Original Article

Pattern of Dyslipidemias, Associated Atherogenic Risk and Insulin Sensitivity in Type 2 Diabetes Mellitus

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ABSTRACT

Dyslipidemia is a major phenotype associated with diabetes mellitus, and this is essential in the development of cardiovascular risk. This study aimed to establish the most common dyslipidemia pattern among type 2 DM, atherogenicity, and associated insulin sensitivity. A cross-sectional study, involving 141 type 2 diabetes patients and 140 healthy individuals as controls, was adopted. A multistage sampling technique was employed, and a semi-structured interviewer-administered questionnaire was used to collect socio-demographic and clinical data. The combined dyslipidemia pattern had the highest percentage among subjects, with a value of 52(45.6%) observed. It was followed by mixed dyslipidemias with a value of 50(43.9%). A higher percentage of increased risk of CVD was found among the subjects for CRI-I (74; 52%), CRI-II (73; 51.8%), and AC (75; 53.2%) compared with control CRI-I (15; 10.7%), CRI-II (7; 5.0%) and AC (15; 10.7%), p-values <0.001. A greater percentage of subjects demonstrated insulin resistance, as indicated by HOMA-IR (79, 56.0%) and QUICKI (64, 45.4%), compared to the controls with p <0.001. HbA1C correlated with HOMA-IR (r=0.235, p=0.005) and QUICKI (r=-0.196, p=0.020). Mixed dyslipidemias (elevated total cholesterol, elevated LDL-C and low HDL-C) as the highest form of dyslipidemia pattern was observed. CRI-I, CRI-II and AC were better predictors of CVD than an ordinary lipid profile. HOMA-IR and QUICKI were better tools for assessment of insulin sensitivity/resistance than plasma glucose and insulin. HbA1c can therefore be adopted as a screening tool for insulin resistance in a large population, not as a diagnostic tool alone.

Keywords: Atherogenic indices, Dyslipidemias, HbA1C, HOMA-IR, Insulin resistance, QUICKI

INTRODUCTION

Diabetes mellitus (DM) is the most common metabolic disorder and a leading cause of death globally. About 400 million people live with diabetes worldwide, and its prevalence is on the increase in developing countries. DM is a progressive, chronic disease caused by a relative or definite insulin deficiency or insulin resistance, leading to hyperglycemia, characterized by metabolic disorders of lipids, carbohydrates, and proteins.[1,2,3] DM is a complex disease with many sub-phenotypes associated with the syndrome. Dyslipidemia is the associated phenotype with diabetes mellitus that is crucial in the development of cardiovascular (CV) risk.[4,5,6] Reports from several studies have shown that diabetic patients have a 2 to 4 times higher risk of developing cardiovascular diseases.[2]

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Cholesterol in Adults (Adult Treatment Panel III, or ATP III) has made diabetes mellitus a coronary heart disease equivalent, elevating it to the highest risk category.[7] The decreased ability of insulin to act effectively on target tissues leads to metabolic abnormalities that cause an increased risk of cardiovascular disease CVD and DM. The features of insulin resistance include central obesity, hypertriglyceridemia.[2,3]

Dyslipidemia may result from single or multiple genetic mutations that result in either overproduction or defective clearance of triglycerides and low-density lipoprotein cholesterol (LDL-C) or underproduction or excessive clearance of HDL-C.[2,3,8] Abnormalities of the lipid profile are one of the constellations of clinical presentations that accompany type 2 diabetes (T2D) in a disorder termed "metabolic syndrome". Insulin resistance is the key concept behind metabolic syndrome.[1,9]

Lipid abnormalities are the commonest cause of diabetic atherosclerosis. The pathophysiology is complex, with dysfunction of the fibrinolytic system, pro-oxidative state, hyperglycaemia, and possibly hyperinsulinaemia also explaining part of the increased susceptibility of people with diabetes to atherosclerotic complications.[2] Dyslipidemia confers an excess atherosclerotic cardiovascular risk in type 2 DM patients[6], and hyperglycemia accelerates atheroma formation in the setting of diabetic dyslipidemia.[5,10] Atherogenic indices are lipid indices calculated from traditional lipid profiles, and reported to be more sensitive in cardiovascular risk assessment.[11]

