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SERUM LIPID ABNORMALITIES IN TYPE 2 DIABETES MELLITUS

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ABSTRACT
Aim: Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. The present study was conducted to determine the lipid profile of diabetes in Delta State.
Methods: Serum total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein (VLDL) as well as Body mass index (BMI) were determined in two hundred subjects. One hundred were diabetic and the other one hundred were apparently healthy individuals used as controls.
Results: The TC, TG, LDL-C, VLDL-C as well as BMI of diabetes were observed to be significantly high ($P<0.05$) when compared with control subjects. HDL-C in diabetes was observed to be significantly low ($P<0.05$) when compared with control subjects.
Conclusion: The result indicates dyslipidemia in diabetes. Adequate and proper management of diabetes to reduce dyslipidemia and further complications are therefore recommended.

Keywords: Diabetes mellitus, Body Mass Index, Lipid profile

INTRODUCTION

Diabetes mellitus (DM) is a heterogenous condition reflecting different metabolic disorder accompanied by a variety of complications This is characterized by hyperglycemia due to absolute or relative deficiency of insulin (Ajala et al; 2009).Lack of insulin whether absolute or relative affects the metabolism of carbohydrate, protein, fat, water and electrolytes (Choudhury et al., 2011). Insulin affects many sites of mammalian lipid metabolism. It stimulates synthesis of fatty acid in liver, adipose tissue and in the intestine. The insulin has also been reported to increase the cholesterol synthesis. The activity of lipoprotein lipase in white adipose tissue is also increased (Jain and Gupta, 1980) .Several authors have reported lipid abnormalities in Diabetic patients showing increased Total Cholesterol (TC), Triglycerides (TG), Low Density Lipoprotein Cholesterol(LDL-C), Very Low Density Lipoprotein Cholesterol (VLDL-C) and decreased High Density Lipoprotein Cholesterol (HDL-C) (Rasha, 2008,Choudhury et al., 2011). All reports on lipid patterns of DM have failed to address the abnormalities of dyslipidemia.

Due to the paucity of literatures on lipid profile pattern and its abnormalities of DM in Delta State, we therefore sought to provide a baseline of lipid profile pattern for Diabetes in Delta State and by extension, if any, examine the mechanism of lipid profile abnormalities in DM patients.

MATERIALS AND METHODS

Study Area

Four Hospitals located at Agbor, Warri, Ughelli, and Asaba were used for this study and these cover the three senatorial district of Delta State.

Study Population

A total of Two hundred (200) participants were recruited for this study. This comprised one hundred (100) Non- Insulin Dependent Diabetes Mellitus (NIDDM) and one hundred (100) non diabetes individuals as controls. Routine urinalysis was also performed on participants to confirm diabetes and to rule out renal or liver pathology. Informed consent was obtained from participants as well as ethical clearance from the various institution ethics committees.

Collection of Samples

Fasting blood samples were collected by standard venepuncture into plain containers. The blood samples were allowed to clot and then

centrifuged at 3000rpm for 10minutes. The sera were separated into cryovail tubes and kept frozen until required for analysis.

Biochemical Analysis

Serum Total Cholesterol and Triglycerides were analyzed by the enzymatic CHOD-PAP method of Trinder (1969) as modified by Richmond (1973) and HDL-Cholesterol analyzed by the method of Burstein et al., (1970). LDL-Cholesterol, VLDL-Cholesterol was calculated using Friedewald (1972) equation. All test kits used were commercially available and products of Randox Laboratories UK. In all analysis, manufacturer`s instructions were adhered to strictly.

Statistical Analysis

The groups mean ± SD was calculated for each analyte and significant difference between means evaluated using the student t-test. Statistical Package for Social Science SPSS version 16.0 software (SPSS Inc., Chicago, IL USA) for windows was used, with $P<0.05$ considered as statistically significant.

RESULTS

The results of our study show a high Body Mass Index (BMI) in diabetes when compared with controls (Fig.1). Total cholesterol, triglycerides, LDL-C and VLDL-C were increased in diabetes mellitus than in control subjects as shown in Table 1 and Fig. 2.

Table 1: Lipid profile (mmol/l) of diabetic subjects and controls

	Age (years)	BMI	FBS (mmol/l)	TC (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
NIDDM	47.3±0.7	26.4±0.2	11.3±0.4	5.7±0.3	3.2±0.1	1.4±0.1	2.8±0.1	1.5±0.1
Controls	30.6±0.7	22.1±0.3	4.7±0.1	3.8±0.3	1.3±0.1	2.0±0.1	1.2±0.1	0.6±0.1
P value	P<0.05	P<0.05	P<0.05	P<0.05	P<0.05	P<0.05	P<0.05	P<0.05

