)

c. 1 HOUR POSTPRANDIAL GLUCOSE_____ (N < 10.0 mmol/dl)

d. 2 HOUR POST PRANDIAL_____ (N<8.6 mmol/dl)

e. 3 HOUR POSTPRANDIAL GLUCOSE _____ (N < 7.8 mmol/dl)

6. From the results, classify the patient:

(a) Normal glucose tolerance

(b). Impaired Fasting Glycaemia

© . Impaired Glucose Tolerance (ONE ABNORMAL VALUE)

(d) . Gestational Diabetes Mellitus

(e). Flat curves