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Research Article

Association Between Age, Body Mass Index, and Lipid Profile in Women with Gestational Diabetes Mellitus in Nigeria

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Abstract

Background: Gestational diabetes mellitus (GDM) is a pregnancy-related metabolic disorder characterized by glucose intolerance and insulin resistance. It is associated with dyslipidemia and increased risk of adverse maternal and fetal outcomes. Understanding the relationship between risk factors such as age, body mass index (BMI), and lipid profile alterations is essential for improved clinical management.
Methods: A hospital-based case–control study was conducted involving 160 women aged 18–40 years at 24–28 weeks of gestation. Participants included 60 women with GDM, 50 non-diabetic pregnant controls ($Control_1$), and 50 non-diabetic non-pregnant controls ($Control_2$). Subjects were recruited from selected tertiary hospitals in Kaduna State, Nigeria. GDM screening was performed using a 50 g oral glucose challenge test (OGCT) followed by a 75 g oral glucose tolerance test (OGTT). Approximately 5 mL of venous blood was collected for biochemical analysis of lipid profile parameters using standard laboratory methods. Data were analyzed using appropriate statistical tests, with significance set at $p < 0.05$.
Results: Total cholesterol (TC) levels were significantly elevated in GDM and $Control_1$ groups compared to $Control_2$ in OGCT samples ($p < 0.05$). In OGTT (2-hour post-load) analysis, TC was unexpectedly higher in $Control_2$ than in GDM subjects. Triglyceride (TG) levels were significantly increased in GDM and $Control_1$ groups in fasting and 2-hour OGTT samples compared to $Control_2$ ($p < 0.05$). No significant differences were observed in TG levels in OGCT samples. Low-density lipoprotein cholesterol (LDL-C) was consistently higher in GDM subjects compared to both control groups across OGCT and OGTT assessments. Overall, lipid profile alterations were more pronounced in GDM, although patterns varied across test modalities.
Conclusion: Women with gestational diabetes mellitus exhibit significant dyslipidemic changes, particularly elevated LDL-C and triglycerides, which may contribute to increased cardiometabolic risk. Age and body mass index likely modulate these lipid alterations, underscoring the importance of early metabolic screening and risk stratification in pregnancy.

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1. Introduction

Insulin secretion and/or action is impaired in diabetes mellitus (DM), resulting in hyperglycemia [1]. However, 1979 and 1980 are the years in which the second Committee on Diabetes of the World Health Organization (WHO) brought order to the assortment and tests used to diagnose DM, then in 1985 also, they did some adjustments to this tests used to diagnose diabetes mellitus [2].

It is estimated that there will be an additional 165 percent rise in diabetes by 2050 if the diabetes incidence increases by 25% in 2030 and 51% in 2045 [3]. Furthermore, it has been projected that 33% of boys and 39% of girls that were given birth to after the year 2000, are likely to develop DM during their life time. This projection was a product of the current obesity epidemic (diabesity) in the United States and some other countries as well, this increase is primarily caused by Type 2 diabetes Mellitus [4].

Among a given ethnic group or population, GDM prevalence varies directly with age [5]. It has been reported that, GDM affects 3% - 10% of pregnancies, depending on the population studied [6]. Among women aged 20–49 years, hyperglycemia is prevalent at 16.9% globally, and in low- and middle-income countries, an estimated 90% of cases occur in pregnancy [7]. There are approximately 14% of pregnancies worldwide who undergo gestational diabetes every year, so identifying them and keeping them under strict metabolic control is essential to prevent immediate and long-term problems for the mothers and their children [8].

A significant increase in GDM prevalence among Indians compared to 2002 (2.1% to 4.1%) was reported, whereas a prevalence of 1.7% was recorded in the Nigerian population [9]. A study in Aba revealed the prevalence of GDM to be 2.2% in 2013 but by 2015 the prevalence has increased to 6.2% [10]. Furthermore, in Zaria North West Nigeria, the prevalence of GDM was revealed to be 3.4% to 13.4% [11].In many communities, however, help is often lacking for those with gestational diabetes and so a good management of their condition is critical to a positive pregnancy outcome.

2. Methods

Study Area

The study area is Kaduna, the capital of Kaduna State. The state is located at the Northern part of Nigeria's High Plains. The vegetation cover is Sudan Savannah type, characterized by scattered short trees, shrubs and grasses. The soil is mostly loamy to sandy type. A substantial amount of clay is found also Kaduna State consists of twenty-three (23) Local Government Areas. It consist of a total of 46,053 km^2 (17,781 sq meter) Area rank 4^{th} of 36. Its population (2006 census) recorded a total of 6,113,503, rank 3^{rd} of 36. Figure 1 showed the Location of Kaduna State in Nigeria Coordinates: 10°20′N 7°45′E

Figure 1: Location of Kaduna State in Nigeria Coordinates: 10°20′N 7°45′E (Olaghere, 2022)

Figure 2 showed the Map of Kaduna State showing all the Local Government Areas.