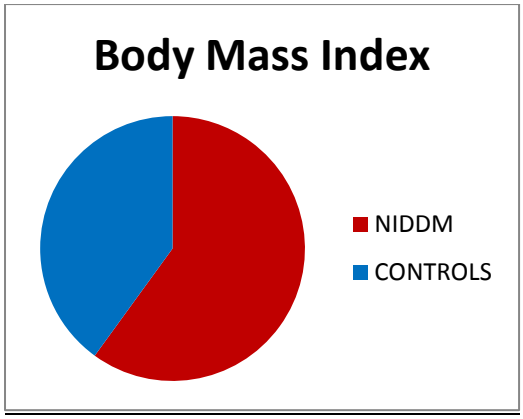

Fig. 1: BMI of Diabetic and Non Diabetic subjects in Pie Chart

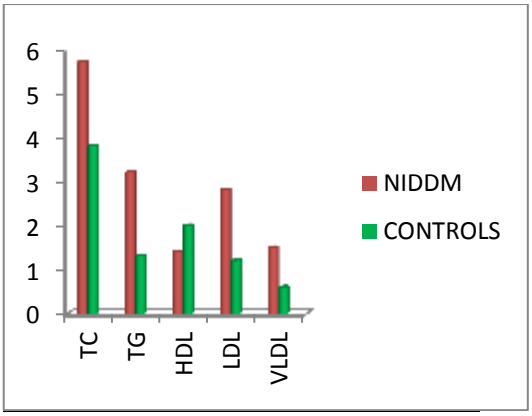

Fig.2: Lipid Profile of Diabetic and Non Diabetic subjects in Bar Chart

DISCUSSION

Lipid and lipoprotein abnormalities are common in the diabetic population due to the effects of insulin deficiency and insulin resistance on key metabolic enzymes. Glucose tolerance, insulin resistance and plasma insulin levels have been implicated in abnormal plasma lipoprotein levels (Simons et al., 2001) and hyperinsulinemia has been linked to the development of atherosclerotic vascular complications (Harris, 2000). The results of this study show statistically significant increased total cholesterol in diabetes patients when compared with control subjects. This is in tandem with the work of Onyemelukwe and Stafford (1981), Bello- Sani et al (2007), and Rasha (2008). Rasha (2008) suggested in his work that the increased level of serum cholesterol in diabetes may be attributed to decrease in muscular exercise or inhibition of cholesterol catabolism but Choudhury et al., (2011) in their work attributed rise in plasma cholesterol level to an increase in plasma

concentration of VLDL-C and LDL, which may be due to hepatic production of VLDL or decreased removal of VLDL-C and LDL from the circulation. Triglycerides level was found to be significantly increased in diabetic patients when compared with controls. This is in agreement with Onyemelukwe and Stafford (1981), Bello- Sani et al., (2007) and Rasha (2008).This is attributed to insulin deficiency which results in faulty glucose utilization, causing hyperglycemia and mobilization of fatty acids from adipose tissue (Anita, 1973). The fatty acids from adipose tissue are mobilized for energy purpose and excess fatty acids are accumulated in liver, which are converted to triglyceride (Anita, 1973). LDL cholesterol in diabetes was found to be significantly increased when compared with controls. This is consistent with the findings of Bello- Sani et al., (2007) but at variance with the work of Qureshi and Uddin (2000). This increase in LDL can be attributed to the fact that, insulin increases the number of LDL receptors, so chronic insulin deficiency as found in type 2 diabetes might be associated with a diminished level of LDL receptor. This causes the increase in LDL particles and results in the increase in LDL-cholesterol level in diabetes mellitus (Qureshi and Uddin, 2000). The VLDL cholesterol of diabetes was significantly increased when compared with controls. This agrees with the work of Rasha (2008).Increased VLDL in diabetes may be as a result of the hyperinsulinemia due to non utilization of insulin by the cells leads to increased triglycerides, LDL and VLDL cholesterol. It is known that insulin and growth hormone promote the production of VLDL cholesterol by increasing the production of Apo-E and Apo-B 48 and by stimulating lipolysis in the adipose tissues and triglycerides in the liver (Young and Bremes, 2001). There is significantly decreased HDL cholesterol in Non-Insulin Dependent Diabetes Mellitus (NIDDM) when compared with control subjects. This is in accordance with the work of previous authors (Onyemelukwe and Stafford, 1981, Bello- Sani et al., 2007). Adu (2013) in his work on nephrotics observed decreased HDL-C and attributed it to urinary losses of Lecithin: Cholesterol Acytransferase (LCAT) which leads to severe deficiency and limit the HDL-mediated uptake of surplus cholesterol from extra hepatic tissues. This is also compounded by marked reduction of the hepatic HDL-C receptors. These limitations greatly affect the homeostasis of HDL-C. Low serum HDL-C levels contribute to structural and functional alterations which led to arterial rigidity (Walter, 2009).Studies carried out in animal models and humans (Kuvin et al., 2002) showed that low HDL-C levels are associated with significant endothelial dysfunction and compromised peripheral vasodilatation. This results in hypertension which is one of the hallmark complications in DM (Putnam et al., 2012). The Body Mass Index (BMI) of diabetes was observed to be significantly higher when compared with controls. This is in agreement with Ogbera et al., (2009) who did a work on the lipid pattern of DM patients. BMI has been used to assess the general obesity of an individual. Ogbera and his colleagues (2009) observed that 40% of DM patients are obese while 34% are overweight with all having lipid abnormalities. Several significant metabolic changes take place in obese individuals that are reflected in plasma concentrations. These include increased number of adipocytes, increased synthesis of cholesterol and fatty acids, increased plasma concentration of fatty acids and triglycerides (Thomas and Gillman, 1993). The most marked characteristic in obese individuals is the altered pattern of lipid metabolism by increasing the plasma concentrations of β lipoproteins transporting lipids (Thomas and Gillman, 1993). The concentrations of insulin are also increased in obese individuals due to pancreatic hypertrophy. This development of the pancreas causes further production of insulin which stimulates production of fatty acids in the liver from carbohydrate and increase fat synthesis in adipose tissue (Thomas and Gillman, 1993). The strength of this study is that it contributes to diabetes data in Delta State Nigeria which are sparse. We therefore recommend lipid profile as well as BMI as a retinue of tests required for the proper management of these diabetes patients to avoid complications.

