

**EVALUATION OF LIPID PROFILE OF PATIENTS WITH DIABETES BASED ON WEIGHTS ATTENDING ABIA STATE UNIVERSITY TEACHING HOSPITAL ABA, NIGERIA**

C. O. Ahiara<sup>1</sup>, I. F. Onyeakplam<sup>1</sup>, D. C. Nwosu<sup>1</sup>, H. U. Nwanjo<sup>1</sup>, C. O. Chima<sup>1</sup>, G. C. Anyanwu<sup>1</sup>, F. C. Emengaha<sup>1</sup>, O. C. Okamgba<sup>2</sup>, J.O. Kabiri<sup>1</sup>, I. O. Onuh<sup>1</sup>, I. C. Amobi<sup>1</sup>, A. U. Obi<sup>3</sup>, Emmanuel Ifeanyi Obeagu<sup>4\*</sup>

<sup>1</sup>Department of Medical Laboratory Science, Imo State University, Owerri, Imo State, Nigeria.

<sup>2</sup>Department of Medical Laboratory Science, Abia State University Uturu, Abia State, Nigeria

<sup>3</sup>Department of Anatomy and Neurobiology, Imo State University Owerri, Imo State, Nigeria.

<sup>4</sup>Department of Medical Laboratory Science, Kampala International University, Uganda.



\*Corresponding Author: Emmanuel Ifeanyi Obeagu

Department of Medical Laboratory Science, Kampala International University, Uganda.

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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, High density lipoprotein and Low-density lipoprotein were not statistically lower ( $p=0.397$ ;  $p=0.537$  and  $p=0.568$  respectively) in 53-64kg compared with 65-74kg of the studied population in relation to weight. The serum Triglyceride, was not statistically higher ( $p=0.890$ ) in 53-64kg compared with 65-74kg of the studied population in relation to weight. The atherogenic index was not statistically higher ( $p=0.657$ ) in 53-64kg compared with 65-74kg of the studied population in relation to weight. From the findings, management of conditions related to cardiovascular disease, arteriosclerotic 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, Weights, Diabetes Mellitus, Cardiovascular Disease, Artherosclerotic Disease.

**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;

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

**EXCLUSION:** The excluded subjects are;

- Male and Female subjects below the age of 18 years
- Subjects of blood sugar below 10mmol/l
- 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.

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

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 the studied population in relation to weight.**

Parameter	53-64kg (n=25)	65-74kg (n=75)	t-value	p-value
Cholesterol(mmol/l)	4.82±0.74	5.00±0.96	-0.851	0.397
Lower 95% C.I.	-0.60	-0.60		
Upper 95% C.I.	0.23	0.23		
Triglyceride(mmol/l)	1.86±0.43	1.84±0.42	0.139	0.890
Lower 95% C.I.	-0.18	-0.18		
Upper 95% C.I.	0.20	0.20		
High density				
Lipoprotein(mmol/l)	1.58±0.31	1.64±0.46	-0.620	0.537
Lower 95% C.I.	-0.26	-0.26		
Upper 95% C.I.	0.13	0.13		
Low density				
Lipoprotein(mmol/l)	2.33±0.70	2.42±0.65	-0.573	0.568

Lower 95% C.I.	-0.39	-0.39	
Upper 95% C.I.	0.21	0.21	
Atherogenic index	0.07±0.14	0.05±0.04	0.657

The serum Cholesterol, High density lipoprotein and Low-density lipoprotein were not statistically lower ( $p=0.397$ ;  $p=0.537$  and  $p=0.568$  respectively) in 53-64kg compared with 65-74kg of the studied population in relation to weight. The serum Triglyceride, was not statistically higher( $p=0.890$ ) in 53-64kg compared with 65-74kg of the studied population in relation to weight. The atherogenic index was not statistically higher( $p=0.657$ ) in 53-64kg compared with 65-74kg of the studied population in relation to weight.

## DISCUSSION

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 with 65-74kg studied population according to weight. There was significant progressive decrease in triglyceride in the studied population according to duration of disease.

## CONCLUSION

From the study since cholesterol, triglyceride and low density lipoprotein were higher in studied population and are associated with risk factors as coronary artery disease, cardiovascular disease, arteriosclerotic 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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