Figure 2: Map of Kaduna State showing all the Local Government Areas. (Kdsg.gov.ng)

Study Design

This is a case-controlled study, in which a total of 160 subjects between the ages of 18- 40 years were recruited for the study. However, 60 subjects were gestational diabetic women, 50 non-diabetic pregnant women as $Control_1$ all attending Antenatal clinics at Barau Dikko Teaching Hospital, Yusuf Dantsoho Memorial Hospital and Gwamna Awan General Hospital Kaduna State, while 50 apparently healthy non-pregnant non diabetic staff as $Control_2$. Arrangements were made with the clinicians whereby subjects who satisfy the study inclusion criteria were selected. Structured questionnaires (Appendix 1) were administered to the study population. Vital information including the name, age, ethnic group, height, weight, blood pressure and pregnancy induced complications were obtained through personal interview followed by blood sample collection.

Inclusion Criteria

All pregnant women within the age of 18–40 years, permanent residents of Kaduna Metropolis, within 24–28 weeks of gestation according to the WHO diagnostic criteria, who give their consent and underwent GDM universal screening, were included in the study. Women who are non- hypertensive and who agreed to participate were also included in the study, were also included. Apparently healthy aged matched non-GDM pregnant women as well as apparently healthy non-pregnant- non-Diabetic women too were included in the study as $Control_1$ & $Control_2$ respectively.

Exclusion Criteria

Primigravidas, Obesse subjects, women who are $\leq$ 18 or $\geq$ 45 years, pregnant women with chronic hypertension, multiple gestation, pre-eclamsia and eclamsia were excluded in the study. All those who personally declined to give consent for inclusion were also excluded from the study.

Informed Consent

Informed written consent was obtained from all subjects before inclusion using approved protocol given by the Kaduna State Ministry of Health ethical committee (appendix II).

Ethical Approval

Ethical approval of the study was obtained from the Ethics Committees of the Kaduna State Ministry of Health in accordance with Helsinki declaration.

Sample Size Determination

The sample size would be determined from a standard formula (Kyriazos, 2018).

$$n = \frac{(Z_1 - a)^2 \times P \times (1 - P)}{d^2}$$

Where,
n= minimum sample size

$Z_1 - a$ = value of standard normal deviation which at 95% confidence level has been found to be 1.96
P = the best estimate of the population prevalence obtained from literature review (3.4%)
d = difference between the true population rate and sample that can be tolerated, that is the absolute precision required (in percentage point) on either side of the population i.e. degree of confidence = 0.05

$$n = \frac{(3.8416)(0.034)(0.9966)}{0.0025}$$

n= 51
Therefore, a total of 51 with 10% (5) of these subjects will be added to the research for attrition making a total of 56 total subjects but for the purpose of these study 60 samples shall be used.

Sampling Techniques

Sampling

Arrangement was made with the clinicians where those subjects who satisfied the study inclusion criteria were selected. A consecutive sampling method in which subjects meeting the criteria for inclusion, were continuously selected until the desired sample size was obtained. Random 50 g oral glucose challenge test was carried out followed by fasting 75 g OGTT were performed in pregnant women between 24 and 28 weeks of gestation representing GDM and C_1. Samples were also collected from apparently healthy non-pregnant, non-Diabetic staff and Students that served as control (C_2). Diagnosis of GDM was established according to the diagnostic criteria of the American Diabetes Association 2015. Findings of the blood samples collected from all subjects were fully documented in the proforma (Appendix I). Assessments such as blood pressure (BP), weight (W), height (H) and body mass index (BMI) were also observed.

Specimen Collection and Processing

Blood specimen (5 ml) for the biochemical measurements, was collected from peripheral vein (antecubital venepuncture). This was done by cleaning the antecubital fossa with methylated spirit and with the application of a tourniquet a few centimeters above the anticubital fossa to distend the veins, an overnight fasting sample of 5 ml venous blood was drawn from each subject and dispensed into plane containers. The coagulated whole blood was centrifuged at 1,000 rpm (Revolution Per minute) for 15 minutes, within 30 minutes of collection. The serum was removed, transferred to Bijou bottles. The samples for glucose, antioxidants, Vitamins C was analyzed immediately. Specimens for other parameters that would not be assayed within 24 hours of collection were stored frozen at -80°C until the time for analysis.

Equipment

Hettich Universal 32 Centrifuge (Germany) was used to spin blood samples (Appendix IV). Beckman Coulter DU-520, general purpose UV/VIS Spectrophotometer (Germany) was used for the measurements of serum glucose & Bio-rad PR 5100 microplate reader was used for measurements of Adiponectin.

Determination of Serum Total Cholesterol (TC)

Serum total cholesterol was measured using the method of Young et al, 1975; [12].

Principle

Under the action of cholesterol esterase (CHE) cholesterol esters are hydrolysed into cholesterol and fatty acids. The cholesterol formed is oxidized by cholesterol oxidase (CHOD) to cholesterol-3-one and hydrogen peroxide. In the presence peroxidase (POD), the released hydrogen peroxide reacts with a phenol substitute and 4-aminoantipyrine to form a red dye compound. The intensity of the red colour produced is directly proportional to the total cholesterol in the sample when read at 510 nm.

