

## Association between maternal lipid profile and gestational diabetes mellitus

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### ABSTRACT

**Background:** Dyslipidemia is the third component of metabolic syndrome and is a well-known cardiovascular risk factor. However, the association of dyslipidemia with gestational diabetes mellitus is still a subject of ongoing research in Nigerian obstetric populations. **Objective:** To determine the relationship between second trimester maternal fasting plasma lipid constituents and gestational diabetes mellitus. **Methods:** This was a prospective nested case-control study that enrolled 288 pregnant women out of which 36 women with GDM (cases) were matched with 72 without GDM (controls) following results of oral glucose tolerance testing and plasma fasting lipid profiles done between 24-28 weeks. The patients were followed up until delivery to document maternal and fetal outcomes. Data was analyzed using Statistical Package for Social Sciences (SPSS). Categorical variables were presented in percentages while continuous variables were expressed as means ( $\pm$ Standard Deviation). Student t-test and Chi-square test or Fishers exact test were used for comparing variables between the two groups. A p-value of  $<0.05$  at 95% confidence interval was considered statistically significant. **Results:** The overall mean plasma lipid levels for the four lipid constituents in the study population were 187.9mg/dL, 163.5mg/dl, 49.1mg/dL and 108.1mg/dL for TC, TG, HDL-c and LDL-c respectively. The mean plasma triglyceride was significantly higher in cases compared to the controls:  $187.0\pm 67.7$ mg/dL vs.  $151.7\pm 66.4$ mg/dL, ( $p = 0.01$ ). Abnormal triglyceride was significantly associated with GDM (AOR: 4.8, 95% CI (1.6-14.4), ( $p= 0.005$ ). **Conclusion:** Maternal dyslipidemia (abnormal triglyceride) was shown to be significantly associated with GDM in this study and it appeared to be causally related.

**Keywords:** Lipid profile. Pregnancy, Gestational diabetes mellitus, Abuja, Nigeria

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### Introduction

Gestational diabetes mellitus (GDM) is a common medical disorder in pregnancy that is associated with increased perinatal and maternal morbidity and mortality<sup>1</sup>. It is an important disease of global public health importance affecting up to 15% of all pregnancies depending on population characteristics.<sup>2</sup>

Women with uncontrolled GDM have a four-fold increase in perinatal mortality rate compared to controls.<sup>3</sup> They are also at a higher risk of gestational hypertension, preeclampsia, caesarean delivery, and the

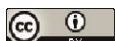
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DOI: 10.31173/bomj.bomj\_189\_17



percentage of such women that would progress to full fledged DM<sup>1</sup> The offspring of women with GDM are at an increased risk of macrosomia, neonatal hypoglycaemia, hyperbilirubinaemia, operative delivery, shoulder dystocia, birth trauma and long term sequelae such as development of type 2 diabetes mellitus and cardiovascular disease in adult life<sup>1,4,5</sup>. Disturbed maternal metabolism, including atherogenic lipid changes in pregnancy, is one of the crucial factors that has been involved in the pathological processes culminating in these adverse outcomes.<sup>6</sup>

In women with GDM, the physiological changes in insulin and lipids are exaggerated and may indicate underlying metabolic dysfunction that transiently manifests during pregnancy.<sup>7</sup>

Lipid parameters, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein-cholesterol (LDL-C), high-density lipoprotein-cholesterol (HDL-C) and phospholipid gradually increase from the 12 week of pregnancy and especially in the second and third trimesters.<sup>6</sup>

Previous studies show that triglycerides are significantly elevated among women with GDM compared with women without diabetes mellitus and this finding persists across all three trimesters of pregnancy.<sup>8,9</sup> Findings related to HDL-C levels on the other hand has not been consistent. While a systematic review and meta-analysis of maternal lipids levels during pregnancy and gestational diabetes revealed that HDL-C levels were significantly lower in women with GDM compared with those without GDM in the second and third trimesters of pregnancy, another study found that an elevated HDL-C especially in the second trimester was associated with a decreased risk of GDM<sup>6</sup>.

There were no differences in aggregate total cholesterol or LDL-C levels between women with GDM and those without insulin resistance.<sup>8</sup>

Despite the increasing relevance of dyslipidemia and its association with GDM, there is paucity of literature relating to this subject in Sub-Saharan Africa and particularly in Nigeria. The prevalence of dyslipidemia and its association with GDM has not been prospectively evaluated in North Central Nigeria.