Because of the central role played by insulin resistance in the pathogenesis of cardiometabolic disease, it confers an increased risk of type 2 diabetes and cardiovascular disease.[1,7,12] Several surrogate indices have been developed to assess insulin resistance.[13,14] These indices, like Glucose/insulin ratio, Homeostatic model assessment of insulin resistance (HOMA-IR) and Quantitative insulin sensitivity check index (QUICKI), amongst others are alternative to Euglycemic-Hyperinsulinemic Clamp which is the gold standard approach for measuring insulin resistance, but rarely used in clinical practice and epidemiological studies because it is laborious and requires intravenous infusions.[13,14]

Various patterns of dyslipidemia have been described in Type 2 diabetes mellitus. Low HDL-C, elevated triglycerides, and elevated small dense LDL-C, termed "Diabetic Dyslipidemia", have been reported by a few studies in DM patients.[1,7] This study aimed to establish the most common dyslipidemia pattern in type 2 DM, atherogenicity and associated insulin resistance. The findings may enhance targeted therapy in tackling dyslipidemias and help to reduce the risk of CVD in DM.

MATERIALS AND METHODS

This was a comparative cross-sectional study. It involved 141 consenting type 2 diabetic patients aged 15–90 years who met the inclusion criteria for the study group. Another 140 healthy, age and sex-matched individuals from the community were used as controls, making a total of 281 study participants. The study was conducted at the endocrinology clinic of Bowen University Teaching Hospital in Ogbomoso.

The sample size was calculated based on two population mean formulae using G-Power statistical free software version 3.1, by considering the following assumptions: 95% confidence level (2-tailed, α=0.05), 80% power (β=0.20), the ratio of sample size (T2DM/control) was 1:1, effect size (d) was 0.36 and 10% anticipated nonresponse rate. The sample size was determined to be 141 for the study group and age and sex-matched 140 healthy controls, thus, a total of 281 study participants were included in this study to enhance representativeness.

A multistage sampling technique was used: Stage 1: The researcher made use of information on patients' cards to sort out those who were being managed for diabetes only at the endocrinology clinic; Stage 2: Involved listing eligible patients determined by assessing their serum urea, creatinine, and urinalysis; Stage 3: The first eligible respondent was selected by simple random sampling through the balloting method. Then, a systematic random sampling technique was used to select subsequent respondents (*K*th respondent) using the sampling interval obtained from the patient's daily lists throughout the investigation at the endocrinology

clinic.

Sociodemographic and clinical data of the DM and control participants were collected using a semi-structured interviewer-administered questionnaire, which was used to obtain important information like age, gender, ethnicity, and anthropometric measurements, which included height, weight, hip and waist circumference, blood pressure, and body mass index (BMI).

Ten millilitres of venous blood were collected after overnight fasting from the ante-cubital vein after cleaning with methylated spirit. Each sample collected was separated into three bottles (Lithium Heparin, KEDTA, and fluoride oxalate). The KEDTA bottle was used for glycated haemoglobin (HbA1c). Blood in lithium Heparin and fluoride oxalate bottles was separated into plasma by centrifugation and stored frozen for biochemical analysis. Fluoride oxalate was used for fasting plasma glucose (FPG). Biochemical parameters, including fasting lipids (TC, TG, HDL-C and, LDL-C), HbA1c, and FPG, were measured using a JENWAY6305 and UNISPEC Chemistry semi-auto analyser. Insulin was analysed using a surgifield SM-MR96A. Commercially available ready-to-use kits by Randox Laboratories Limited, Crumlin, United Kingdom, were used. The assays were carried out according to the manufacturer's instructions. LDL-C was estimated using Friedewald's formula.[15]

Data Analysis: The data were checked for completeness and consistency. Statistical Package for the Service Solution (SPSS) version 26 software (IBM Corporation, SPSS, Inc., IL, USA) was used for the analysis. Results were reported as frequencies and percentages for categorical variables and mean ± standard deviation (SD) for normally distributed continuous variables. Statistical differences between the groups were determined by the Pearson chi-square test for categorical variables and the Student t-test for continuous variables. The association between continuous variables was obtained using Pearson's correlation. The level of significance was set at a p-value less than 0.05.