Procedure

Three test tubes were prepared and labeled as blank, standard and test. Into the test tubes, 1000 μl of working reagent will be pipetted. Cholesteol standard, 10 μl and sample, 10μl was added into the tubes labeled standard and test respectively. The contents were mixed and read against reagent blank at 500 nm. The results were calculated thus:

$$\text{Cholesterol concentration (mmol/L)} = \frac{\text{OD of sample}}{\text{OD of standard}} \times 5.09 \text{ mmol/L}.$$

Determination of Serum Triglycerides

Serum triglyceride was measured using the method of Fossab and precipe (1982) (Loomba, 2018).

Principle

Triglyceride (TG) is determined after enzymatic hydrolysis with lipase. Triglycerides are hydrolysed by lipase to glycerol and free fatty acid (FFA). The glycerol reacts with adenosine triphosphate (ATP) to form glycerol-3-phosphate and adenosine diphosphate (ADP) under the

influence of glycerol kinase (GK). glycerol-3-phosphate is oxidised by glycerol phosphate oxidase (GPO) to dihydroxyacetone phosphate and hydrogen peroxide. Hydrogen peroxide is then combined with 4-aminoantipyrine to form quinoneimine (coloured compound), the intensity of which is proportional to the concentration of TG present in the sample. The coloured complex is read at 500 nm.

Procedure

Three test tubes were prepared and labeled as blank, standard and test. Into the test tubes, 1000 μl of working reagent was pipetted. Triglycerides standard, 10 μl and sample, 10μl will be added into the tubes labeled standard and test respectively. The contents were mixed and against reagent blank at 505 nm. The results were calculated thus:

$$\text{Triglycerides concentration (mmol/L)} = \frac{\text{OD of sample}}{\text{OD of standard}} \times 2.23 \text{ mmol/L}.$$

Determination of Serum High Density Lipoprotein Cholesterol (HDL-C-C)

Serum HDL-C-cholesterol was measured using Young D.S. et al method (1975) (Pencina, 2019)

Principle

When serum is combined with the polyethylene glycol reagent (PEG 6000), all the beta lipoproteins, very low density lipoproteins (VLDL-C) and low density lipoproteins (LDL-C) are precipitated. The HDL-C fraction (alpha-fraction) remains in the supernatant is then treated as a sample and assay for cholesterol by an enzymatic red dye method.

Procedure

One precipitating tube was prepared and labeled as test. Into the tube, 500 μl of precipitating reagent (reagent A) was pipette and 500 μl of sample was added. The contents were mixed and incubated at room temperature for 5 minutes. This was then centrifuged at 3000 RPm for 10 minutes. The supernatant was collected and treated as a sample and assay for cholesterol.

Three test tubes were prepared and labeled as blank, standard and test. Into the test tubes, 1000 μl of working reagent was pipetted. Cholesterol standard, 25 μl and supernatant, 25μl was added each into the tubes labeled standard and test respectively. The contents were mixed and read against reagent blank at 500 nm. The results were calculated thus:

$$\text{HDL-C-Cholesterol concentration (mmol/L)} = \frac{\text{OD of sample}}{\text{OD of standard}} \times 5.09 \text{ mmol/L}.$$

Determination of Serum Low Density Lipoprotein Cholesterol (LDL-C-C)

Serum LDL-C-C concentrations were derived from Friedewald equation (Friedewald, Levy & Fredrickson, 1972) (Akpan, 2019).

Quality Control (QC)

All analytical tests were done according to the standard operating procedure. It will involve the use of inter and intra-run of control sera along with the samples. Pre-analytical and post analytical precautions would be observed.

Validity of the Instrument

The questionnaire designed by the researcher was shown to the supervisor and face validity and reliability were determined before the questionnaires were administered.

Reliability of the Instrument

The questionnaire was subjected to scrutiny by the researcher‘s supervisor. Content validity was established based on assessment of each item of the questionnaire for clarity, consistency and relevance. The result of the pilot study revealed 75% consistency i.e. Cronbach alpha value of 0.75

Statistical Analysis

The data obtained was treated accordingly using Statistical Program for the Social Sciences (SPSS 17.0) for windows (SPSS Inc, Chicago, 1L). Serum antioxidant status, MDA and serum glucose levels of all the subjects were assessed, compared against age and BMI groups of the subjects using the two tail student’s t-test. ANOVA was then used to compare between different groups. Other analytes estimated along with the TAS were correlated to find relationship between them and their effect in controlling GDM. Correlation between TAS, SOD, Catalase, GPx and glucose as well as MDA was carried out using Pearson’s linear correlation analysis. A p-value of equal to or less than 0.05 ($p \leq 0.05$) was considered significant.

3. Results

Distribution of The Study Population

One hundred and sixty women aged 18-40 years consented and were recruited for the study. They were made up of 60 (37.50%) GDM subjects with mean age 30.93 ± 0.55 years, 50 non GDM pregnant women (31.25%) with mean age 27.68±0.50 years and 50 non GDM non-pregnant (31.25%) with mean age 29.24 ± 0.42 years as shown in Table 1.