This study therefore aimed to determine the association between second trimester maternal fasting plasma lipid levels (TC, HDL-c, LDL-c and TG) and gestational diabetes mellitus in an obstetric population in North Central Nigeria.

## Method and materials

### Study design /setting

This was a prospective nested case-control study involving pregnant women who booked and attended the antenatal clinic (ANC) of the University of Abuja Teaching Hospital, Gwagwalada, Abuja.

The hospital is a 350-bed Federal Government owned tertiary institution situated in Gwagwalada, a high population density area in Abuja, Nigeria's Federal Capital Territory. It provides health care services to the inhabitants of Abuja as well as serving as a referral centre for primary, secondary and tertiary health facilities within the Federal Capital Territory and four neighbouring states.

### Enrolment of participants

Consecutive pregnant women who met the inclusion criteria and gave their consent were recruited to take part in the study. These criteria include: age  $\geq 18$  years, singleton pregnancies, estimated gestational age  $\leq 28$  weeks and negative past history of GDM.



Women with pre-gestational DM (type I and II), hypertensive diseases of pregnancy, multiple gestation were excluded from the study. Written informed consent was obtained from eligible participants before being enrolled in the study. Structured interviews with the aid of a proforma were conducted at first contact to collect information on socio-demographic characteristics, medical and obstetric history of each participant. Gestational age was determined on best available estimate; either by using the patient's last menstrual period (LMP) or estimation of gestational age from ultrasound scan. They were seen at the ANC and given appointment for their fasting plasma lipid profile and oral glucose tolerance test (OGTT) which were done on the same day between 24 to 28 weeks gestational age during their regular antenatal care follow up.

A total of 288 women were enrolled, out of which 38(13.2%) were diagnosed as having GDM following OGTT. Seventy-six (76) GDM negative women were selected from the remaining cohort as controls based on matching criteria for date of sample collection, using a ratio of 1:2 for the cases and controls respectively. These 114 women (38 cases and 76 controls) were followed up until delivery for determination of fetomaternal outcomes as secondary outcome measures. 5/114(4.4%) delivered outside the hospital and were lost to follow up. Thus 109 participants completed the study and had materno-fetal outcomes. The flow chart of participants involved in the study is as shown in figure 1.

#### Data Collection Methods

##### Blood pressure measurement

The maternal blood pressure was measured using a mercury sphygmomanometer (model Riester Desk sphygmomanometer, Rudolf

Riester GmbH Germany) and stethoscope (Littman's Cardiology III<sup>®</sup>). Measurements were taken from the right upper arm in a sitting position in accordance with the recommendation of the International society for the study of hypertension in pregnancy.<sup>12</sup>

##### Oral glucose tolerance test (OGTT)

We used WHO criteria for GDM diagnosis<sup>13</sup>. The 75-g OGTT was done at 24 to 28 weeks gestational age using Glucose Oxidase Method and the diagnosis of GDM was made when one or more of the following thresholds were met or exceeded:

- Fasting plasma glucose = 5.1-6.9 mmol/l (92 -125 mg/dl)
- 1-hour post 75g oral glucose load =  $\geq$ 10.0 mmol/l (180 mg/dl)
- 2-hours post 75g oral glucose load = 8.5 - 11.0 mmol/l (153-199 mg/dl)

##### Lipid Profile Assay

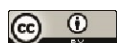
A. Total Cholesterol was assayed using the Modified Liebermann-Burchard reaction.

B. High Density Lipoprotein Cholesterol (HDL-C) was assayed using the Phosphotungstic acid and magnesium chloride method

- Triglycerides (TG) was estimated using glycerol phosphate oxidase method D. Low density lipoprotein cholesterol (LDL-C) was subsequently calculated using quantitative measurements of total cholesterol and HDL-C and plasma TG using the empirical relationship of Friedewald's formula:

$LDL-C \text{ in mg/dl} = TC - (HDL-C + TG \text{ in mg/dl} / 5 \text{ OR } TG \text{ in mmol/l} / 2.2)$ .