Ethical Approval and Consent to Participate. Ethical approval was obtained from the Bowen University ethical review committee. Written informed consent was obtained from each participant after an adequate explanation. All information gathered was kept confidential

Definition of Terms[15,16,17,18,19]

Dyslipidemia

According to the National Cholesterol Education Program Adult Treatment Panel III, dyslipidemia was defined as elevated LDL-C >130 mg/dl, TC >200 mg/dl mmol/L, fasting TG >150 mg/dl, and/or HDL-C lower than 40 mg/dl in men or 50 mg/dl in women.

Pattern of dyslipidemias

Isolated dyslipidaemia = high TC, high LDL-C, high TG or low HDL-C occurring in isolation with the other parameters within normal range.

Two-combined parameter dyslipidaemia = combination of any two of the serum lipid profile abnormalities. For example, high TC and TG, high TC and low HDL, and high TG and LDL.

Mixed dyslipidaemia = combination of any three or all four abnormal serum lipid profile parameters.

The Atherogenic Indices:

Atherogenic Index of Plasma (AIP) = log TG/HDL-C ratio (Low risk <0.1, intermediate 0.11-0.24, high risk>0.24)

Castelli's Risk Index I (CRI-I) = TC/HDL-C ratio (Increased risk >4)

Castelli's Risk Index II (CRI-II) = LDL-C/HDL-C ratio (Increased risk>3)

Atherogenic Coefficient (AC) = [(TC- HDL)/HDL] or [(Non-HDL)/HDL] ratio. (>3 Abnormal).

Insulin Indices[13,14]

Fasting glucose/Insulin ratio (mg/L÷μIU/mL): Insulin Resistance <7≥ Insulin Sensitive

HOMA-IR = [fasting insulin (μIU/mL)]×[fasting glucose (mmol/L)]/22.5

Insulin Sensitive <2 ≥ Insulin Resistance

QUICKI =1/[log fasting insulin, μIU/mL)+log (fasting glucose, mg/dL)]

Insulin Resistance<0.35≥Insulin Sensitive

HbA1c: Good glycemic control <6.5%≥ Poor glycemic control[20,21]

RESULTS

There was a corresponding increase in the prevalence of DM with an increase in age: The highest prevalence, 51(36.2%) was observed in the sixth decade of life (70 years and above), followed closely by the age group (60 – 69 years) with 37(26.2%). Others were 32(22.7%), 12(8.5%) and 9(6.4%) for 50- 59 years, 40 – 49 years and < 36 years, respectively. A higher prevalence of 90(63.8%) was observed in females compared to 51(36.2%) in male gender. Table 1

Higher prevalence of dyslipidemias was found among the diabetic patients, 114(80.9%), compared with 75(53.6%) in the control group, and the difference was statistically significant, p <0.001. There was an incremental difference in the prevalence of dyslipidemias along decades of life, with the highest percentage, 45(88%), observed in the seventh decade (< 70 years). This was closely followed by age 60 – 69 years, 31(83.8%). The age group (≥39 years) had the lowest percentage, 6(66.7%). Others were 9(75.0%) and 23(71.9%) for ages 40 - 49 years and 50 -59 years, respectively, and the difference was not statistically significant with a p-value of 0.287. Similar trends were also observed in the control group, except that the difference was statistically significant, with a p-value of 0.017, 18(40.9%), 27(52.9%), 16(66.7%), 8(53.3%), 6(100.0%) for ≥39, 40 – 49, 50 – 59, 60 – 69, ≥70 years, respectively. Table2a