Table 1: Distribution of the study population

Subjects	N	Percentage (%)	Mean Age(years)
GDM	60	37.50	30.93 ±0.55
$Control_1$	50	31.25	27.68 ±0.50
$Control_2$	50	31.25	29.24 ±0.42

N=Number of patients, GDM = Gestational Diabetes Mellitus,
$Control_1$= non GDM pregnant women and $Control_2$ = non diabetic non pregnant.

Estimated Gestational Age and Body Mass Index of GDM Patients and Controls Subjects

Table 2, revealed Estimated Gestational Age of their pregnancies as well as their Body mass index. A greater number of the women (45) were in their 28^{th} week with 23 women having GDM and 23 non-GDM pregnant controls. However, 38 were in their 24^{th} week having, 19 subjects each for both GDM and Control subjects. Ten women (7 GDM and 3 Controls) were found to be in their 25^{th} week of pregnancy while, only 8(5 GDM /3 Controls) and 9 (6 GDM and 3 Controls) subjects were in their 26^{th} and 27^{th} week respectively.

The subjects with BMI $\geq$ 30 kg/m^2 were 44 in number, 18 had GDM while 9 and14 were non GDM pregnant and non-diabetic non-pregnant subjects respectively. Also, 54 women (27 GDM, 19 $Control_1$ and 9 $Control_2$) were within the range of 25.0-29.9 kg/m^2. However, the normal BMI group had a total of 62 subjects consisting of, 10 GDM, 22 $Control_1$ and $Control_2$, had BMI between 18.5-24.9 kg/m^2.

Table 2: Estimated Gestational Age and Body Mass Index of GDM patients and controls Subjects

Groups	GDM (n = 60)	C_1 (n = 50)	C_2 (n = 50)	Total (n =160)
EGA (weeks)				
24	19	19	-	38
25	7	3	-	10
26	5	3	-	8
27	6	3	-	9
28	23	22	-	45
BMI (kg/m^2)				
18.5-24.9	10	22	27	62
25.0-29.9	27	19	9	54
$\geq$ 30	18	9	14	44

n=Number of patients, EGA = Estimated Gravitational Age, BMI = Body Mass Index,
GDM = gestational diabetes mellitus, $Control_1$= non GDM pregnant women and
$Control_2$= non diabetic non pregnant.

Lipid Profile (Mean±Sem) of GDM Patients and Controls Loaded With OGCT And OGTT Samples

For OGCT, the TC, HDL-C, and LDL-C levels in GDM was significantly increased ($p \leq 0.05$) when compared to C_1 and C_2 except for TG level in which there was insignificant increase in C_2 compared to G and C_1. Intergroup ($GvsC_1$, $GvsC_2$ and C_1vsC_2) comparison shows that, significant differences were observed in groups $GvsC_2$ and C_1vsC_2 for LDL-C. However, TG was still similar in $GvsC_1$, $GvsC_2$ and C_1vsC_2 but the significant difference was seen in $GvsC_2$for TC: HDL-C ratio. Furthermore, for OGTT (FBG), the TG and LDL-C increased significantly ($p \leq 0.05$) in GDM than C_1 and C_2 while for TC, HDL-C the levels were found to be similar. Also, with an intergroup comparison for TG and LDL-C, differences were seen in $GvsC_1$, $GvsC_2$ and C_1vsC_2. For OGTT (2HBG) a significant increase in GDM patients for TG and LDL-C levels with the same trend seen for TC and HDL-C but in C_2. Intergroup differences occur in $GvsC_1$, $GvsC_2$ and C_1vsC_2 for TC, TG, LDL-C and HDL-C. The differences are also occurred for TC in $GvsC_1$ while TC and HDL-C in $GvsC_2$ all presented in table 3.

Table 3: Lipid Profile of GDM patients and controls loaded in OGCT and OGTT Samples

