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**EVALUATION OF LIPID PROFILE OF PATIENTS WITH DIABETES BASED ON SEX  
AND AGE GROUPS ATTENDING ABIA STATE UNIVERSITY TEACHING HOSPITAL  
ABA, NIGERIA**

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**ABSTRACT**

The evaluation of lipid profile in diabetic patients attending Abia State Teaching Hospital Aba, was carried out. Venous blood samples were collected from 150 participants who gave consent. This comprises 100 diabetic patients as test and 50 healthy subjects as control. Cholesterol, Triglyceride, High density lipoprotein and Low-density lipoprotein were determined using semi-auto analyzer (Rato 9200). Data from this study were analyzed using statistical package for the social sciences (SPSS). The serum Cholesterol, Triglyceride and High-density lipoprotein were not statistically higher ( $p=0.772$ ,  $p=0.228$  and  $p=0.643$  respectively) in male diabetic patients compared with female diabetic patients. The serum Low density lipoprotein was not statistically lower ( $p=0.378$ ) in male diabetic patients compared with female diabetic patients. Atherogenic index was not statistically higher ( $p=0.354$ ) in male diabetic patients compared with female diabetic patients. From the findings, management of conditions related to cardiovascular disease, atherosclerotic disease, anemia and stress in diabetics may benefit patients if lipid profile, and some hematological parameters are included as part of their routine laboratory investigations.

**KEYWORDS:** Lipid Profile, Diabetes mellitus, *cardiovascular disease, atherosclerotic disease, Sex, Age.*

**INTRODUCTION**

Diabetes mellitus (DM), is a group of metabolic disorders in which there is high blood sugar level over a prolonged period and it is commonly referred to as diabetes.<sup>[1-6]</sup> Frequent urination, increased thirst, and increased hunger are symptoms of high blood sugar.<sup>[7-12]</sup> Many complications are resulted as a cause of untreated diabetes.<sup>[13-18]</sup> Diabetic ketoacidosis, hyperosmolar hyperglycemic state, or death are as a result of acute complications. However, cardiovascular disease, stroke, chronic kidney disease, foot ulcers, and damage to the eyes are included as long-term complication.<sup>[19-24]</sup>

Diabetes mellitus results from either the pancreas is not generating enough insulin or the cells of the body do not respond appropriately to the insulin produced.<sup>[19]</sup>

Reduced HDL cholesterol, a predominance of small dense LDL particles, and elevated triglyceride levels are abnormalities associated with a clustering of interrelated

plasma lipid and lipoprotein in Insulin resistance and form 2 diabetes.<sup>[1]</sup> Every single of these dyslipidemic structures is related with an amplified risk of cardiovascular disease. Increased hepatic secretion of large triglyceride-rich VLDL and impaired clearance of VLDL appears to be of central importance in the pathophysiology of this dyslipidemia. Small dense LDL particles arise from the intravascular processing of specific larger VLDL precursors. Typically, reduced plasma HDL levels in type 2 diabetes are manifest as reductions in the HDL2b subspecies and relative or absolute increases in smaller denser HDL3b and HDL3c. Although behavioral interventions such as diet and exercise can improve diabetic dyslipidemia, for most patients, pharmacological therapy is needed to reach treatment goals.

All the abnormalities occur in many patients despite normal LDL cholesterol levels. These changes are also a feature of the insulin resistance syndrome (also known as

the metabolic syndrome), which underlies many cases of type 2 diabetes.<sup>[1]</sup> In fact, pre-diabetic individuals often exhibit an atherogenic pattern of risk factors that includes higher levels of total cholesterol, LDL cholesterol, and triglycerides and lower levels of HDL cholesterol than individuals who do not develop diabetes. Insulin resistance has striking effects on lipoprotein size and subclass particle concentrations for VLDL, LDL, and HDL.

## MATERIALS AND METHODS

### Study Area

The study was carried out at Abia State University Teaching Hospital (ABSUTH), Aba city in Abia state, South East of Nigeria.