The most common single dyslipidemia observed in both groups was LDL-C, with percentages of 75.9%(n=107) and 44.3%(n=62). The mean value of abnormal total cholesterol was 87(61.7%) among subjects, while it was 46(32.9%) for controls. The combined dyslipidemia pattern had the highest percentage among subjects, with a value of 52(45.6%) observed. It was followed by mixed dyslipidemias with a value of 50(43.9%). Isolated dyslipidemias had the lowest prevalence, with a value of 12(10.5%). A high TC, high LDL-C and low HDL-C combination had the highest prevalence among type 2 diabetic patients. In the control group, combined dyslipidemias had the highest percentage of 42(56.0%), followed by isolated dyslipidemias with a prevalence of 25(33.3%). Table2b

In the mean values for anthropometric measures, significant differences were observed in BMI with values of 27.44±5.39 and 25.98±5.22 for Subjects and controls, respectively, with a p-value of 0.021. In the same vein, mean Systolic BP was significantly higher in the subjects (134.40±20.01mmHg) than in the controls (123.59±14.40mmHg) with a p-value of <0.001. Other parameters showed no significant difference. Table3a

The mean value of total cholesterol was significantly higher among subjects, with a value of (220.79±63.32mg/dl) compared with that of controls (175.99±55.67mg/dl), with a p-value of <0.001. The mean value of HDL-C level showed a significant difference between the subjects, with a value of (63.25±38.88mg/dl) compared with controls (74.10±30.57mg/dl) (p-value of 0.010). The mean plasma level of LDL-C was significantly higher in the subjects, with a value of (184.55±36.41mg/dl) compared with the controls (93.49±53.78mg/dl) (p-value 0.014). Triglycerides showed no significant difference. In assessing the atherogenic indices, the values of CRI-I (4.68±3.28), CRI-II (3.92±6.16) and AC (3.69±3.27) in subjects were significantly higher than those of controls CRI-I 2.62±1.06), CRI-II (1.44±0.94) and AC (1.62±1.06), with a p-value <0.001. The valve for AIP in Controls (-0.21±0.43) was higher than controls (-0.025±0.41), but the difference was not significant, with a p-value=0.505. Table3b.

Comparing insulin sensitivity among the participants, fasting plasma glucose (7.27±4.24), glycated HB (5.45±0.82), and HOMA-IR (0.59±0.38) values were higher than those of the controls; fasting plasma glucose (4.84±0.92), glycated Hb (4.91±1.44) and HOMA-IR (0.43±0.09), while QUICKI for subjects (0.34±0.04) was lower than that of the controls (0.38±0.05). The differences were statistically significant, with a p-value <0.001. Although the values of insulin levels (10.45±11.41) and glucose/insulin ratio (1.19±1.19) for the controls were higher than those of the patients with Insulin (8.09±10.69) and glucose/insulin ratio (1.07±0.84), the differences were not significant. HOMA-IR demonstrated significantly higher values among

subjects (3.56±5.96) than controls (1.71±1.94), with a p-value of 0.001. A significant difference was also observed between subjects (0.34±0.04) and controls (0.38±0.05), p-value <0.001. Table3c.

Cardiovascular risks were assessed among participants. Subjects showed higher percentage in abnormal levels of TC (87; 61.7%), LDL-c (107; 75%) and HDL-C (66; 46.8%) compared with controls, TC (46; 32.9%), LDL-c (62; 44.3%) and HDL-c (13;9.3%) and the differences were significant, p-values<0.001. No significant difference was observed in the triglyceride levels. Statistically significant higher percentage of increased risk of CVD was found among the subjects for CRI-I (74; 52%), CRI-II (73; 51.8%), and AC (75; 53.2%) compared with control CRI-I (15; 10.7%), CRI-II (7; 5.0%) and AC (15; 10.7%), p-values <0.001. No significant difference was observed in AIP between the two groups. Subjects had a significantly higher percentage of high levels of FBG, with a value of 52(36.9%) compared with controls with a value of 1(0.7%), p<0.001. No significant difference was observed in the glycated Hb percentage for poor glycemic control. Greater percentage of subjects demonstrated insulin resistance in HOMA-IR 79(56.0%) and QUICKI 64(45.4%) than controls; HOMA-IR 31(22.1%) and QUICKI 6(4.3%). The differences were statistically significant, with a p-value of <0.001. Table4.