Parameter (mmol/L)	GDM (n=60)	C_1(n = 50)	C_2(n = 50)	F-value	p- value	GvC_1	GvC_2	C_1vC_2
RBG-TC	5.95 ± 0.14^a	5.52 ± 0.16^b	5.04 ± 0.14^c	10.427	0.000	0.033	0.008	0.022
TG	2.09 ± 0.06^a	2.23 ± 0.06^a	3.90 ± 1.52^a	1.450	0.238	0.905	0.119	0.168
HDL-C	$1.71 \pm 0.03a$	1.64 ± 0.03^a	1.53 ± 0.03^b	10.973	0.000	0.078	0.000	0.006
LDL-C	2.66 ± 0.04^b	2.64 ± 0.05^a	2.06 ± 0.05^b	57.796	0.000	0.864	0.000	0.000
FBG-TC	4.55 ± 0.11^b	4.86 ± 0.20^a	4.88 ± 0.13^a	1.583	0.209	0.142	0.122	0.983
TG	2.36 ± 0.03^a	1.99 ± 0.05^b	0.93 ± 0.04^c	338.099	0.000	0.000	0.000	0.000
HDL-C	1.45 ± 0.03^a	1.55 ± 0.05^b	$1.49 \pm 0.03^{a,b}$	2.116	0.124	0.041	0.338	0.295
LDL-C	2.52 ± 0.03^a	2.47 ± 0.06^a	1.91 ± 0.03^b	56.597	0.000	0.490	0.000	0.000
2HBG-TC	4.47 ± 0.13^a	4.89 ± 0.16^b	5.06 ± 0.11^b	5.205	0.006	0.028	0.002	0.397
TG	2.23 ± 0.03^a	2.06 ± 0.05^b	0.89 ± 0.05^c	259.194	0.000	0.006	0.000	0.000
HDL-C	1.41 ± 0.03^a	1.50 ± 0.03^b	1.53 ± 0.02^b	5.305	0.006	0.025	0.002	0.414
LDL-C	2.41 ± 0.03^a	2.40 ± 0.04^a	1.93 ± 0.03^b	64.642	0.000	0.957	0.000	0.000

n=Number of patients, GDM = gestational diabetes mellitus, $Control_1$= non GDM pregnant women, $Control_2$ = non diabetic non pregnant, BG= Random Blood Glucose, FBG= Fasting Blood Glucose, 2HBG= Two Hours Blood Glucose, SEM=standard error of mean and significantly different=($p \leq 0.05$).

Lipid Profile (Mean±Sem) According to Age Groups of GDM Patients for OGCT And OGTT Samples

The mean values of lipid profile in GDM patients reveal that all the analytes in both the OGCT and OGTT subjects are similar ($P \geq 0.05$). However, LDL-C were found to be significantly higher ($p \leq 0.05$) in patients within the age group of 26 – 35 years compared to ≤ 25 & ≥ 35 years in OGTT (2HBG) type as shown in table 4.

Table 4: Lipid Profile according to age groups of GDM patients for OGCT and OGTT samples

Parameters (mmol/L)	≤ 25 (n = 17)	26-35 (n = 28)	≥ 35 (n = 15)	F-value	p- value	≤ 25 vs 26-35	≤ 25 vs ≥ 35	26-35 vs ≥ 35
RBG-TC	5.86±0.24	5.89±0.22	6.16±0.26	0.372	0.691	1.000	1.000	1.000
TG	2.10±0.12	2.07±0.67	2.13±0.13	0.087	0.916	1.000	1.000	1.000
HDL-C	1.69±0.05	1.70±0.04	1.75±0.05	0.404	0.670	1.000	1.000	1.000
LDL-C	2.64±0.07	2.64±0.06	2.70±0.07	0.223	0.801	1.000	1.000	1.000
FBG-TC	4.74±0.24	4.54±0.15	4.36±0.25	0.724	0.489	1.000	1.708	1.000
TG	2.29±0.06	2.41±0.05	2.36±0.05	1.306	0.279	0.388	1.000	1.000
HDL-C	1.50±0.07	1.43±0.03	1.41±0.05	0.955	0.391	0.693	0.688	1.000
LDL-C	2.57±0.07	2.52±0.04	2.45±0.11	0.735	0.484	1.000	0.701	1.000
2HBG-TC	4.14±013	4.75±0.24	4.33±0.19	0.229	0.117	0.146	1.000	0.575
TG	2.21±0.06	2.24±0.04	2.23±0.07	0.070	0.933	0.575	1.000	1.000
HDL-C	1.35±0.03	1.47±0.05	1.38±0.04	2.294	0.110	0.152	1.000	0.461
LDL-C	2.35±0.03	2.47±0.05	2.35±0.02	3.300	0.044	0.114	1.000	0.116

n=Number of patients, RBG= Random Blood Glucose, FBG= Fasting Blood Glucose, 2HBG= Two Hours Blood Glucose, SEM=standard error of mean, significantly different= ($p \leq 0.05$).

Lipid Profile (Mean±Sem) According to BMI Distribution Of GDM Patients For OGCT And OGTT Samples

The mean values of all the lipid profile in GDM patients for OGCT & OGTT are all similar ($p \geq 0.05$) as seen in Table 5, except TG which was significantly higher ($p \leq 0.05$) in OGTT sample with BMI ≥ 30 kg/m^2 when compared with group 18.5 – 24.9 kg/m^2 & 25.0 – 29.9 kg/m^2. An intergroup comparison revealed insignificant difference ($p \geq 0.05$) in all the compared groups.

4. Discussion

This study was carried out to determine the age, BMI, and Lipid Profiles of Gestational Diabetes Mellitus subjects in Kaduna State. A total of 160 subjects consisting of 60 GDM, 50 non-GDM pregnant women and 50 apparently healthy non-pregnant women were used for the study. Standard procedures carried out using the 50 g OGCT and 75 g OGTT according to WHO, 1999 criteria [13].