### Study Population

The size of population was calculated using the method of Aroye 2004 with the formula  $n=(z^2pq)/d^2$ , and one hundred and fifty(150) subjects were recruited into the study. This comprises fifty(50)non-diabetic subjects as control and one hundred(100) diabetic subjects as test subjects.

## SELECTION CRITERIA

**INCLUSION;** Those selected are;

- (i) Male and Female subjects of age 18 years to 74years,
- (ii) Diabetic patients with blood sugar 10mmol/l and above,
- (iii) The subjects that gave their consent.

**EXCLUSION;** The excluded subjects are;

- (i) Male and Female subjects below the age of 18 years
- (ii) Subjects of blood sugar below 10mmol/l
- (iii) The subjects that did not give consent.

## LABORATORY PROCEDURES

The reagents were commercially purchased and the manufacturers' standard operating procedures(S.O.P) were strictly adhered to.

### DETERMINATION OF CHOLESTEROL:(enzymatic endpoint method) of Turfitt 1994.

**Assay procedure;** 1000µl of cholesterol liquor and 10µl of sample, standard and control were added into test

tubes respectively, and incubated for 10 minutes at 37°C. The absorbance of standard and test were read(measured) against reagent blank within 60 minutes at 500nm wavelength.

$$\text{Conc. of cholesterol} = \frac{\text{Abs. of test}}{\text{Abs. of Std.}} \times \text{Conc. of Std.}$$

Abs=absorbance

Conc.=concentration

Std=standard.

### DETERMINATION OF TRIGLYCERIDE (enzymatic colorimetric method) of Trinder<sup>[25]</sup>

**Assay procedure;**

1000µl of triglyceride liquor and 10µl of sample, standard and control were added into test tubes respectively, and incubated for 10 minutes at 37°C. The absorbance of standard and test were read(measured) against reagent blank within 60 minutes at 500nm wavelength.

$$\text{Conc. of triglyceride} = \frac{\text{Abs. of test}}{\text{Abs. of Std.}} \times \text{Conc. of Std.}$$

Abs=absorbance

Conc.=concentration

Std=standard.

### DETERMINATION OF HIGH-DENSITY LIPOPROTEIN(HDL)

**ASSAY PROCEDURE;** 100µl of precipitating reagent and 100µl of sample were added into dry test-tube and incubated at room temperature for 5mins. The solution was centrifuged at 3000 rpm (revolution per minute). 50µl of the sample supernatant, standard solution and distilled water (as blank solution) were added into 1000µl of cholesterol reagent respectively. The solutions were incubated for 15mins at 25°C. The absorbance was read at 505nm against the blank within 60mins.

$$\text{Concentration of HDL cholesterol(mg/dl)} = \frac{\text{Abs.T} \times \text{Conc.Std} \times 2}{\text{Abs.Std}}$$

**CALCULATION OF LDL (Freidewald's Formula)**

$$\text{Total cholesterol} - \frac{\text{triglyceride}}{5} - \text{HDL Cholesterol}$$

## RESULTS

**Table 1: Mean ± SD values of lipid profile of patients with diabetes in relation to sex.**

Parameters	Male (n=56)	Female (n=44)	t-value	p-value
Cholesterol(mmol/l)	4.99±0.94	4.93±0.87	0.291	0.772
Lower 95% C.I.	-0.31	-0.30		
Upper 95% C.I.	0.42	0.41		
Triglyceride(mmol/l)	1.90±0.43	1.80±0.42	1.213	0.228
Lower 95% C.I.	-0.06	-0.06		
Upper 95% C.I.	0.27	0.27		
High density Lipoprotein(mmol/l)	1.66±0.44	1.62±0.44	0.465	0.643
Lower 95% C.I.	-0.13	-0.13		

Upper 95% C.I.	0.21	0.21		
Low density Lipoprotein(mmol/l)	2.36±0.67	2.48±0.64	-0.885	0.378
Lower 95% C.I.	-0.38	-0.38		
Upper 95% C.I.	0.14	0.14		
Atherogenic index	0.058±0.01	0.046±0.02		0.354

The serum Cholesterol, Triglyceride and High-density lipoprotein were not statistically higher ( $p=0.772$ ,  $p=0.228$  and  $p=0.643$  respectively) in male diabetic patients compared with female diabetic patients. The serum Low density lipoprotein was not statistically

lower( $p=0.378$ ) in male diabetic patients compared with female diabetic patients. Atherogenic index was not statistically higher( $p=0.354$ ) in male diabetic patients compared with female diabetic patients.

