**Serum Lipids in Diabetic and Hypertensive Patients Attending Clinic in Aba Metropolis, South-Eastern Nigeria**

Adikema N. A1٭ U. L. Udodirim2

1Department of Medical Laboratory Science, Rivers State University of Science and Technology, Port Harcourt, Nigeria.

2Pathology Department Abia State University Teaching Hospital, Aba Nigeria

٭Corresponding Author: [adikemandu@yohoo.com](mailto:adikemandu@yohoo.com), PO Box 10801, Port Harcourt, Nigeria



**Received 17 November 2013**  **Accepted 30 March 2014**

**Abstract**

Systolic and diastolic blood pressures and body mass index were carried out in 150 patients attending Abia State Teaching Hospital, Aba, South-Eastern part of Nigeria, and in another 50 persons from the same locality, who were neither hypertensive nor diabetic (controls); bringing to total number of participants to 200. The result showed that the mean total cholesterol (TC) 6.73±1.19 mm/L, triglyceride (TG) 2.66±0.83 mm/L, low density lipoprotein cholesterol (LDL-C)3.84±1.07mm/L, and fasting blood glucose (FBS)10.19±3.69mm/L were significantly higher (P<0.05) in hypertensive diabetic subjects compared with the control subjects with values: TC 4.59±0.94mm/L, TG 1.59±0.33mm/L, LDL-C 2.8±0.49mm/L. The systolic and diastolic blood pressures were also significantly higher (P<0.05) in hypertensive diabetics 172.00±12.21 mmHg, and 105.50±9.96mm/Hg, respectively, compared with the control subjects 120.56±13.79mmHg, and 76.8±8.5mmHg respectively. The mean serum levels of high density lipoprotein cholesterol (HDL-C) in hypertensive and diabetic patients: 0.89±0.58mm/L was significantly lower at (P<0.05) than of control: 1.71±0.40mmHg. The mean levels of TC, TG and LDL-C, were significantly higher (P<0.05) in hypertensive non diabetic subjects compared with normal healthy subjects studied. The HDL-C of non diabetic hypertensive subjects: 0.96±0.45mm/L was significantly lower at (P< 0.05) than values obtained in control subjects:. 1.71±0.40mm/L. The mean glucose level showed no significant difference (P>0.05) between the hypertensive non diabetic subjects and control subjects. No significant difference was found in the mean body mass index (BMI) of the hypertensive, diabetic and control subjects. The findings, which provide data from the South –Eastern part of Nigeria, agree with literature report on effect of diabetes and hypertension on the blood level of these parameters in persons from other localities. Of particular interest is the level of HDL-C and TC in both hypertensive and diabetic patients which is suggestive of the higher risk of developing cardiovascular disease. The results underscore the necessity of carrying out complete lipid assay and FBS in patients who present either of these conditions. This should be in addition to looking out for other clinical manifestations of the diseases and following up treatment.

**Key Words**: Blood Glucose, Blood Pressure, Diabetes, Hypertension

**ـــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــــ**

**Introduction**

I

n serum lipid molecules are present in combination with proteins. Such lipid-protein complexes are called lipoprotein. This combination promotes solubility of lipid in an aqueous environment [1, 2]. Lipoproteins have a core of insoluble (non polar) cholesterol esters and Triglycerides. The inner core is surrounded by proteins (apolipoprotiens), phospholipids and free cholesterol with their water soluble groups facing outwards [3, 4]. Lipoproteins are classified according to their density, composition and electrophoresis mobility ([5]. Density is determined by ultracentrifugation and depends on the relative content of protein and lipids. The increasing order of lipoproteins are: chylomicrons, very low density Lipoprotein (VLDL), low density lipoprotein (LDL) and high density lipoprotein (HDL) ( [6]. Electrophoresis mobility is determined by electrophoresis and depends on the charge resulting from the apoprotein content [4]. Electrophoresis of normal plasma separates the lipoproteins into alpha-lipoproteins (HDL) pre-beta lipoproteins (VLDL) and beta-lipoproteins (LDL). Chylomicrons remain at the origin. The molecules in the lipoproteins are also called “apoproteins”. The main classes of apoprotein are apo A, apoB, apo C, apo D and apo E. They have different subunits or subgroup that have been identified ( [7]. Some of the apoprotiens especially apo B are incorporated into the lipoprotein but others are interchangeable freely between lipoproteins. The main classes of apoprotein are apo A, apo B, apo C, apo D and apo E. they have difference subunits or subgroup that have been identified [7]. Some of the apoprotiens especially apo B are incorporated into the lipoprotein but others are interchangeable freely between lipoproteins. In health, apoproteins fulfill a number of roles in the body. They are involved in: activating important enzymes in the lipoprotein metabolic pathway, promoting the solubility of lipids in plasma and enhance the stability of the lipoprotein particles, necessary for lipoproteins secretion by hepatic and intestinal cells and facilitating the uptake of lipoproteins, into cells through their recognition, by specific cells surface receptors.