Dyslipidemia (DLP) in pregnancy in the general population has been defined by both the National Cholesterol Education Programme/Adult Treatment Panel III (NCEP/ATP III)<sup>14</sup> and WHO<sup>15</sup> as a state that arises as a result of abnormalities in the



plasma lipids (increased TC, TG, LDL-c and/or decreased HDL-c occurring either singly or in combination). On this basis, the agreed cut offs are: TC: 150 - 220mg/dl, TG: 40 - 140mg/dl, HDL-c: 45 - 65mg/dl, LDL-c: >100 mg/dl.<sup>14</sup>

In pregnancy, there are no agreed standardized definitions of DLP, and there is paucity of data regarding DLP in pregnant women globally. However, the percentile criteria using values obtained in pregnancy by semester was employed by Knopp et al.<sup>16</sup> This is when there is an elevation of TC, TG, and LDL-concentrations above the 95<sup>th</sup> percentile and HDL-c below the 5<sup>th</sup> percentile for that gestational age. Also, in pregnancy, Feitosa et al<sup>17</sup> defined DLP as when TC  $\geq 200$ mg/dL, TG  $\geq 150$ mg/dL, LDL-c  $\geq 160$ mg/dL and HDL-c  $\leq 50$ mg/dL occurring either singly or in combination.

The definition of DLP using the NCEP/ATP III<sup>14</sup> cut offs is the most current and frequently referenced diagnostic criteria for DLP because of its cardiovascular risks' stratification, and were thus used for this study.

#### 4.7. Ethical considerations

Approval for the conduct of the study was granted by the University of Abuja Teaching Hospital Health Research and Ethics Committee (Approval number: FCT/UATH/HREC 1035). Clients' voluntary participation, confidentiality, beneficence, non-maleficence, justice and dignity were maintained.

#### Data analysis

Data analysis was done using Statistical Packages for Social Sciences (SPSS) version 21 (IBM SPSS Armonk, NY: IBM Corp). Categorical variables were presented in percentages while continuous variables were expressed as means ( $\pm$ Standard Deviation).

Student *t*-test and Chi-square test or Fisher's exact test were used for comparing variables between the two groups. Associations of TC, TG, HDL-c and LDL-c with GDM were assessed by logistic regression. A p-value of <0.05 at 95% confidence interval was considered statistically significant.

#### Results

Overall, 38/288 of women had OGTT results diagnostic of GDM, giving a prevalence of 13%, while 86% of the study population had DLP either singly or in combination for any of the lipid constituents. The respective prevalence of DLP for TC, TG, HDL-c and LDL-c were 18.8%, 56.0%, 36.0% and 59.7% respectively.

The baseline characteristics were similar for both groups except for age, weight and systolic blood pressure (Table I).

Comparison of risk factors for GDM in the cases and controls showed that previous history of fetal macrosomia and maternal weight of  $\geq 90$ kg were significantly associated with the occurrence of GDM ( $p = <0.001$  and  $p=0.003$  respectively), (Table II).

The overall mean plasma lipid levels for the four lipid constituents in the study population were 187.9mg/dL, 163.5mg/dl, 49.1mg/dL and 108.1mg/dL for TC, TG, HDL-c and LDL-c respectively. Comparing the two groups, both had fairly similar fasting plasma concentration for all the lipid constituents, except triglyceride (TG), which was higher in the cases than the control group as graphically, represented in figure 2 ( $187.0 \pm 67.7$ mg/dL compared with  $151.7 \pm 66.4$ mg/dL in the controls,  $p = 0.010$ ). Based on normal and abnormal plasma concentrations of the different lipid constituents using defined cut-off values, abnormal plasma TG was significantly associated with GDM (OR = 3.7, 95% CI (1.5-



8.5),  $p$  value = 0.003). With adjustment for several confounders the odds increased (AOR :4.8, 95% CI (1.6-14.4). The statistical significance persisted even after adjusting for these confounders ( $p$  = 0.005). Other lipid constituents ((Tc: AOR :2.2, 95% CI (0.9-5.4), HDL-c: AOR :1.2, 95% CI (0.5-3.2)) were associated with higher adjusted odds for GDM. However, these differences were not statistically significant. LDL-c revealed a lesser adjusted odd for development of GDM (AOR :0.4, 95% CI (0.1-0.9),  $p=0.330$ (Table III). Overall, the secondary maternal outcomes in the study population were preterm birth (7.3%), primary post-partum haemorrhage (1.8%) and preeclampsia (6.4%). Neonatal outcomes including macrosomia, birth asphyxia and shoulder dystocia were

recorded in 14.7%, 1.8% and 0.9% respectively.

