IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 15, Issue 6 Ver. XII (June 2016), PP 70-74
www.iosrjournals.org

Gestational Diabetes And Type 2 Diabetes Mellitus.

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Abstract*: Gestational diabetes mellitus (GDM) is the type that develops during pregnancy and it occurs in about 3-5% of pregnant women, usually in the third trimester. This is widely believed to be due to "insulin resistance"- a situation where it is hard for the mother's body to make use of insulin. The placenta that supports the growth of the baby also produces some hormones that help the baby and these hormones hinder the action of insulin thus preventing it from doing its job-controlling blood sugar. Gestational diabetes usually goes away after the birth of the baby but, however many women go on to develop type 2 diabetes mellitus years later, with the prevalence depending on ethnic background. In this study between January 2003 and April 2009, a total of 478 pregnant women attending ante-natal clinics in three private hospitals were involved. Within that period, 8(30%) were found to have type 2 diabetes mellitus out of 33(6.9%) with gestational diabetes.*
Keywords*: Gestational diabetes, Type 2 diabetes mellitus, hyperglycemia*

I. Introduction

Gestational diabetes mellitus (GDM) is any glucose intolerance first detected during pregnancy and it is known to terminate after delivery. This study sought to see the relationship between GDM and development of Type 2 DM among a small percentage of pregnant women in a city population in South West Nigeria to give an insight into the prevalence. GDM is a public health challenge as it poses problems to the mother and the child. The result of the study would help to reducing the maternal and foetal complications by taking more precautionary measures and indirectly achieving Millennium Developmental Goal of reducing maternal and childhood mortality.

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia which results from defects in the secretion and action of insulin [1, 2, 3]. Several millions of people have diabetes globally including Africa [4, 5]. The deficient action of insulin arises from inadequate insulin secretion and/or diminished tissue responses to insulin and in most cases impairment of insulin secretion and defects in insulin action coexist in the same patient [1].There are many pathogenic processes that accounted for the development of diabetes. These include the progressive autoimmune destruction of the β-cells of the pancreas with resultant impaired insulin secretion; other abnormalities with resultant resistance to insulin action [6].

Uncontrolled or poorly controlled diabetes results in marked hyperglycaemia with subsequent complications like, ketoacidosis, non-ketonic hyperosmolar syndrome, damage and failure of different organs, especially the kidneys, nerves, eyes and heart. The symptoms of marked hyperglycaemia include polyuria, polydipsia, weight loss, and blurred vision [1].

1.1. Types of Diabetes

In the recent years new classification identified four types of diabetes mellitus as Type 1 diabetes, Type 2 diabetes, Gestational diabetes and Diabetes due to other specific mechanisms or conditions [1, 3, 7].While Type 1 diabetes mellitus is caused by an absolute deficiency in insulin secretion, the Type 2 diabetes is caused by a combination of resistance to insulin action and an inadequate compensatory insulin secretion response and Gestational diabetes occurs in pregnancy [1].

1.1.1 Type 1 Diabetes Mellitus

This is regarded as an auto immune disease and is formerly known as IDDM (Insulin Dependent Diabetes Mellitus) or juvenile-onset. It accounts for 5–10% of the diabetes. Though this immune-mediated diabetes commonly occurs in childhood and adolescence, it can occur at any age. The hallmark of T1DM is the cellular-mediated autoimmune destruction of the beta cells of the pancreas [8]. It has two forms-(i) idiopathic

type1 and (ii) immune-mediated type 1. Markers of the immune destruction of the beta cell include islet cell autoantibodies, autoantibodies to insulin, autoantibodies to GAD (GAD65), and auto antibodies to the tyrosinephosphatases IA-2 and IA-2. [9,10] One and usually more of these autoantibodies are present in 85–90% of individuals when fasting hyperglycemia is initially detected. The autoimmune destruction of beta cells has multiple genetic predispositions as well as environmental factors. Its strong HLA associations, with linkage to the DQA and genes are influenced by the DRB genes. These HLA-DR/DQ alleles can be either predisposing or protective [1, 11]. Depending on individuals the rate of beta cell destruction is quite variable. It can be rapid in some individuals (mainly infants and children) and slow in others (mainly adults). The risk of ketoacidosis at latter stage of the disease when there is little or no insulin secretion is manifested by low or undetectable levels of plasma C-peptide [12]. Patients with this type of diabetes are rarely obese but can also be prone to other autoimmune disorders such as Graves' disease, pernicious anemia, Hashimoto's thyroiditis, Addison's disease and autoimmune hepatitis [13, 14, 1].