In physiological fluid and in tissues, most lipid molecules are present in combination with proteins. Such lipid-protein complexes are called lipoprotein. This combination promotes solubility of lipid in an aqueous environment [1],[2]. Electrophoresis of normal plasma separates the lipoproteins into alpha-lipoproteins (HDL) pre-beta lipoproteins (VLDL) and beta-lipoproteins (LDL). Chylomicrons remain at the origin [4].

When the consumption of calories exceeds its utilization, there is an accumulation of fat from dietary lipids and endogenous conversion of excess carbohydrates. This presents a risk of various diseases including Type II diabetes [8]. The common method of measuring obesity is by calculating the individuals’ BMI. In adults, a BMI of 25-29 indicates that a person is overweight, and a BMI of 30 and above means the person is considered to be obese [9].

The clinical features of a patient suffering from diabetes mellitus are results of abnormal glucose metabolism which result from effective insulin deficiency. The insulin deficiency and alterations of normal glucose metabolism will lead to impaired liver and tissue utilization of glucose, and also to hepatic glycogenolysis and gluconeogenesis from protein and fatty acid carbon residues [10]. Diabetes Mellitus is a metabolic disease which is associated with abnormally high glucose levels in the blood as a result of too little insulin or resistance to insulin action or both [11]. Diabetes causes about 5% of all death globally each year, with 80% of people with diabetes living in low and middle income countries. It has a world wide distribution and its prevalence shows both ethnic and regional differences [12, 13]. In diabetes, blood glucose level rises and increased blood glucose level leads to increased glomerular filtration of glucose. The renal threshold is exceeded and glycosuria is present. High urinary glucose concentration produces an osmotic diuresis which will result to polyuria. Cerebral cellular dehydration due to hyperosmolality causes severe thirst (polydipsia). The hyperglycaemia also reduces the resistance to bacterial infection, causing itching 11], [14]. Protein metabolism is disturbed as excess protein is broken down in the process of gluconeogenesis. This will result in loss of potassium, nitrogen and phosphate from the cells to the extra cellular fluid compartment [15]. Fat is also drawn from adipose tissue to provide energy requirements and the patient losses weight. The increased lipolysis of triglycerides in adipose tissue increases free fatty acid flux to the liver and enhanced TG and VLDL-C secretion. The blood glucose level rises and increased blood glucose level leads to increased glomerular filtration of glucose. As the renal threshold is exceeded glycosuria is present. Blood glucose level measured after an overnight fast gives the fasting blood glucose (FBS). A fasting blood glucose test result more than 7mmol/L (125 mg/dL) on two separate occasions, and positive urine glucose test on two separate occasions unequivocally establishes diabetes [16].

Some diabetics are also afflicted by hypertension. Hypertension is referred to as a persistent rise in blood pressure. It is defined as systolic blood pressure level higher than 140mmHg and a diastolic blood pressure higher than 90mmHg [17].

Abnormal lipid values have been reported in diabetes and hypertension. This observation prompted the present study to assess the level of serum lipids and determine the lipoprotein cholesterol mostly depleted in hypertensive and in diabetic patients attending clinic in Aba metropolis. The objective is to produce additional evidence for better management of these diseases.

**Methodology**

A total of 200 adults aged 40-70years participated in this study. Of these, 50 were persons diagnosed to be hypertensive and diabetic; 60 were diagnosed to be diabetic and not hypertensive; another 40 were also diagnosed to be hypertensive but not diabetic, and the remaining 50 were healthy volunteers who were confirmed to be neither hypertensive nor diabetic. All the 150 persons attended clinic of the medical out-patient department of Abia State University Teaching Hospital Aba (ABSUTH) and were referred for tests for the first time. They had not undergone treatment before sample collection. The healthy persons were volunteer staff and students of ABSUTH who admitted not being on drugs. Persons under medication were also excluded. All the 200 participants (subjects) gave informed consent, after receiving due explanation of the purpose for the investigation. Ethical clearance was sought and obtained for the study from ABSUTH, Aba before commencement of the study.

He subjects were asked to fast overnight before samples were collected. The blood samples collected for measurement of serum total cholesterol and lipoproteins were placed into plain containers, left to coagulate spontaneously and centrifuged at 3,000 revolutions per minute for 3 minutes before separation of serum. Samples were analyzed soon after separation, or stored at 4oC for 1-8 days before analysis. Samples that could not be analyzed within this period were kept frozen.

