lipid profile parameters was observed. FBS levels were positively linked with TG, TC, and LDL-c but negatively associated with HDL-c.

**Conclusion:** A correlation between FBG and serum lipid profile parameters has been observed. FBS levels were positively linked with TG and TC, but negatively associated with HDL-C, the good cholesterol. It can be suggested that failure to control type 2 diabetes leads in dyslipidaemia. Thus, effective control of hyperglycaemia in type 2 diabetics and periodic check and treatment of dyslipidaemia can prevent associated adverse health outcomes.

## INTRODUCTION

Type 2 diabetes (T2DM) is a severe global health issue that has reached alarming levels. Recently, more than half a billion (536.6million people) have been reported to have DM worldwide and about 14.2 million of them lived in Africa (IDF, 2021).[1] According to WHO (2015), Nigeria has the greatest number of people living with diabetes in Africa, with more than 1.5 million cases.[2] The occurrence of T2DM may be due to abnormalities in carbohydrate, protein and lipid metabolism mainly due to insulin resistance (in overweight and obese individuals) or insulin deficiency.[3] T2DM is often characterized by defects in lipids metabolism and dyslipidemia that trigger cardiovascular disease (CVD), coronary artery disease (CAD) and macrovascular-related complications.[4] Alteration of lipoproteins and lipid profile parameters in T2DM contributes to oxidative stress and formation of free radicals which damage the endothelial tissue and accelerate the progression of atherosclerosis in blood vessels and the subsequent CVD. It is estimated that, over 30 percent of patients admitted for CVD had T2DM in sub-Saharan Africa.[3, 4] The important risk factor, in the development of CVD in type 2 diabetics, is abnormalities of the lipid profile parameters and comorbidity with high blood pressure in old age.[5] The risk increases with increased blood levels of TC, TG, LDL-C, and decreased HDL-C.[6] Diabetes-related CVD are decreased by the normal lipid profiles especially HDL-c that circulate in the blood because of its role of removing bad cholesterol (LDL-C) from the blood. The decreased uptake of free fatty acids that circulate in the blood may be increased by TG hydrolysis owing to the lipase action. This may increase free fatty acids that play in favor of insulin resistance and atherogenic dyslipidemia.[7,8] Elevated remnant lipoprotein is correlated with high TG because it is composed of high cholesterol and TG.[9, 10] Extensive research on atherogenic dyslipidemia and the risk of CVD in type 2 diabetics has been carried out in developed countries. However, in Nigeria, few studies have been carried out to understand the pattern of dyslipidemia and its link with hyperglycemia in type 2 diabetics.[9,10] The aim of the present study was to determine the influence of hyperglycemia on the lipid profile parameters in type 2 diabetes. Thus, the regulation of dyslipidemia to reduce CVD-related mortality in diabetes could be further elucidated.

## MATERIALS AND METHODS

### Site of Study

The study participants were recruited from males and females with type 2 diabetes attending diabetic clinics at Specialist

Hospital Sokoto, Women and Children Hospital Sokoto, Maryam Abacha Hospital Sokoto, Sir Yahayya Memorial Hospital Birnin Kebbi and Kebbi Medical Centre. Apparently healthy individuals used as controls (age-, sex, ethnic- matched) were recruited within the metropolis.

**Study Population**

Nigeria as a multinational state is inhabited by more than 250 ethnic groups speaking over 500 distinct languages, all identifying with a wide variety of cultures. The study was conducted among the three major ethnic groups: the Hausa/Fulani, Yoruba and Igbo situated in the north, west, and east respectively, together comprising over 70% of the total population.

**Study Design**

The total of 300 participants recruited comprised of 100 participants each for Hausa, Igbo and Yoruba ethnic groups, aged 18-54 years. The total of 174 were Males and 126 were Females. The participants were divided into six groups (3 groups were type 2 diabetics and 3 groups were healthy control). Of the 174 male participants Hausa/Fulani has 56 males, Igbo has 64 males and Yoruba has 54 males. The remaining 44 participants, 36 participants and 46 participants were female for Hausa/Fulani, Igbo and Yoruba respectively. Ethnic backgrounds were identified through clinical records, informed consent form and questionnaire administered.