When compared with the controls, macrosomia was significantly higher among babies delivered by women with GDM (OR;8.3, 95% C I (2.5-28.1), ( $p$  = < 0.001). Women with GDM also had higher odds of having postpartum haemorrhage, preeclampsia and birth asphyxia (OR; 2.0, 95% C I (0.1-33.3), (OR; 1.5, 95% C I (0.3-7.3) and (OR; 2.0, 95% C I (0.1-33.3) respectively, but these were not statistically significant. Conversely, women with GDM had lesser odds of having preterm births (OR; 0.6, 95% C I (0.1-3.3), Table IV.

The mean neonatal birth weight was  $3.545\pm 0.682$ kg and  $3.075\pm 0.575$ kg for the cases and controls respectively ( $p=0.001$ ).

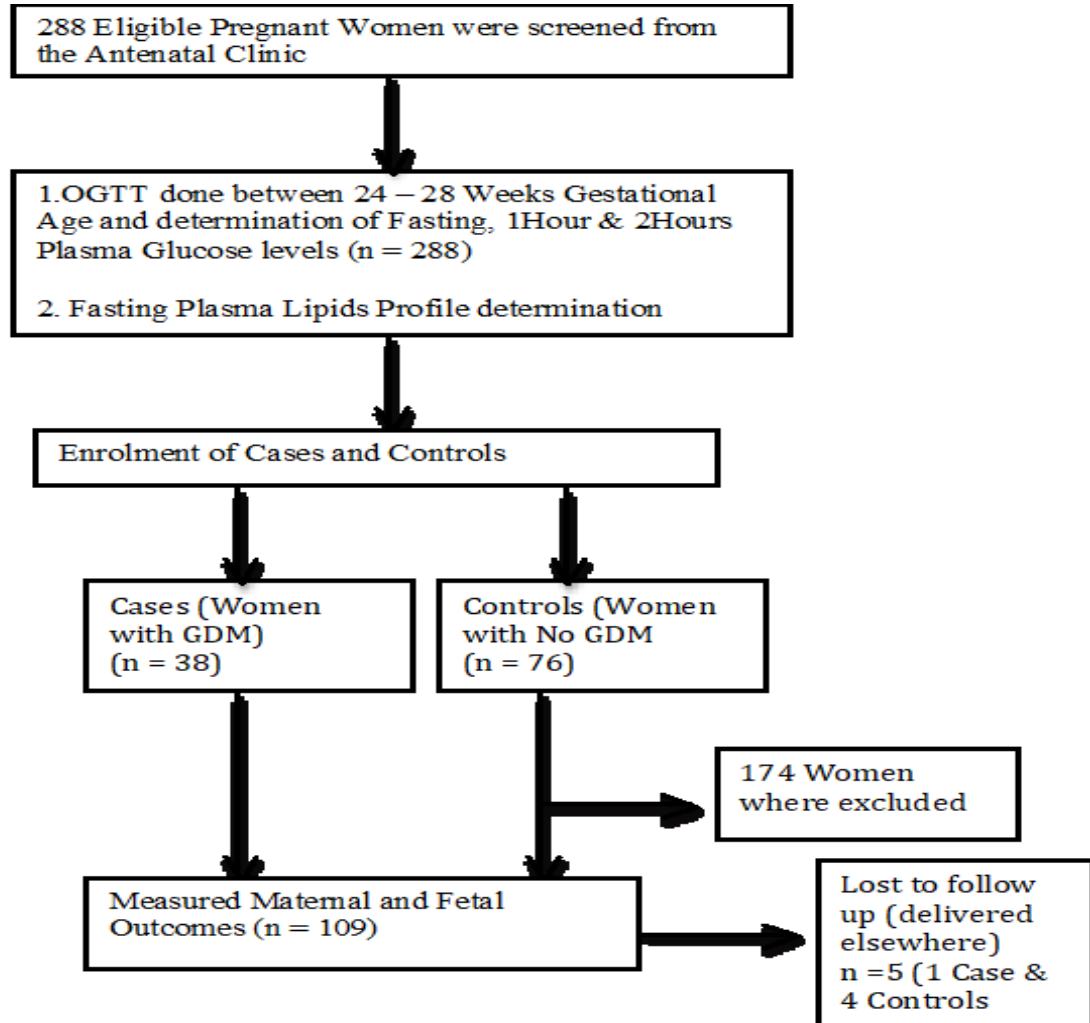


Figure 1: Flow Chart of participants involved in the Study

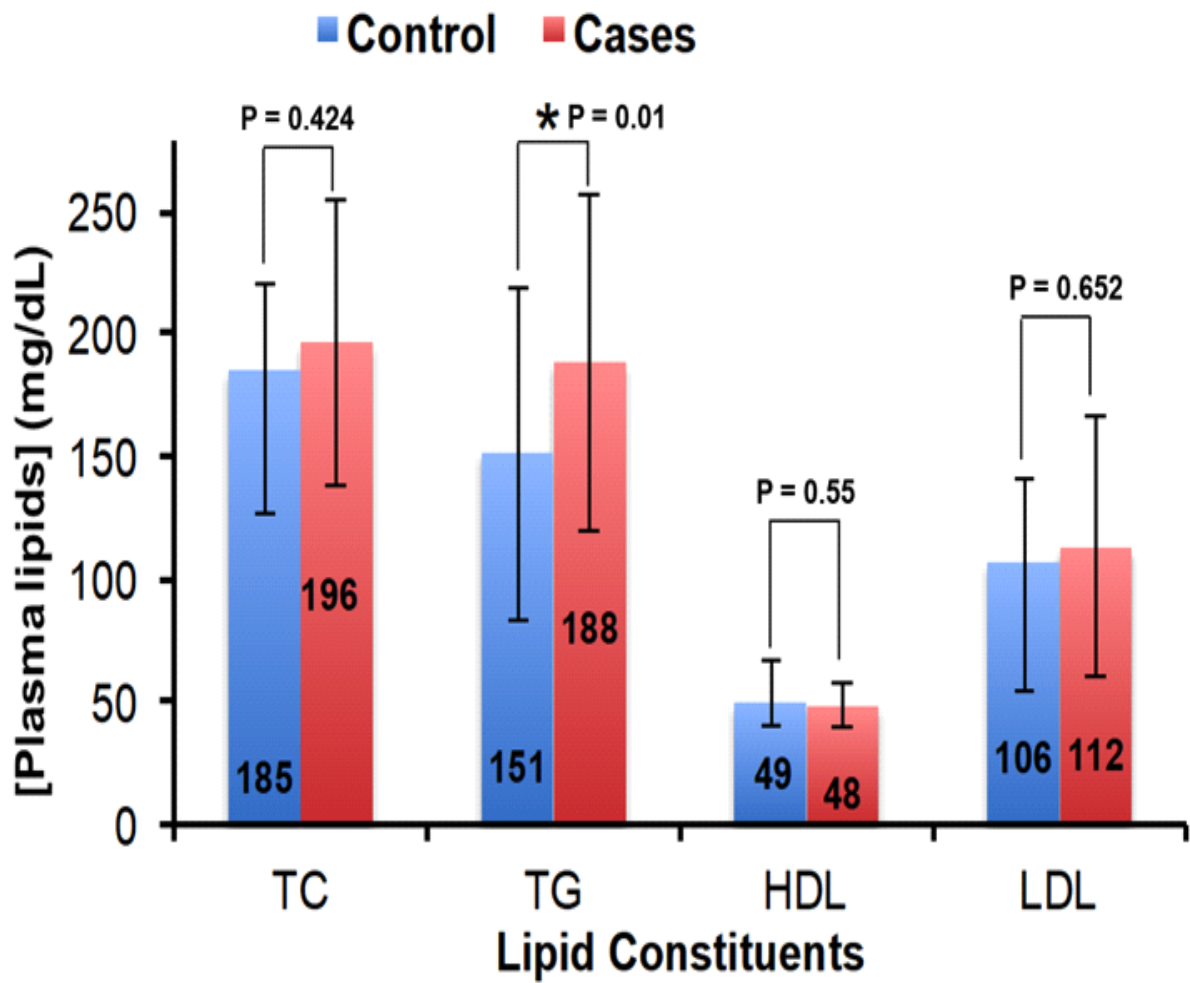


Figure 2: Mean Maternal Plasma Lipid Levels in Women with GDM and Controls

**Table 1:** Characteristics of the Study Population

Characteristics	Cases (GDM)	Controls (No GDM)	Chi-square	P value