Measurements of blood pressure, height and weight of subjects were done under standard conditions at the time of sample collection. All assays were performed using standard operating procedures (SOP) for glucose (FBS) and lipoproteins (TC, TG, VLDL-C, LDL-C, and HDL-C) [1, 4, 26-28]. The physical measurements and blood samples were taken and analyzed between July and November 2010, using LAB-TECH Electronic Controlled Photo Colorimeter for absorbance readings.

**Statistical Analysis**

The data generated were entered and analyzed using the Z test for two sample means to determine whether any significant difference exists between two sample means for each of the parameters with regard to hypertensive diabetics and non hypertensive non diabetics (controls); hypertensive non diabetics, and non hypertensive diabetics versus controls, respectively. The computation was done using the SPSS package.

**Results**

Table 1 shows the result of the mean ± standard deviation (SD) of the age in years, systolic and diastolic blood pressure, TC, TG ,LDL-C, and HDL-C of subjects measured in hypertensive diabetics and non-hypertensive non diabetic (control) subjects. The result of the study showed that the mean values of systolic blood pressure, diastolic blood pressure, TC, TG, LDL-C as well as FBS were significantly higher (P < 0.05) in hypertensive diabetics compared with normal healthy individuals. The result in this Table also showed that the mean level of HDL-C was significantly lower (P<0.05) in hypertensive diabetics compared with the normal healthy individuals studied. In addition, there were no significant difference (P>0.05) in age, and body mass index.

Table.2 shows the results of the same parameters measured for established hypertensive non diabetics and normal health subjects. The result shows that the mean levels of systolic blood pressure, diastolic blood pressure, TC, TG and LDL-C were significantly higher (P<0.05) in hypertensive non diabetic individual compared with their control subjects. Also, the mean concentration of HDL-C was significantly lower (P<0.05) in hypertensive non diabetics when compared with the age matched control subjects. However, FBS, age in years and BMI mean values showed no significant differences (P>0.05) between hypertensive non diabetes and normal healthy individual.

Table 3 shows the results of the mean value of the biochemical and physical parameters measured in non hypertensive diabetic subjects and those of normal healthy subjects (control). The result shows that the mean levels of FBS, TC, TG and LDL-C were significantly higher (P>0.05) in non hypertensive diabetics compared with their age matched control. Moreover, the mean level of HDL-C was significantly lower (P<0.05) in non hypertensive diabetic subjects as against their control counterpart. There were no significant differences (P> 0.05) in systolic blood pressure, diastolic blood pressure and BMI between the non hypertensive diabetics and the control.

Table.4 compares the result of the biochemical and physical parameters measured in non hypertensive diabetic and hypertensive non diabetics. The result shows that there were no significant differences (P>0.05) in the mean levels of all the parameters measured between diabetic and hypertensive subjects, except for glucose which significantly increased (P<0.05) in diabetic, and blood pressure (diastolic and systolic) which were significantly increased (P<0.05) in hypertensive subjects.

Figure 1 is the summary of the mean ± SD of serum lipids concentrations of the different study groups.

**Table 1** Result (mean ± standard deviation) for parameters measured in Hypertensive Diabetic and Non-hypertensive non diabetic (control) subjects.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Parameters | Hypertensive  diabetics  n = 50 | Non-hypertensive, non diabetics  n = 50 | z-statistic | z-critical | P-value | Remark |
|
| AGE (years) | 62.10 ±12.36 | 61.76 ± 12.37 | 0.1375 | 1.9600 | >0.05 | NS |
| SYSTOLIC B.P(mmHg) | 172.00±12.21 | 120.56 ± 13.79 | 19.7510 | 1.9600 | <0.05 | S |
| DIASTOLIC B.P(mmHg) | 105.50 ±9.96 | 76.8 ± 8.5 | 15.4995 | 1.9600 | <0.05 | S |
| BMI (kg/m2) | 29.37 ± 3.37 | 28.55 ± 4.07 | 1.0952 | 1.9600 | >0.05 | NS |
| FBS (mmol/L) | 10.19 ± 3.69 | 4.31 ± 0.74 | 10.9599 | 1.9600 | <0.05 | S |
| TC (mmol/L) | 6.73 ± 1.19 | 4.59 ± 0.94 | 9.9807 | 1.9600 | <0.05 | S |
| HDL-C (mmol/L) | 0.89 ± 0.58 | 1.71 ± 0.40 | -8.2673 | 1.9600 | <0.05 | S |
| TG (mmol/L) | 2.66 ± 0.83 | 1.59 ± 0.33 | 8.4317 | 1.9600 | <0.05 | S |
| LDL-C (mmol/L) | 3.84 ± 1.07 | 2.8 ± 0.49 | 9.3806 | 1.9600 | <0.05 | S |

**Keys:** NS: No significant differences in mean levels, SD: Standard deviation, S: Significant differences in mean levels, BMI: Body mass index

**Table 2** Result (mean ± standard deviation) for parameters measured in non diabetic hypertensive and non-hypertensive non diabetic (control) subjects.