Pearson correlation was used to demonstrate the correlation between HbA1C and parameters for assessing insulin sensitivity among the diabetic group. FPG (r=0.199, p=0.018) and Insulin (r=0.182, p=0.030) showed weak positive correlation with HbA1C. HOMA-IR (r=0.235, p=0.005) demonstrated weak positive correlation with HbA1C. QUICKI (r=-0.196, p=0.020) demonstrated a weak negative correlation with the glycated Hb. The correlations were significant, p<0.05. No significant correlation was however observed in the glucose/insulin ratio. Figure1

Table 1: Age and sex distribution among type 2 DM

Variables	Frequency	Percentage (%)
Sex		
Male	51	36.2
Female	90	63.8
Age group (years)		
<39	9	6.4
40 - 49	12	8.5
50 – 59	32	22.7
60 - 69	37	26.2
≥70	51	36.2

Table 2a: Prevalence of Dyslipidemias and their Age and Sex distribution among the participants.

Variables	Study Participants			Statistics
	Subjects(%)		Controls(%)	
Lipids Level				**χ=23.740**
Normal	27(19.1)		65(46.4)	**df=1**
Dyslipidemias	114(80.9)		75(53.6)	**p<0.001***
Age Group with Dyslipidemias (years)	Subjects (%)		Controls (%)	
≥39	Group	Statistics	Group	Statistics
40 – 49	6(66.7)		18(40.9)	
50 – 59	9(75.0)		27(52.9)	
60 – 69	23(71.9)	LR=4.999**	16(66.7)	LR=12.027**
≥70	31(83.8)	df=4	8(53.3)	df=4
	45(88.2)	p-value=0.287	6(100.0)	p=0.017*
Sex Group with Dyslipidemias		χ=0.990		χ=1.077
Male	39(76.5)	df=1	33(58.9)	df=1
Female	75(83.3)	p value 0.320	42(50.0)	p=0.299

**Statistically Significant **Likelihood Ratio*

Table 2b: Pattern of Dyslipidemias among the Study participants

Pattern	Study Participants	
	Subjects(%)	Controls(%)
High TC	87(61.7)	46(32.9)
High TG	6(4.3)	12(8.6)
Low HDL-C	66(46.8)	13(9.3)
High LDL-C	107(75.9)	62(44.3)
Isolated Dyslipidemias	12(10.5)	25(33.3)
High TC	3(2.6)	2(2.7)
High TG	0(0.0)	1(1.3)
High LDL-C	6(5.3)	15(20.0)
Low HDL-C	3(2.6)	7(9.3)
Combined Dyslipidemias	52(45.6)	42(56.0)
High TC + low HDL-C	1(0.8)	1(1.3)
High TC+ High LDL	34(29.8)	32(42.8)
High TC + High TG	0(0.0)	1(1.3)
Low HDL-C+ High LDL-C	17(14.9)	4(5.3)
Low HDL-C +High TG	0(0.0)	1(1.3)
High LDL-C + High TG	0(0.0)	3(4.0)
Mixed Dyslipidemias	50(43.9)	8(10.7)
High TC +High TG + High LDL-C + Low HDL-C	0(0.0)	0(0.0)
High TC + High TG + low HDL-C	0(0.0)	0(0.0)
High TC + Low HDL-C + High LDL-C	44(38.7)	2(2.7)
High TC + High TG + High LDL-C	5(4.4)	6(8.0)
Low HDL-C + High TG + High LDL-C	1(0.8)	0(0.0)
Total	114(100%)	75(100%)

Table3a: Comparison of Mean ± SD Values of Anthropometric Parameters among Subjects and Controls.

Variables Categories	Mean ± SD	P- value	95% Cl	
			Lower	Upper
Body Mass Index		0.021*	0.222	2.714
Subject	27.44± 5.39			
Control	25.98± 5.22			
Hip circumference		0.891	-3.971	4.567
Subject	102.67±17.61			
Control	102.38±18.73			
Waist Circumference		0.945	-4.382	4.701
Subject	91.65±18.71			
Control	91.49±19.95			
Waist Hip Ratio		0.327	-0.712	0.238
Subject	0.89± 0.12			
Control	1.13± 2.86			
Systolic BP		0.000*	6.721	14.916
Subject	134.40±20.01			
Control	123.59±14.40			
Diastolic BP		0.672	-2.472	3.827
Subject	81.17±15.53			
Control	80.49±10.87			

*BP=Blood Pressure *Statistically Significant*

Table3b: Comparison of Mean ± SD values Lipids and Atherogenic indices among Subjects and Controls.