	n = 38 Mean ±SD or n (%)	n = 76 Mean ±SD or n (%)		
<b>Age group</b>	32.3±5.1 <sup>#</sup>	29.9±5.0 <sup>#</sup>	2.396 <sup>§</sup>	0.019
≤19	0(0.0)	1(1.3)	0.504	0.478 <sup>β</sup>
20-24	2(5.3)	12(15.8)	2.606	0.137
25-29	12(31.6)	23(30.3)	0.021	0.886
30-34	10(26.3)	25(32.9)	0.515	0.473
≥35	14(36.8)	15(19.7)	3.908	0.048
<b>Education</b>				
No Formal	4(10.5)	11(14.5)	0.345	0.770 <sup>β</sup>
Secondary	10(26.3)	17(22.4)	0.218	0.640
Tertiary	24(63.2)	48(63.2)	0	1.000
<b>Ethnicity</b>				
Hausa	0(0.0)	1(1.3)	0.504	0.478 <sup>β</sup>
Igbo	11(28.9)	20(26.3)	0.089	0.766
Yoruba	5(13.2)	15(19.7)	0.758	0.384
Others	22(57.9)	40(52.6)	0.283	0.595
<b>Parity</b>	2±1.3 <sup>#</sup>	2±1.2 <sup>#</sup>	1.572 <sup>§</sup>	0.120
Primiparous	16(42.1)	46(60.5)	3.465	0.063
Multiparous	21(55.3)	28(36.8)	3.508	0.061
Grand multiparous	1(2.6)	2(2.6)	0(0.0)	1.000 <sup>β</sup>