1.1.2. Type 2 Diabetes Mellitus

Type 2 Diabetes formerly known as Non-insulin dependent Diabetes Mellitus (NIDDM) or Adult-onset diabetes is more prevalent (90–95% among the diabetics) with an epidemic proportion among the world population [5,15]. It is characterized by chronic hyperglycemia due to inability of pancreatic insulin secretion to regulate body glucose levels. Thus its onset is initiated by increased insulin resistance and progressive deterioration of β-cell function. This type is a major risk infection for many complications with associated cardiovascular morbidity and mortality especially in uncontrolled diabetes but proper glyceamic control reduces the associated diabetic complications and thus the mortality [1,14]. The use of anti-diabetic therapy, like insulin in the treatment of diabetes usually lowers the blood glucose level.

Characteristically there is progressive destruction leading to the dysfunctioning of the beta cells coupled with insulin resistance and altered insulin secretion with resultant chronic hyperglyceamia due to the failure of the damaged beta cells to regulate the glucose level [16]. Thus both pancreatic beta-cell dysfunction (resulting in impaired insulin secretion) and insulin resistance are known to be responsible for Type 2 diabetes [17].

Although the pathogenesis is unknown autoimmune destruction of beta cells does not occur in Type 2 DM. Obesity is common in most patients of this type with increased percentage of body fat distributed predominantly in the abdominal region. This association of obesity and diabetes has led to more type 2 diabetes in women of childbearing age and the number of pregnant women with undiagnosed type 2 diabetes has increased [18]. Unlike in Type 1, ketoacidosis rarely occurs spontaneously in this type of diabetes but infection could trigger such. Due to the gradual development of hyperglycemia this type of diabetes frequently goes undiagnosed for many years with not severe or unnoticed symptoms at earlier stages and such patients have increased risk of developing macro vascular and micro vascular complications. Despite a degree of hyperglycaemia clinical symptoms, may be present for a long period of time before diabetes is detected [1, 2,19,20]. The insulin levels in the patients appear normal or elevated. The risk factors include age, obesity, and lack of physical activity. Type 2 diabetes mellitus occurs more frequently in women with prior GDM and in individuals with hypertension or dyslipidemia with its frequency varying among different racial/ethnic subgroups [21].

1.1.3 Gestational Diabetes

Gestational diabetes mellitus(GDM) is the type that develops during pregnancy and it occurs in about 3-5% of pregnant women, usually in the third trimester but could be as high as 25% based on risks factors among the populations [22,23]. GDM which is on the increase globally is widely believed to be due to "insulin resistance"- a situation where it is hard for the mother's body to make use of insulin. Thus the inability of the pancreas to maintain the blood sugar level at a normal range occurs [24]. Some placental hormones hinder the action of insulin and thus prevent it from controlling blood sugar. The hallmark of GDM is increased insulin resistance with onset or first occurrence during pregnancy [25,26] Usually GDM cases resolve with delivery but there is a high rate of recurrence in subsequent pregnancies. Most cases of GDM are asymptomatic but possible symptoms are blurred vision, increased thirst, fatigue, increased urination, nausea and vomiting ,yeast infections, urinary tract infections etc. Many risk factors identified for GDM include polycystic ovarian syndrome, maternal age, ethnic background, overweight/obesity family history of Type 2 diabetes among others [27,28].

Not only the women with GDM but also their children also have an increased risk for childhood and adult obesity and Type 2 diabetes later in life and therefore adequate and proper treatment is needed to reduce the risks of GDM for both mother and child [29,30].

1.2. Gestational Diabetes and Type 2 Diabetes

Gestational diabetes usually goes away after the delivery of the baby but, however many women go on to develop type 2 diabetes mellitus years later, with the prevalence depending on ethnic background [31,32, 33]. National Diabetes Education Program stated that women who have had GDM are seven times more likely to develop type 2 diabetes than women who have not had GDM in pregnancy and that even the children from such pregnancies affected by GDM may also have a greater risk for obesity and type 2 diabetes [34] The mechanisms in both GDM and Type 2 DM are similar.

In their study Kwak *et al* ,2013[35] noted as many as half of Asian women who had gestational diabetes developing type 2 diabetes within eight years of giving birth.