Demographic characteristics (Age and BMI,) were studied in GDM, non-GDM pregnant ($Control_1$) and apparently healthy non-Diabetic non-pregnant ($Control_2$) subjects. Age, BMI and SBP were significantly higher in GDM subjects than C_1&C_2.

The Age of the subjects was significantly higher in GDM patients compared with $Control_1$ as well $Control_1$ vs $Control_1$. However, this is similar to the reported analysis of Age significantly higher in GDM subjects compared to non-Diabetic pregnant control subjects. These variations may be due to age range variance among the recruited subjects. Revealed age of GDM subjects was not significant ($p \geq 0.05$) when compared to non-diabetic pregnant control subjects and this may be due to anti-oxidant effect on vitamins which may likely be aginst calorie deposit. Furthermore, a study by Sun [14] revealed Age distributions were significantly ($p \leq 0.05$) in GDM when compared to the corresponding non-GDM pregnant Control groups, this could be due to acquired educational certification with age. Also, differences in demographic characteristics, genetic background, environmental factors and diagnostic criteria for diagnosis of GDM could account for the inconsistency among the studies.

Table 5: Lipid Profile according to BMI Distribution of GDM patients for OGCT AND OGTT samples

Parameter (mmol/L)	**Normal 18.5-24.9 (n=13)**	**Over weight 25.0 -29.9 (n=26)**	**Obese ≥30 (n=21)**	**F- value**	**p-value**	**18.5-24.9V 25.0 – 29.90**	**18.5-24.9V ≥ 30.0**	**25.0 – 29.90V ≥ 30.0**
RBG-TC	6.38±0.22	5.76±0.22	5.92±0.24	1.493	0.233	0.620	0.455	0.165
TG	2.18±0.09	2.08±0.09	2.06±0.10	0.341	0.712	0.102	0.121	0.020
HDL-C	1.79±0.04	1.67±0.04	1.71±0.05	1.416	0.251	0.120	0.088	0.032
LDL-C	2.79±0.07	2.60±0.06	2.64±0.06	1.914	0.157	0.190	0.148	0.043
FBG-TC	4.61±0.25	4.39±0.17	4.71±0.20	0.765	0.470	0.220	0.095	0.315
TG	2.37±0.09	2.34±0.05	2.38±0.04	0.155	0.856	0.022	0.008	0.038
HDL-C	1.49±0.08	1.40±0.03	1.48±0.04	1.139	0.327	0.086	0.007	0.078
LDL-C	2.60±0.08	2.45±0.03	2.55±0.08	1.525	0.226	0.157	0.058	0.099
2HBG-TC	4.09±0.39	4.47±0.14	4.71±0.22	1.531	0.225	0.380	0.619	0.0238
TG	2.32±0.06	2.25±0.05	2.15±0.05	0.253	0.114	0.070	0.175	0.104
HDL-C	1.34±0.08	1.41±0.03	1.46±0.04	1.423	0.249	0.078	0.120	0.043
LDL-C	2.39±0.07	2.41±0.03	2.41±0.04	0.050	0.952	0.020	0.013	0.008

n=Number of patients, GDM = gestational diabetes mellitus, RBG= Random Blood Glucose, FBG= Fasting Blood Glucose, 2HBG= Two Hours Blood Glucose, SEM=standard error of mean, values with different (a, b) superscripts are significantly different ($p \leq 0.05$) and 18.5-24.9 V 25.0 – 29.90, 18.5-24.9 V ≥ 30.0, 25.0 – 29.90V ≥ 30.0

The Body Mass Index (BMI) which is a known risk factor in GDM. The BMI in this study was found to be significantly higher in GDM patients compared to non-GDM pregnant subjects ($Control_1$) & non-diabetic non-pregnant subjects ($Control_2$). When an intergroup comparison was made, $Control_1$ was found to be similar ($p \leq 0.05$) to $Control_2$. A similar finding revealed BMI of pregnant women with GDM to be significantly higher ($p \leq 0.05$) than the non-diabetic pregnant Control subjects. Our finding also agreed with a report which revealed that, BMI was significantly increased in GDM patients compared to non-GDM pregnant Control subjects, this increased BMI in pregnant women could be due to changes in estrogens levels as well as fat deposition occurring during pregnancy. However, revealed BMI of GDM subjects to be statistically insignificant ($p \geq 0.05$) when compared to non-diabetic pregnant control subjects and this may be due to anti-oxidant effect on vitamins which may likely be against calorie deposit. Furthermore, increase BMI could be due to the excessive energy consumed, other socioeconomic factors as well as their educational background with the surrounding environmental factors such as extreme temperature/ radiation, stress, undernutrition, drug exposure.