Variables Categories	Mean ± SD	P- value	95% Cl	
			Lower	Upper
Total Cholesterol	220.79±63.32	0.000*	30.799	58.811
Subject	175.99±55.67			
Control				
Triglyceride		0.158	-28.159	4.606
Subject	49.99±67.79			
Control	61.77±71.68			
HDL- C		0.010*	-19.069	-2.633
Subject	63.25±38.88			
Control	74.10±30.57			
LDL- C		0.014*	18.592	163.538
Subject	184.55±36.41			
Control	93.49±53.78			
AIP		0.505	-0.065	0.132
Subject	-0.21±0.43			
Control	-0.25±0.41			
CRI-I		0.000*	1.486	2.633
Subject	4.68±3.28			
Control	2.62±1.06			
CRI-II		0.000*	1.448	3.520
Subject	3.92±6.16			
Control	1.44±0.94			
AC		0.000*	1.495	2.638
Subject	3.69±3.27			
Control	1.62±1.06			

**Statistically Significant*

Table3c: Comparison of Mean ± SD values of plasma Insulin level and Insulin Sensitivity/Resistance among Subjects and Controls.

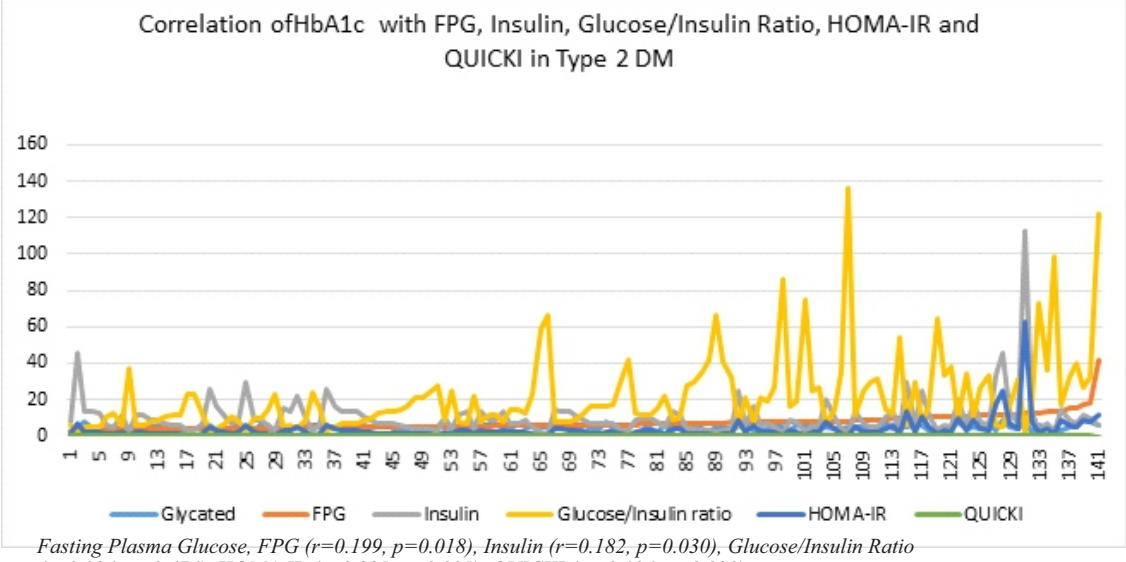
Variables Categories	Mean ± SD	P- value	95% Cl	
			Lower	Upper
FPG		0.000*	1.709	3.153
Subject	7.27±4.24			
Control	4.84±0.92			
Insulin		0.074	-0.235	4.958
Subject	10.45±11.41			
Control	8.09±10.69			
HbA1c		0.000*	-0.809	-0.258
Subject	5.45±0.82			
Control	4.91±1.44			
Glucose/Insulin Ratio		0.326	-2.189	6.560
Subjects				
Controls	21.43±21.50			
	19.24±15.19			
HOMA-IR		0.001*	0.807	2.892
Subjects	3.56±5.96			
Controls	1.71±1.94			
QUICKI		0.000*	-0.456	-0.025
Subjects	0.34±0.04			
Controls	0.38±0.05			