**Table 2: Mean  $\pm$  SD values of lipid profile of patients with diabetes in relation to age group.**

Parameter	35-44yrs (n=10)	45-54yrs (n=23)	55-64yrs (n=32)	65-74yrs (n=35)	f-Value	p-value
Cholesterol(mmol/l)	5.13±0.46	5.22±0.59	5.07±1.07	4.63±0.98	2.516	0.063
Lower 95% C.I.	4.80	4.97	4.68	4.29		
Upper 95% C.I.	5.45	5.47	5.4	4.96		
Triglyceride(mmol/l)	2.11±0.21	2.01±0.28	1.85±0.52	1.67±0.38	5.087	0.003
Lower 95% C.I.	1.96	1.89	1.66	1.53		
Upper 95% C.	2.26	2.13	2.04	1.79		
High density Lipoprotein(mmol/l)	1.43±0.24	1.66±0.31	1.65±0.50	1.66±0.47	0.831	0.480
Lower 95% C.I.	1.25	1.52	1.46	1.49		
Upper 95% C.I.	1.60	1.79	1.82	1.82		
Low density Lipoprotein(mmol/l)	2.74±0.4	2.55±0.61	2.51±0.56	2.14±0.77	3.559	0.017
Lower 95% C.I.	2.45	2.29	2.30	1.87		
Upper 95% C.I.	3.02	2.81	2.70	2.40		
Atherogenic index	0.16±0.06	0.08±0.04	0.04±0.02	0.03±0.09		0.006

There was statistical progressive decrease ( $p=0.003$  and  $p=0.017$ , respectively) in serum Triglyceride and Low-density lipoprotein levels of the studied population in relation to age group. There was no statistical progressive decrease ( $p=0.063$  and  $p=0.480$  respectively) in serum cholesterol and High-density lipoprotein of the studied population in relation to age group. There was statistical progressive decrease( $p=0.006$ ) in the atherogenic index in relation to age group (Table 2).

## DISCUSSION

The higher levels of Cholesterol, Triglyceride and Low-density lipoprotein, could possibly be due to altered metabolism of triglyceride-rich lipoproteins which is crucial in the pathophysiology of the atherogenic dyslipidemia of diabetes. Alterations include both increased hepatic secretion of Very Low-Density Lipoprotein (VLDL) and impaired clearance of VLDL and intestinally derived chylomicrons. An important consequence of retarded clearance is prolonged plasma retention of both VLDL and postprandial chylomicrons as partially lipolyzed remnant particles. These remnants, which include cholesterol-enriched intermediate-density lipoproteins (IDLs), are particularly atherogenic in humans and in a number of animal models.

Increased hepatic production and/or retarded clearance from plasma of large VLDL also results in increased production of precursors of small dense LDL particles. Moreover triglyceride enrichment of the lipolytic products through the action of cholesteryl ester transfer protein, together with hydrolysis of triglyceride and phospholipids by hepatic lipase, leads to increased production of small dense LDL particles. Plasma residence time of these LDL particles may be prolonged because of their relatively reduced affinity for LDL receptors.

## CONCLUSION

From the study since cholesterol, triglyceride and low density lipoprotein were higher in patients with diabetes and are associated with risk factors as coronary artery disease, cardiovascular disease, atherosclerotic diseases, atherothrombotic complications and deficiency of vitamin k, care has to be taken in managing the disease. Also, high density lipoprotein and hemoglobin were lower in studied population, which are not to the advantage of the patients. This is because low HDL is associated with an increased risk of cardiovascular disease

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