Similarly, one of the vital signs taken during every antenatal care visit is blood pressure. The SBP and DBP revealed significant increase in the GDM compared to non-GDM pregnant subjects ($Control_1$) & non-diabetic non-pregnant subjects ($Control_2$). This finding is similar to the study carried out by [14] which also revealed that, blood pressure (SBP and DBP) was significantly increased in GDM than apparently healthy non-GDM pregnant controls. This may not be unconnected with weight gain and decreased vascular oncotic pressure due to relative edema in pregnancy. Furthermore, when an intergroups comparison were done, similar increase ($p \geq 0.05$) was observed in all the groups (GDM vs non-GDM pregnant $Control_1$, GDM vs non-diabetic non-pregnant $Control_2$ & non-GDM pregnant $Control_1$ vs non-diabetic non-pregnant $Control_2$). There have been reports on similar blood pressure between GDM patients and non-GDM pregnant control subjects. In Thailand, the Age and BMI of GDM patients were reported to be similar ($p \geq 0.05$) in both pregnant subjects that are with GDM (GDM patients) or without GDM as control subjects. Also, in another case-controlled study by Milajerdi using four hundred and sixty-two subjects and Guan who analyzed 461 GDM patients with 626 non-GDM pregnant control subjects, revealed that the Age and DBP of the GDM patients and controls were statistically not significant ($p \geq 0.05$) but SBP was found to be significantly higher in GDM than control subjects. The Age, SBP based our findings, also suggest a link with GDM disease and its progression.

It has been fully documented that in GDM pregnancies that, all well controlled maternal lipids are strong predictors for fetal growth. For OGCT (RBS) type, the TC, HDL, LDL levels in GDM were significantly increased when compared to non- GDM pregnant $Control_1$ and non-Diabetic non-pregnant $Control_2$. However, OGTT (FBG), the TG and LDL increased significantly in GDM than the Controls subjects while for OGTT (2HBG), a significant increase in GDM patients for TG and LDL-C levels with the same trend seen for TC and HDL but in C_2. These is in agreement with the findings of [12, 15, 16]. Increased lipids may become toxic, causing endoplasmic reticulum stress, mitochondrial dysfunction, and the generation of ROS leading to diabetic complications. More also, for OGCT in our study, the TC, HDL-C, LDL-C levels in GDM were significantly higher ($p \leq 0.05$) when compared to non-GDM pregnant $Control_1$ and non-pregnant non-diabetic $Control_2$ except for TG level in which there was an insignificant increase in non-pregnant non-diabetic $Control_2$ compared to GDM and C_1. Studies have shown significantly higher ($p \leq 0.05$) level of TG in GDM than healthy pregnant control subjects. Intergroup (GDM vs non-GDM pregnant $Control_1$, GDM vs non-pregnant non-diabetic $Control_2$ and non-GDM pregnant $Control_1$ vs non-diabetic non-pregnant $Control_2$) comparison showed significant differences were observed in GDM groups vs non-diabetic non-pregnant C_2 (this increase may be due to increase nutritional requirement in pregnancy) and non-GDM pregnant $Control_1$ vs $Control_2$ for TC, HDL-C and LDL-C which may as well be connected to increased nutritional requirement in pregnancy. However, TG was still similar in GDMvs $Control_1$, GDM vs non-diabetic non-pregnant $Control_2$ and non-GDM pregnant $Control_1$ vs non-diabetic non-pregnant $Control_2$.

Furthermore, for OGTT (FBG) type, the TG and LDL-C increased significantly ($p \leq 0.05$) in GDM than non-GDM pregnant $Control_1$ and non-diabetic non-pregnant $Control_2$ while for TC and HDL-C the levels were found to be similar. This is similar to the study by [17] that observed TG and LDL-C to be significantly elevated in pregnant and non-pregnant subjects. This present study is also in agreement with the report of [18], who observed similar levels of TC in GDM and non-GDM pregnant Controls. Also, when an intergroup comparison was carried out for the OGCT type, there was significant differences in the Total Cholesterol (TC) level in GDM vs non-GDM pregnant $Control_1$, GDM vs non-diabetic non-pregnant $Control_2$ and non-GDM pregnant $Control_1$ vs non-diabetic non-pregnant $Control_2$ groups. For OGTT type, a significant increase was revealed for TG and LDL-C levels in GDM patients compared to non-GDM pregnant $Control_1$ and non-diabetic non-pregnant $Control_2$. In addition, significantly lower levels of TC and HDL-C was in seen GDM patients compared to non-GDM pregnant $Control_1$ and non-diabetic non-pregnant $Control_2$. This is found to be consistent with the report of [19, 20] who

observed significantly higher ($p \leq 0.05$) levels of LDL-C in GDM women compared to their corresponding non-GDM pregnant control subjects. Furthermore, our findings for TC and HDL-C in GDM vs non-diabetic non-pregnant $Control_2$, are in line with other studies and there were heightened oxidative stress during pregnancy which have been revealed to be propagated by the abnormalities of LDL-C and HDL-C functions. Other studies by showed that, LDL-C and TG where significantly raised in GDM patients than control subjects. This increase may be due to increase in steroid production during pregnancy as well as increase progesterone concentration. While HDL-C was lower in GDM patients than controls, the difference in TC was reported to be similar [21]. Furthermore, also, recorded lower levels of HDL-C- Cholesterol in GDM patients than Control subjects this may be due to some presence of hepatic lipase inhibitors. Intergroup (GDM vs non-GDM pregnant $Control_1$, GDM vs non-diabetic non-pregnant $Control_2$ and non-GDM pregnant $Control_1$ vs non-diabetic non-pregnant $Control_2$)