Statistically Significant

Table 4: Cardiovascular risk assessment using lipids and atherogenic, fasting blood glucose and HbA1c among Study participant

Variables	Study Participants		Statistics
	Controls(%)	Subjects(%)	
TC			**χ=23.447**
Normal	94(67.1)	54(38.3)	**df=1**
Abnormal	46(32.9)	87(61.7)	**p<0.001***
TG			χ=2.183
Normal	128(91.4)	135(95.7)	df=1
Abnormal	12(8.6)	6(4.3)	p=0.217
LDL-c			χ=29.265
Normal	78(55.7)	34(24.1)	df=1
Abnormal	62(44.3)	107(75.9)	p<0.001*
HDL-c			χ=48.940
Normal	127(90.7)	75(53.2)	df=1
Abnormal	13(9.3)	66(46.8)	p<0.001*
AIP			χ=0.713
Low Risk	108(77.1)	112(79.4)	df=2
Intermediate Risk	15(10.7)	11(7.8)	p=0.700
High Risk	17(12.1)	18(12.8)	
CRI-I			χ=56.630
Normal	125(89.3)	67(47.5)	df=1
Increased Risk	15(10.7)	74(52.5)	p<0.001*
CRI-II			χ=75.467
Normal	133(95.0)	68(48.2)	df=1
Increased Risk	7(5.0)	73(51.8)	p<0.001*
AC			χ=58.222
Normal	125(89.3)	66(46.8)	df=1
Increased Risk	15(10.7)	75(53.2)	p-value<0.001*
HbA1C			χ=3.486
Good glycemic Control	132(94.3)	124(87.9)	df=1
Poor glycemic control	8(5.7)	17(12.1)	p=0.92
FPG			χ=60.038
Normal	139(99.3)	89(63.1)	df=1
High	1(0.7)	52(36.9)	p<0.001*
Glucose/Insulin Ratio			χ=0.620
Sensitive	120(85.7)	116(82.3)	df=1
Resistance	20(14.3)	25(17.7)	p=0.516
HOMA-IR			χ=0.620
Sensitive	109(77.9)	62(44.0)	df=1
Resistance	31(22.1)	79(56.0)	p<0.001*
QUICKI			χ=63.452
Sensitive	134(95.7)	77(54.6)	df=1
Resistance	6(4.3)	64(45.4)	p<0.001*

Statistically Significant



Fasting Plasma Glucose, FPG (r=0.199, p=0.018), Insulin (r=0.182, p=0.030), Glucose/Insulin Ratio (r=0.036, p=0.676), HOMA-IR (r=0.235, p=0.005), QUICKI (r=-0.196, p=0.020)

Figure 1: Correlation of HbA1c with FPG, Insulin, Glucose/Insulin Ratio, HOMA-IR and QUICKI in Type 2 DM

DISCUSSION

The prevalence of DM was higher in the fifth and sixth decades of life. The sex distribution shows a male-to-female ratio of 1:1.8. The findings were similar to those reported in other studies.[1,7,10,11,12] The prevalence of dyslipidemias was significantly higher in diabetic patients (80.9%) compared to controls (53.6%). Some studies have also documented similar findings.[6,7,10,12] A meta-analysis in Nigeria reported values between 25% and 97.1%.[2] The high prevalence of dyslipidemias in diabetics has been linked to insulin resistances, which affect the normal metabolism of lipids in the body.[3,7]