<b>GA (Weeks)</b>	38.2±1.7 <sup>#</sup>	38.3±1.9 <sup>#</sup>	0.292 <sup>§</sup>	0.771
<b>Weight (kg)</b>	85.5±20.8 <sup>#</sup>	71.4±15.3 <sup>#</sup>	3.704 <sup>§</sup>	0.001
<90	23(60.5)	66(86.8)	10.247	0.001
≥90	15(39.5)	10(13.2)	10.247	
<b>Blood Pressure (mmHg)</b>				
Systolic BP	113.9±17.3 <sup>#</sup>	107.1±11.2 <sup>#</sup>	2.215 <sup>§</sup>	0.031
Diastolic BP	69.5±11.4 <sup>#</sup>	66.6±8.4 <sup>#</sup>	1.390 <sup>§</sup>	0.170

<sup>#</sup>mean± Standard Deviation      <sup>§</sup>t- test      <sup>β</sup> Fishers exact





**Table 2:** Risk Factors for Gestational Diabetes Mellitus

Risk Factors	Cases (GDM)	Controls (No GDM)	P value
	n = 38 n (%)	n = 76 n (%)	
First degree relative	5(13.2)	9(11.8)	0.840
Previous GDM	2(5.3)	2(2.6)	0.600
Previous Macrosomia	13(34.2)	5(6.6)	<0.001
Intra uterine fetal death	2(5.3)	1(1.3)	0.257
Early neonatal death	0(0.0)	1(1.3)	0.478
Congenital Anomaly	1(2.6)	1(1.3)	0.614
Maternal weight ≥ 90kg	15(39.5)	10(13.2)	0.001