Generally, our findings just like the work of [21] who recorded TG and LDL-C to be significantly different in GDM subjects when compared to controls but TC and HDL levels were insignificant between GDM and Controls. Furthermore, [15] reveals that the levels of lipids, including TC, TG and LDL-C, increased gradually during pregnancy. These increases are consistent with the above stated studies and are physiological requirement for maintaining stable fuel supplementation to the growing fetus. For determination of Lipid Profile using OGTT sample, this may not be replaced completely but OGCT can also be helpful in GDM analysis because Lipid Profile levels change remarkably during gestation and abnormal levels of TG especially are usually associated with pregnancy complications like GDM [22]. Our result is in agreement with the findings among other studies and revealed no increase in TC but significant increase TG in GDM patients when compared to Controls subjects these parameters were significant ($p \geq 0.05$). However, it has been reported that there was significant increase in TG level of GDM subjects than non-control subjects.

Other studies revealed, TG levels were significantly elevated in women with GDM compared with those without GDM. Also, HDL-C levels were significantly lower in women with GDM compared with those without GDM and no differences in aggregate Total cholesterol or LDL-C levels between women with GDM and those without. The findings, observed TC, TG, HDL-C and LDL-C to be significantly increased ($p \leq 0.05$) in GDM patients than control subjects. This could be due to the fact that a larger sample size was used for the research work. This rise in lipids becomes toxic and contributes to endoplasmic reticulum stress, mitochondrial dysfunction, and the generation of reactive oxygen species, which together trigger inflammation as well as insulin resistance. In a study by Arribas, that was carried out in two groups of 36 women each, healthy pregnant and Diabetic group revealed, similarity ($p \geq 0.05$) in all the lipid parameters of both GDM and pregnant controls.

The levels of TC, TG, HDL-C and LDL-C in all the age groups ($\leq$25vs26-35, $\leq$25vs$\geq$35 & 25-35vs$\geq$35) when compared, was found to be non-significant for both OGCT and each OGTT type. However, for the OGTT (2HBG) type, LDL-C in age $\leq$25 was significantly increased compared to 26-35 & $\geq$35 age groups. This is similar to the findings of Cibickova, Langova, Schovanek, Macakova, Krystynik & Karasek, [21] who revealed also, higher level of LDL-C in GDM subjects. Even though, TG for OGCT type, was seen to be statistically lower in $\leq$25 years group when compared to other groups. This could probably due the risk of developing Diabetes and its complications. It can be observed that on LDL-C, a significant change was observed within the groups but not across the group of $<$ 25 vs 25-30, this can also be observed on TG, this may be due to the fact that both group observed a significant changes thereby making it non-significant when compared with each other.

The Lipid profile of all the BMI groups (18.5-24.9 vs 25.0 – 29.90, 18.5-24.9 vs$\geq$30.0, 25.0 – 29.90 vs$\geq$30.0) when compared, was found to be similar ($p \geq 0.05$) for both OGCT and OGTT type. However, in a study by [23] it was discovered that, while other lipid profile components versus weight remain similar ($p \geq 0.05$) in GDM subjects, significant increase in TG was positively correlated with weight ($r=0.089$, $p=0.005$) which is a component of BMI. This TG elevation thereby increases the risk of women having GDM. Also, in some studies, it has been revealed that, increase Triglycerides levels in GDM patients is linked to BMI levels too [24] who discovered lipid profile components versus weight remain Statistically insignificant in GDM subjects and weight is a determinant of BMI. Therefore, TG levels could be used as follow-up parameters during pregnancy, while other lipids may be meaningful only in early or late pregnancy period. Similarly, lifestyle of the study area could be a contributing factor to the result obtained in this study.

5. Conclusion

The findings of this study highlight the clinical and public health significance of gestational diabetes mellitus (GDM) in Kaduna State, Nigeria, particularly in relation to age, body mass index and cardiovascular risk profiles. Women with GDM demonstrated significantly higher age, body mass index (BMI) compared with non-GDM pregnant and non-pregnant controls, indicating a strong association between GDM and adverse cardiometabolic status.

Lipid profile alterations were also observed, with significant increases in total cholesterol, triglycerides, and low-density lipoprotein cholesterol in GDM subjects depending on OGCT and OGTT classification, while high-density lipoprotein cholesterol showed variable changes. However, most lipid parameters were not significantly influenced by age or BMI stratification, except for isolated variations such as elevated LDL-C in the 26–35-year age group and higher glucose levels in overweight participants.

Correlation analyses further demonstrated significant associations between age, BMI, as well as selective relationships between BMI and lipid fractions, adiponectin, and other metabolic indices, underscoring the interconnected nature of metabolic dysfunction in GDM.

Overall, this study suggests that GDM in Kaduna State is characterized by a complex interplay of dyslipidemia reinforcing the need for early screening, lifestyle intervention, and improved metabolic monitoring to reduce maternal and fetal complications.

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