**Ethical consideration and clearance**

Ethical approvals were duly sought obtained from Kebbi State Ministry of Health and Sokoto State Ministry of Health with reference numbers: MOH/KSREC/VOL,1/56 and SMH/1580/V.IV respectively.

**Informed consent**

Informed consent for inclusion into the study was duly obtained from each participant using standard protocol prior to recruitment.

**Instrument for data collection**

Standard self-administered questionnaire was prepared and administered to all the study participants to obtain their socio-demographic characteristics including, gender, age, tribe, occupation, life style, family history of DM, history of DM, etc,.

**Sample Size Determination**

Calculation of sample size of the study, using Cochran formula (1977).[11]

$n=Z^2P(1-P)/d^2$

Where, n= desired sample size

P= Prevalence rate of diabetes in Nigeria = 7.0% = 0.07 (Michael *et al.*, 2024).[12]

Z= 95% confidence interval=1.96

d= degree of accuracy= 0.05

Therefore,

n=118, approximately 150

**Inclusion Criteria**

Newly diagnosed type 2 diabetics (with first diagnosis< 2 years), of both sexes (aged between 18 to 54 years old) with apparently non-existing complication and without any disease condition were included for this study. Age-, sex-, tribe- and BMI-matched healthy individuals who have given their informed consent served as controls.

**Exclusion Criteria**

Patients with other disease conditions such as hypertension, HIV/AIDS, tuberculosis, thyroid disorder and other apparently diabetes-related complications were excluded from the study. Pregnant women were excluded. Other disease conditions such as hypertensive retinopathy, hypertensive nephropathy, and alcoholic neuropathy were also excluded from the study.

**Sample Collection and Analytical Techniques**

Blood pressure was measured using the Belsk digital blood pressure monitor (Northfield, IL 60093 USA). The body height and weight were also measured using appropriate instrument (Mechanical Brecknell HS-200M scale, UK). A systolic blood pressure of ≥ 140 mmHg and diastolic blood pressure of ≥ 90 mmHg were considered hypertensive.[13] Body mass index (BMI) was calculated using the equation: Weight in (Kg)/Height ($M^2$). Values of 20-25, <30 but > 25, >30 and <20 were considered Normal, overweight, obese and underweight respectively. Blood glucose was tested in capillary blood samples by glucose oxidase and peroxidase methods using Accu-Check Aviva (68305 Mannheim, Germany).[14] Four milliliters of venous blood were collected in a red top tube with a clot activator collected from each participant by venipuncture using a 21G needle. It was then subjected to centrifugation at 3000 rpm for five min and the obtained serum was used to test TG, TC, and HDL.[15] Humastar 80 Auto Analyzer (Human, Wiesbaden. Germany) was utilized to measure the concentration of TC, TG, and HDL in the sera. The Friedewald formula (modified) was used to determine LDL-C (mg/dl) = (Non-HDL-C × 90%) − (TG × 10%). The concentration of TC, TG, and HDL was measured at 546 nm.[16] The normal reference values were defined as: < 200 mg/dl for TC, < 150 mg/dl for TG, < 100 mg/dl for LDL-C. For, HDL-C, values considered were 41- 60 mg/dl for males, and 51-60 mg/dl for females.15 All reagents, controls, and calibrators were stored in a fridge at 2-8 °C.

**Statistical Analysis**

All statistical analyses were performed with IBM SPSS V1 (IBM corporation, Armonk, New York, US) and Microsoft 2010 based Excel (Microsoft, US). All data were presented as mean and standard deviation. One-way analysis of variance (ANOVA) followed by Bonferroni multiple comparison test to evaluate the mean difference of the data between the groups (control and type 2 diabetics). The correlations were investigated using Pearson's coefficient of correlation (r) between study variables. Analysis was done at the 95% confidence level and the statistical significance was considered when p-value <0.05.