**Table 3:** Risk of GDM Stratified Against Plasma Lipid Constituents

Plasma Lipids	GDM	No GDM	OR (CI)	P value	AOR	P value
	n = 38 n (%)	n = 76 n (%)				
Total cholesterol			2.2(0.9-4.8)	0.058	2.2(0.9-5.4)	0.097
Normal	19(50.0)	52(68.4)				
Abnormal	19(50.0)	24(31.6)				
HDL-c			0.9(0.4-2.1)	0.891	1.2(0.5-3.2)	0.690
Normal	24(63.2)	47(61.8)				
Abnormal	14(36.8)	29(38.2)				
LDL-c				0.347	0.4(0.1-0.9)	0.330
Normal	18(47.4)	29(38.2)	0.6(0.3-1.5)			
Abnormal	20(52.6)	47(51.8)				
Triglycerides			3.7(1.5-8.5)	0.003	4.8(1.6-14.4)	0.005
Normal	10(26.3)	43(56.6)				
Abnormal	28(73.7)	33(43.4)				

OR- Odd Ratio    CI-confidence Interval    AOR- Adjusted Odds Ratio

*\*Adjusted for Age, Weight, Ethnicity, Educational status, History of previous GDM, first degree relative with DM, previous macrosomic baby, previous IUID, History of baby with congenital anomaly*



**Table 4:** Secondary Maternal and Neonatal Outcomes for Cases and Controls

Outcomes	GDM	No GDM	OR(CI)	P-value
	n=37 n(%)	n=72 n(%)		
Preterm Birth (PTB)	2(5.4)	6(8.3)	0.6(0.1-3.3)	0.714 <sup>β</sup>
Primary Postpartum Haemorrhage	1(2.7)	1(1.4)	2.0(0.1-33.3)	1.000 <sup>β</sup>
Preeclampsia	4(10.8)	3(4.2)	1.5(0.3-7.3)	0.684 <sup>β</sup>
Macrosomia	12(32.4)	4(5.5)	8.3(2.5-28.1)	<0.001 <sup>β</sup>
Birth Asphyxia	1(2.6)	1(1.3)	2.0(0.1-33.3)	1.000 <sup>β</sup>
Shoulder Dystocia	1(2.6)	0	-	1.000 <sup>β</sup>
Hypoglycaemia	2(5.4)	0	-	0.442 <sup>β</sup>
Birth Trauma	1(2.6)	0	-	1.000 <sup>β</sup>

<sup>β</sup>Fisher's exact test OR- Odd Ratio CI-confidence Interval

### Discussion

The overall prevalence of GDM in this study was 13%. This finding is consistent with the wide range prevalence of 1-16% found globally,<sup>5,18-21</sup> and comparable to a prevalence of 13.9% reported by Kuti et al in Ibadan.<sup>22</sup> Lower prevalence of 5.4% and 3.3% were reported by Adegbola et al<sup>21</sup> and Ugege et al<sup>20</sup> respectively in Lagos and Uyo, Nigeria. The prevalence of 13% in this study compared to the low prevalence in the latter studies may be attributable to the use of the “new” 2013 WHO diagnostic criteria<sup>13</sup> which employs lower cut off values, allowing more women to be diagnosed with GDM compared to the 130mg/dl (7.2mmol/L) cut off using 50g glucose as obtained in the study by Adegbola et al<sup>21</sup> as well as the use of a cut off of  $\geq 7$ mmol/l and/or 2 h post 75 glucose value of  $\geq 7.8$ mmol/l for diagnosis of GDM in the study by Ugege and colleagues.<sup>20</sup>

The overall mean plasma lipid levels for all the lipid constituents except for triglycerides (TC: 187.9 vs 211.2  $\pm$  88.2, TG: 163.5 vs 215.8  $\pm$  327.8, HDL-c: 49.1vs 53.7  $\pm$  15.4, LDL-c: 108.1 vs 114.9  $\pm$  43.6) were comparable to previous findings for lipid levels in the second trimester of pregnancy.<sup>17</sup>

The overall prevalence of DLP of 86% reported in this study is similar to the

prevalence of 83.8% reported in the 2<sup>nd</sup> trimester of pregnancy by a study in Brazil that assessed women’s lipid profiles in all the trimester using both the percentile criteria and a National guideline.<sup>17</sup> Our study had similar patients characteristics the aforementioned study which could be an explanation for the comparable findings.