The most common single lipid derangement was an elevated LDL-C level among participants. Several studies have reported similar findings.[3,6,10] However, combined dyslipidemia was the most common dyslipidemia pattern in diabetics and non-diabetics. The second highest among diabetic patients was mixed dyslipidemias among diabetic patients. Isolated dyslipidemias were observed as the second highest in the control group. That is, more components of lipids were affected among diabetic patients compared to the controls. This may be explained by insulin resistance, which causes several metabolic disorders in diabetic patients.[1,7] Elevated total cholesterols, low HDL-C and elevated LDL-C were observed as the most common Mixed Dyslipidemias among diabetic patients. This is contrary to what has been reported in other climates where elevated triglycerides, low HDL-C and elevated LDL-C were the usual picture termed "Diabetic Dyslipidemias".[5,20,21] The finding of this study may be as a result of paradoxical triglyceridemia occurring in blacks, and this is the reason why the triglyceride level has been reported as a poor predictor of cardiovascular disease among blacks.[22,23,24,25,36]

The mean plasma lipid levels were significantly higher in subjects than in controls, except for the triglyceride level. The study revealed higher mean values of CRI, CRII, and Atherogenic Coefficient in diabetic patients, and these differences were significant. The findings were in agreement with reports from other studies.[27,28,29] AIP did not show a significant difference, likely because it is derived from triglycerides levels, which tend to exhibit paradoxical patterns in individuals of African decent[23,25] Significant differences were also observed in the plasma levels of fasting plasma glucose and glycated haemoglobin between subjects and controls. This may be due to the expected glucose tolerance found in diabetic patients as reported by other studies.[23,25] Although not statistically significant, the level of plasma insulin was found to be lower in subjects compared with the control

group. This may explain the glucose intolerance observed in the subjects.[12,13,32] For the fasting plasma glucose/insulin ratio, no significant difference was observed between the subjects and the control group. This corroborates the reports by previous studies that the G/I ratio does not appropriately reflect the physiology underlying the determinants of insulin sensitivity/resistance.[13,33] On the other hand, more participants demonstrated insulin resistance among the diabetic group using HOMA-IR and QUICKI compared with the non-diabetic group. These indices are more accurate in assessing insulin resistance in epidemiological studies compared to ordinary plasma insulin levels. They can be employed in place of the Euglycemic-Hyperinsulinemic Clamp, which is the gold standard for assessing insulin sensitivity/resistance, but is laborious and not economical for routine clinical use [13,33,35]

In correlating HbA1c with insulin indices, HOMA-IR demonstrated a positive correlation, while QUICKI, on the other hand, showed a negative correlation. The correlations were significant, though weak. Further studies with a larger sample size may be needed to draw a logical conclusion. However, this emphasises the importance of HbA1c in the diagnosis and management of diabetic mellitus, which may serve as a surrogate marker to assess the level of insulin resistance/sensitivity in an individual. It is easier to analyze than HOMA-IR/QUICKI.[35,36,37] Its analysis has been automated and has become point-of-care testing (POCT). Its availability, accessibility and affordability make it suitable as an opportunistic screening test not only for diabetes but also for an insulin sensitivity/resistance test, since fasting is not required.

CONCLUSION

In conclusion, mixed dyslipidemias (elevated total cholesterol, elevated LDL-C and low HDL-C) was the highest form of dyslipidemia pattern observed. Contrary to diabetic dyslipidemia (low HDL-C, elevated triglycerides and elevated LDL-C) reported in other climates. Atherogenic indices-CRI-I, CRI-II, and AC, apart from AIP, were better predictors of CVD than an ordinary lipid profile. HOMA-IR and QUICKI were better tools for the assessment of insulin sensitivity/resistance than plasma glucose and insulin.

Recommendations

Anti-lipid agents targeting a specific pattern of dyslipidemia rather than broad-spectrum agents should be encouraged. Atherogenic indices can be adopted as a means of assessing the risk of CVD and the therapeutic goal for its treatment. Also HbA1c can be adopted as a screening tool for insulin resistance in a large population, not as a diagnostic tool alone.

Study Limitation: This is a hospital-based study; population-based epidemiologic studies with a large sample size may be required to validate and generalize the findings of this study.

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Conflict of Interests

No conflicts of interests