## RESULTS

Table 1 shows the Socio-demographic and clinical characteristics of the study participants. The total of 300 participants recruited for this study comprised of 174 (58%) Males and 126 (42%) Females. Of the 174 male participants, Hausa/Fulani has 56 (56%), Igbo has 64 (64%) and Yoruba has 54(54%); while females were 44(44%), 36(36%) and 46(46%) for Hausa/Fulani, Igbo and Yoruba respectively. Table 1 further presents the body mass index (BMI) of the study participants. The mean average BMI of the diabetics was higher compared to control subjects (F=2.741; p <0.05). No statistically significant difference observed in mean systolic and diastolic blood pressure for diabetics of the ethnic groups compared to their corresponding control (F=0.127; p>0.05).

Table 2 shows the mean comparisons of FBG (mg/dl) across the groups. The mean values for FBG and lipid profile parameters of the study ethnic groups were nearly the same. The level in the diabetics group was significantly higher than those in the control group (*p*<0.0001). The mean comparisons of TC, TG and LDL-c across the groups were significantly higher in diabetics compared to their corresponding control groups (*p*<0.0001). However, the mean comparisons of HDL-c (mg/dl) across the groups were lower in diabetics of different ethnic groups compared to their corresponding control groups (F=6.015; p<0.05).

The FBS was associated with increased TC, TG and LDL-c (r = 0.483269, 0.623952 and 0.390193 respectively). Similarly, BMI was found to be associated with increased TC, TG and LDL-c (r=0.432284, 0.569792 and 0.339701 respectively) p=0.000001) Table 3. However, both FBS and BMI were associated with decrease in serum HDL-c (r= -0.278993 and -0.247900 respectively) p=0.000001(Table 3).

**Table 1: Socio-demographic and clinical characteristics of the study participants**

| **Groups** | | **Mean age (Years)** | **Gender Male N (%)/Female N (%)** | **BMI ($Kg/m^2$)** | **SBP (mmHg)** | **DPB (mm/Hg)** | **Hypertensive: no N(%)/yes N(%)** |
|---|---|---|---|---|---|---|---|
| All Participants (n=300) | T2DM (n=150) | 55.04±6.64*† | 87(58)/63(42)[&] | 30.71±5.15*† | 128.95±9.47 | 80.80±5.01 | 150(100)/0(0) |
| | Control(n=150) | 45.97±7.58 | 87(58)/63(42) | 24.15±4.18 | 114.06±7.08 | 79.24±4.57 | 150(100)/0(0) |
| Hausa/Fulani (n=100) | T2DM(n=50) | 55.57±7.39*† | 28(56)/22(44) | 27.59±4,90 | 123.74±9.30 | 78.17±5.00 | 50(100)/0(0) |
| | Control(n=50) | 42.27±8.11 | 28(56)/22(44) | 23.21±4.62 | 115.18±7.78 | 69.78±3.80 | 50(100)/0(0) |
| Igbo (n=100) | T2DM(n=50) | 55.04±6.34 | 32(64)/18(36) | 32.6±4.60*† | 129.37±8.36 | 72.64±4.19 | 50(100)/0(0) |
| | Control(n=50) | 46.98±7.48 | 32(64)/18(36) | 25.6±3.71 | 116.71±6.41 | 74.48±4.89 | 50(100)/0(0) |
| Yoruba (n=100) | T2DM(n=50) | 54.51±6.18 | 27(54)/23(46) | 29.81±5.50 | 131.74±8.42 | 78.57±4.60 | 50(100)/0(0) |
| | Control(n=50) | 48.65±7.14 | 27(54)/23(46) | 23.6±2.72 | 110.30±5.94 | 71.48±4.18 | 50(100)/0(0) |
| | F value | 6.015 | 3.474 | 2.751 | 0.122 | 0.127 | 4.384 |
| | P value | <0.05 | >0.05 | <0.05 | >0,05 | >0.05 | >0.05 |

Values are mean ± Standard Deviation of the mean of age, SBP, DBP and BMI ,n= number of participants, T2DM= type 2 diabetics, %= percentage, $Kg/m^2$, kilogram per meter square; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP. Blood pressure, n, sample size; %, percentage,;†= comparison between patients and control, &= comparison within the group, and *=p≤0.05 was considered significant in the statistical analysis t-test.