The prevalence of 56.0% for TG alone was much higher than 34.2 % obtained in the second trimester but much more comparable to third trimester value of 64.7% in the study by Feitosa et al.<sup>17</sup>

Plasma TG is the only major lipid constituent affected by diet.<sup>23</sup> Therefore, similar prevalence for TG in both studies may be explained by the fact that both populations of women are of African descent and may have similar diets. Other plausible explanations for the similarities is the fact that pregnancy is a hyper estrogenic state, and estrogen is the principle modulator of hypertriglyceridemia.<sup>24</sup> Previous studies have also showed that the most dramatic change in lipid profile is serum TG.<sup>25</sup>

The mean age of the women with GDM was higher than the mean age of the women without GDM. This finding is consistent with the fact that GDM is associated with



increasing maternal age<sup>1,5, 18</sup> and comparable with mean age of 32.0 years and 32.1±5.8 reported by Kuti et al<sup>22</sup> and Atiba et al<sup>28</sup> respectively. A significantly higher proportion of older women ≥35 years in the GDM group was also comparable to report by Bener et al<sup>18</sup>.

Abnormal triglyceride level using defined cut off values significantly correlated with increased odds of development of GDM (OR 3.7, CI 1.5-8.5,  $p = 0.003$ ). This persisted even after adjusting for multiple confounders (AOR 4.83, CI 1.62-14.40,  $p = 0.005$ ). This finding is in agreement with several studies suggesting varying degrees of associations between DLP (consistently for TG) and GDM.<sup>6,8,11,25-27</sup> A meta-analysis<sup>8</sup> of sixty studies concluded that TG is significantly elevated all through pregnancy, but more so in the third trimester among women with GDM than those without, this is similar to the findings in our study. Our study finding regarding TG is however at variance with reports by Atiba et al<sup>28</sup> in Nigeria where no statistically significant difference in TG was seen between the two groups. While a population-based study in China<sup>6</sup> reported an association between maternal HDL-C concentrations and GDM, our study showed no difference between GDM and non GDM patients. It is also at variance with another report<sup>8</sup> which showed that HDL-C levels were significantly lower in women with GDM compared with those without GDM in the second and third trimesters of pregnancy. The inconsistencies in these findings suggests the need for more elaborate studies relating to dyslipidemia and gestational diabetes mellitus in Nigeria and the rest of the world.

Preterm birth though not statistically significant, was lower among women with GDM (5.4% vs. 8.3%,  $p = 0.714$ ). This however is not consistent with the general conviction

that GDM is associated with increased risk of preterm birth. The plausible explanation for the findings in this study is the fact that the preterm births were not spontaneous, and may be due to other obstetric causes such as premature rupture of membranes, preeclampsia, and antepartum haemorrhage. Development of preeclampsia as a secondary outcome in this study, though not statistically significant, was also surprisingly more common among those women without GDM (7.9% vs. 5.3%,  $p = 0.684$ ). This is also contrary to the popular association between GDM and Preeclampsia.<sup>6,25-27</sup> Be that as it may, preterm births and preeclampsia have all been reported to be associated with varying degrees of dyslipidaemia.<sup>6,8,27</sup> This association was however not tested in this study.

Macrosomia was statistically significantly higher in women with GDM, than those without GDM (31.6% vs. 5.3%  $p = <0.001$ ). This is consistent with the fact that macrosomia is an immediate consequence of GDM<sup>1, 5,30</sup> and a cluster of metabolic alterations associated with maternal obesity drives fetal overgrowth.<sup>5, 28</sup> It is also the most consistent and easily measurable sequelae of GDM.<sup>21, 31</sup>

#### Conclusion and Recommendations

Results from this study showed a high prevalence of GDM among pregnant women in Nigeria's Federal Capital Territory. Of all the four lipid constituents studied, only abnormal triglyceride was independently and significantly associated with GDM. Additionally, women with GDM had a significantly increased risk of fetal macrosomia.

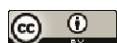
Lipid profiles estimation during pregnancy, particularly triglycerides can be a useful tool in identification of women at risk of GDM towards prompt management and

prevention of adverse pregnancy outcomes. The inclusion of lipid profile estimation as adjunctive investigations for identification of

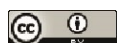
women at risk of GDM should be considered during antenatal care.

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**Cite this Article as:** Jibrin BI, Akaba GO, Isah AY, Dalili MS, Ekele BA. Association between maternal lipid profile and gestational diabetes mellitus. *Bo Med J* 2020; 17(1): 1-5 **Source of Support:** Nil, **Conflict of Interest:** None declared

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