1.3. Diagnosis Of Diabetes Mellitus

Plasma glucose measurement is ideal for quantitative values. The criteria for the diagnosis of Diabetes mellitus according to WHO guideline is as shown below:

1. Fasting Plasma Glucose ≥126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 hours preferably overnight.
2. 2-hour plasma glucose ≥200 mg/dl (11.1 mmol/l) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.
3. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a Random plasma glucose ≥200 mg/dl (11.1 mmol/l). [36,37]

II. Objectives

In order to improve the understanding of the relationship between GDM and Type 2 DM, this study investigated volunteered pregnant women to establish or otherwise the relationship between gestational diabetes and Type 2 diabetes mellitus.

III. Materials and Methods

a. Materials

Spectrophotometer
Reflotron Chemistry Machine
Dip-stick (Urinalysis) Strips
Floride oxalate anticoagulated bottles
Randox Glucose Kit

b. Methods

Volunteered antenatal patients with an average of two weeks appointments between visits were screened for urine sugar during their routine tests, using urinalysis tests (Dip stick method).

With consents sought, those with glucosuria had Fasting Blood Sugar (FBS) test and Oral Glucose Tolerance Test (OGTT) done on scheduled appointments. Participants had their venous blood drawn for following laboratory tests:

1. Fasting blood glucose test
2. Oral Glucose Tolerance Test(OGTT)

i. Fasting Blood Glucose Test

The glucosuric patients were billed for FBS test.

Venous blood sample was taken for Fasting blood glucose value after about 8hours (overnight) without food and drink.

Those with hyperglycaemia were then booked for OGTT

ii. OGTT Procedure

The OGTT was done for the hyperglyceamic patients in the morning after an overnight fast of at least 8 hours.

75.0 g anhydrous glucose was dissolved in 100mls of clean water and taken by the patient and venous blood sample collected for the plasma glucose measurements at fasting and at 1 hour and 2 hours were performed with the glucose kit following the manufacturer's instructions. Colorimetric kinetic enzymatic assay method involving measuring the absorbance with Spectrophotometer was used.

The diagnosis of GDM was made and established when any of the following plasma glucose values were exceeded:

Fasting: ≥110mg/dl (6.1 mmol/l)

1 hour: ≥180 mg/dl (10.0 mmol/l)
2 hours: ≥153 mg/dl (8.5 mmol/l) .

These values are diagnostic of diabetes mellitus thus all the pregnant patients with the above values were diagnosed of GDM [38] and were then monitored properly by their physicians.

IV. Results

Among the 478 pregnant women screened for diabetes mellitus 36 had glucosuria out of which 33 were hyperglycaemic while the remaining 3out of those with glucosuria had normoglycaemia with FBS ranging between 55 and 90 mg/dl. Those with hyperglycaemia had FBS ranging between 125 and 560 mg/dl.

The total number of antenatal patients that participated in this study is 478 out of which 33(6.9%) had gestational diabetes (Table 1). Only 26(78.8%) of these which were still attending their respective hospitals after a period of six years and 3 months were followed up (Table 2).

8(30%) of them have developed Type 2 diabetes mellitus and 18 are non-diabetic within that period (Table 3).

Table 1. Showing the occurrence of GDM among the Pregnant Women Studied

Category of Women	**No of Women**	**Percentage (%)**
Total Pregnant Women	478	
Total Women with GDM	33	6.9

Table 2. Showing the GDM Women studied (n=33)

GDM Women	**No of Women**	**Percentage (%)**
GDM women available	26	79
GDM Women missing	7	21

Table 3. Showing the occurrence of T1DM among the GDM Women

GDM Women	**No of Women**	**Percentage (%)**
GDM women available	26	100
GDM with T2DM	8	30.8
GDM without T2DM	18	69.2

GDM - Gestational Diabetes Mellitus
T2DM - Type 2 Diabetes Mellitus

V. Discussion and Conclusion

Diagnostic Implication: The study shows a seemly link between gestational diabetes and the tendency to have type 2 diabetes mellitus later in life among the women studied which are basically of Black African ethnicity background thus women with gestational diabetes should be monitored properly even after their deliveries Such monitoring would allow the prevention of perinatal and post natal complications by administering appropriate treatment. Further studies with larger number of subjects would give more accurate prevalence, data and The diagnostic threshold values of GDM varies among different organizations but WHO values were used in this study

One of the limitations is the proper follow up of the patients over the years as many have relocated and probably also changed clinics without proper database to trace them. The long term implications of GDM on maternal and child health cannot be overemphasized.

VI. Inclusion and Exclusion

Known diabetes patients and non-pregnant women were excluded.

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