**Table 2: FBG and lipid profiles of the study population**

| Groups | | FBG (mg/dL) | TC (mg/dL) | TG (mg/dL) | HDL-c (mg/dL) | LDL-c (mg/dL) |
|---|---|---|---|---|---|---|
| All Participants (n=300) | T2DM(n=150) | 122.20±17.60**† | 207.99±89.82**† | 183.16±60.76**† | 37.26±12.90 | 101.27±52.19*† |
| | Control(n=150) | 93.26±8.68 | 161.68±27.64 | 137.14±24.18 | 46.64±11.15*† | 90.16±28.14 |
| Hausa/Fulani (n=100) | T2DM(n=50) | 127.70±23.48**† | 203.74±89.49**† | 181.21±59.85**† | 34.92±13.72 | 100.05±51.46*† |
| | Control(n=50) | 92.80±8.95 | 159.71±27.70 | 132.91±18.89 | 46.61±11.23*† | 93.51±30.30 |
| Igbo (n=100) | T2DM(n=50) | 118.12±10.62**† | 216.51±90.35**† | 189.41±63.40**† | 39.72±12.37 | 104.82±53.98*† |
| | Control(n=50) | 91.68±8.94 | 162.60±27.15 | 139.38±26.00 | 46.55±11.19*† | 89.10±26.95 |
| Yoruba (n=100) | T2DM(n=50) | 120.80±14.98**† | 203.71±90.29**† | 178.87±0.67**† | 37.15±12.68 | 98.94±51.67*† |
| | Control(n=50) | 95.31±7.82 | 162.74±28.35 | 139.14±26.66 | 46.75±11.18*† | 87.88±27.13 |
| | F- value | 17.384 | 17.474 | 14.083 | 6.015 | 9.815 |
| | P-value | <0.01 | <0.01 | <0.01 | <0.05 | <0.05 |

Values are mean ± Standard Deviation of the mean of FBS, LP, lipid profile n, number of participants; %, percentage,; †= comparison between patients and control, *=p≤0.05 and **=p≤0.01 were considered significant in the statistical analysis t-test.

**Table 3: Correlation between FBG and BMI with serum lipid profiles of the study population**

| Parameters | | TC (mg/dL) | TG (mg/dL) | HDL-c (mg/dL) | LDL-c (mg/dL) |
|---|---|---|---|---|---|
| FBG (mmol/L) | r value | 0.483269** | 0.623952** | -0.278993** | 0.390193** |
| | P value | 0.000001 | 0.000001 | 0.000001 | 0.000001 |
| Body Mass Index (kg/m$^2$) | r value | 0.432284** | 0.569792** | -0.247900** | 0.339701** |
| | P value | 0.000001 | 0.000001 | 0.000001 | 0.000001 |

**** Correlation is significant at the < 0.01 level (2-tailed).**

## DISCUSSION

The current study involved three hundred (300) participants, hundred (100) from each of the three ethnic groups (Hausa/Fulani, Igbo and Yoruba). Each group has fifty (50) diabetics and fifty (50) control subjects, non-hypertensive with significant increased BMI in diabetic groups compared to their corresponding control groups ($p<0.05$). The mean values for FBG and lipid profile parameters of the study ethnic groups were nearly the same. The current study determined the mean FBG concentration in the diabetics of the study groups to be significantly higher compared to their corresponding control groups ($p< 0.01$). Significantly increased serum levels of TC and TG were observed for diabetic groups compared to their corresponding control groups (both $p<0.01$). Similarly, increased serum level of LDL-c and decreased serum level of HDL-c among diabetics of the study groups were observed compared to their corresponding control groups (both $p<0.05$). The glucose levels in this study groups are consistent with the glycaemic condition of type 2 diabetes as defined by ADA, (2022).[17] In type 2 diabetes, hyperglycaemia is the result, initially, of the inability of the body cells to fully respond to insulin (insulin resistance). With the onset of insulin resistance, the hormone is less effective which, in due course, prompts an increase in insulin production. Over time, inadequate production of insulin can develop as a result of failure of the pancreatic beta cells to keep up with demand leading to overt hyperglycaemia.[1] Hyperglycaemia may have many effects on the vascular endothelium which leads to the development of dyslipidemia.[18]