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REFERENCES

Adu EM (2013). Serum lipid profile abnormalities among patients with nephrotic. International Journal Medicine Biomedical Research 2(1):13-17.

Ajala MO, Ogunro PS, Idogun SE, Osundeko O. (2009). Relationship between plasma antioxidant status and leptin in controlled and non-controlled diabetic non-obese women. International Journal of Endocrinology Metabolism 4: 214-221.

Anita FP (1973). Clinical Nutrition. 2nd ed. R Dayal, Delhi Pp 642-646

Bello – Sani F, Bakari AG, Anumah FE (2007). Dyslipidaemia in persons with type 2 diabetes mellitus in Kaduna, Nigeria. International Journal Diabetic Metabolism 15: 9-13.

Burstein M, Scholnick HR, Morfin R (1970). Rapid method for the isolation of lipoproteins from human serum by precipitation with polyamions. Journal lipid Research 11**:** 583 – 593.

Choudhury MRS, Alam T, Rahman AKMS (2011). Studies on lipid profile in patients with non insulin dependent diabetes mellitus. KYAMC Journal 2(1):123-127.

Friedewald WT, Levy RT, Fredickson DS (1972). Estimation of the concentration of LDL – Cholesterol without use of plasma ultracentrifuge. Clinical Chemistry 18**,** 499 – 520.

Harris MI (2000). Healthcare and Health status and outcomes for patients with type 2 diabetes. Diabetes care 23:754-758.

Jain AP, Gupta DP (1980). Study of blood lipid in Diabetics without any manifest vascular complications. Journal Diabetic Association India 199: 29-34.

Kuvin JT, Ramet ME, Patel AR, Pandian NG, Mendelsohn ME, Karas RH (2002). A novel mechanism for the beneficial vascular effects of high-density lipoprotein cholesterol: enhanced vaso-relaxation and increased endothelial nitric oxide synthetase expression. American Heart Journal 144:165-172

Ogbera AO, Fasanmade OA, Chinenye S, Akinlade A (2009). Characterization of lipid parameters in diabetes mellitus – a Nigerian report. Internal Archives Medicine 2:19

Onyemelukwe GC, Stafford WL (1981). Serum lipids in Nigerians: the effect of diabetes mellitus. Tropical Geographical Medicine 33: 323-328.

Putnam K, Shoemarker R, Yiannikouris F, Cassis LA (2012). The renin-angiotensin system: a target of and contributor to dyslipidemias, altered glucose homeostasis, and hypertension of the metabolic syndrome. American Journal Heart Circulation Physiology 302: 1219-1230.

Qureshi BH, Uddin I (2000). Dyslipidaemia and diabetes in Al- Qassim region, Saudi Arabia. Diabetes International 10:1

Rasha SN (2008). A study of serum lipid profile in obese NDDM patients. Journal Al-Nahrain University 11 (3):106-110.

Richmond W (1973). Cholesterol enzymatic colorimetric test CHOP–PAP method of estimation of total cholesterol in serum. Clinical. Chemistry 191:1350-1356.

Simons LA, Simons J, Friedlander T, McCallum J (2001). Cholesterol and other lipids predict coronary heart disease and ischemic stroke in the elderly but in those below 70years. Atherosclerosis 12: 173-181.

Thomas JH, Gillham B (1993). An example of metabolic disturbance: Obesity. In Wills' Biochemical Basis of Medicine (2nd edn), Butterworth-Heinemann, London. Pp 507-516

Trinder P (1969). Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Annual Clinical Biochemistry 6: 24-27.

Walter M (2009). Interrelationships among HDL, metabolism, aging, and atherosclerosis. Arteriosclerosis Thrombotic Vascular Biology 29: 1244-1250

Young DS, Bremes EW (2001). Specimen Collection and other pre-analytical chemistry: In Tietz Fundamentals of clinical chemistry, 5th edn. Burtis CA, Ashwood ER. (Eds) India: WB Saunders.