In the current study, a significant positive correlation between FBG and TC, TG and LDL-c (r=0.483269, 0.623952 and 0.390193 respectively) and a significant negative correlation (r= -0.278993) between FBG and HDL-c were observed. The hardening of the blood artery wall may be caused by the LDL-c because it accumulates LDL in the artery wall which narrows the blood vessel. This abnormality of LDL which is known as bad cholesterol may result from the impairment of blood glucose.[18] These findings agree with those reported in several other studies providing further evidence that in poor glycemic control, the likelihood of a high concentration of LDL-C would be increased.[19] Therefore, blood glucose could be utilized to predict the level of LDL-C in diabetic patients because blood glucose can cause the defect of LDL which leads to the formation of plaque inside the artery.

A significant positive correlation was observed between the BMI and TC, TG, and LDL-c, but negative association with HDL-c. The same observation was reported by another study.[10] The association between BMI and lipid profile parameters may therefore be due to lifestyle changes. Several studies reported that increased in BMI accounted for lipid abnormality.[10] Similarly, this study showed a significant link between BMI and lipid profile status. It is evident that dyslipidemia is among the consequences of high BMI.[20] The levels of serum HDL-C were significantly lower in diabetics compared to controls. Contrasting results were reported in other studies.[21, 22] However, similar to this study, the levels of serum HDL-C were significantly lower in patients than in control.[22]

Diabetics may possess various lipid abnormalities predisposing to the threat of cardiovascular ailments. This condition arises owing to especially glucose catabolism, resulting in hyperglycemia and dyslipidemia.[23] Other studies have reported a non-significant weak positive correlation between FBG and TC in type 2 diabetics.[24, 25] Ultimately, the results imply that uncontrolled diabetes leads to defects in cholesterol metabolism. Thus, periodic check and treatment of dyslipidemia can prevent associated adverse health outcomes. Triglycerides transport adipose fat and blood glucose to and from the liver where they are produced.[26] Hypertriglyceridemia contributes to the plaque formation inside of the walls of blood vessels leading to their narrowing and preventing normal blood flow with increased risk of cardiovascular complications.[27] Several other studies reported a moderate positive and significant correlation between triglyceride and blood glucose.[28, 29] Therefore, the concentrations of blood glucose may be utilized in prognosticating the serum TGs and cardiovascular risk due to alteration of lipoprotein caused by hyperglycemia. Hardening of the blood artery wall may be caused by the LDL-c which accumulates LDL in the artery wall that narrows the blood vessel. The abnormality of LDL, a bad cholesterol, may results from the impairment of blood glucose.[28] Several other studies provided further evidence that in poor glycemic control, the LDL-c increases.[9, 10, 30] Therefore, blood glucose could be utilized to predict the level of LDL-c in diabetics because blood glucose may lead to defect in LDL which results in the formation of plaque inside the artery.

## CONCLUSION

A correlation between FBG and serum lipid profile parameters has been observed. FBS levels were positively linked with TG and TC, but negatively associated with HDL-C, the good cholesterol. It can be suggested that failure to control type 2 diabetes leads in dyslipidemia. Thus, effective control of hyperglycemia in type 2 diabetics and periodic check and treatment of dyslipidemia can prevent associated adverse health outcomes.

**Limitation the study**

Standard biochemistry procedures were employed in this study to quantify TC, HDL-c, TG, LDL-c, and FBG. The present results, therefore, are correct and valid. Study patients in the present research were from the three major ethnic groups in Nigeria. The present results therefore may serve as Nigerian estimates even though, there are more 250 ethnic groups in Nigeria.

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