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| Parameters | Hypertensive non diabetic  n = 40 | Non-hypertensive,  non diabetic  n = 50 | | z-statistic | z-critical | P-value | Remark |
| AGE (years) | 62.03 ± 12.71 | | 61.76 ± 12.37 | 0.0995 | 1.9600 | >0.05 | NS |
| SYSTOLIC B.P(mmHg) | 169.37 ± 9.14 | | 120.56 ±13.79 | 20.1092 | 1.9600 | <0.05 | S |
| DIASTOLIC B.P(mmHg) | 104.37 ±10.63 | | 76.8 ± 8.5 | 13.3435 | 1.9600 | <0.05 | S |
| BMI (kg/m2) | 29.34 ± 2.65 | | 28.55 ± 4.07 | 1.1802 | 1.9600 | >0.05 | NS |
| FBS (mmol/L) | 4.58 ± 0.74 | | 4.31 ± 0.74 | 1.6939 | 1.9600 | >0.05 | NS |
| TC (mmol/L) | 6.21 ± 1.16 | | 4.59 ± 0.94 | 7.1360 | 1.9600 | <0.05 | S |
| HDL (mmol/L) | 0.96 ± 0.45 | | 1.71 ± 0.40 | -8.0949 | 1.9600 | <0.05 | S |
| TG (mmol/L) | 2.52 ± 0.84 | | 1.59 ± 0.33 | 6.5907 | 1.9600 | <0.05 | S |
| LDL (mmol/L) | 3.61 ± 0.91 | | 2.8 ± 0.49 | 8.3211 | 1.9600 | <0.05 | S |

**Table 3** Result (mean ± standard deviation) for parameters measured in non hypertensive diabetic and Non-hypertensive non diabetic (control) subjects.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Parameters | Non hypertensive diabetics  n = 60 | Non-hypertensive, non diabetics  n = 50 | z-statistic | z-critical | P-value | Remark |
|
| AGE (years) | 63.95 ± 11.42 | 61.76 ± 12.37 | 0.9572 | 1.9600 | >0.05 | NS |
| SYSTOLIC B.P(mmHg) | 122.03 ± 12.01 | 120.56 ± 13.79 | 0.5914 | 1.9600 | >0.05 | NS |
| DIASTOLIC B.P(mmHg) | 78.75 ± 9.08 | 76.8 ± 8.5 | 1.1619 | 1.9600 | >0.05 | NS |
| BMI (kg/m2) | 29.12 ± 3.64 | 28.55 ± 4.07 | 0.7181 | 1.9600 | >0.05 | NS |
| FBS (mmol/L) | 9.77 ± 3.16 | 4.31 ± 0.74 | 12.9722 | 1.9600 | <0.05 | S |
| TC (mmol/L) | 6.41 ± 0.98 | 4.59 ± 0.94 | 9.8987 | 1.9600 | <0.05 | S |
| HDL (mmol/L) | 0.93 ± 0.46 | 1.71 ± 0.40 | -9.4270 | 1.9600 | <0.05 | S |
| TG (mmol/L) | 2.56 ± 0.77 | 1.59 ± 0.33 | 8.6990 | 1.9600 | <0.05 | S |
| LDL (mmol/L) | 3.67 ± 0.77 | 2.8 ± 0.49 | 10.3880 | 1.9600 | <0.05 | S |

**Table 4** Result (mean ± standard deviation) for parameters measured in Non hypertensive diabetic and hypertensive non diabetic subjects.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Parameters | Non hypertensive diabetic  n = 60 | Hypertensive non diabetic  n = 40 | z-statistic | z-critical | P-value | Remark |
|
| AGE (years) | 63.95 ± 11.42 | 62.03 ± 12.71 | 0.7725 | 1.9600 | >0.05 | NS |
| SYSTOLIC B.P(mmHg) | 122.03 ± 12.01 | 169.37 ± 9.14 | -22.3353 | 1.9600 | <0.05 | S |
| DIASTOLIC B.P(mmHg) | 78.75 ± 9.08 | 104.37 ± 10.63 | -12.5049 | 1.9600 | <0.05 | S |
| BMI (kg/m2) | 29.12 ± 3.64 | 29.34 ± 2.65 | -0.3928 | 1.9600 | <0.05 | NS |
| FBS (mmol/L) | 9.77 ± 3.16 | 4.58 ± 0.74 | 12.2470 | 1.9600 | <0.05 | S |
| TC (mmol/L) | 6.41 ± 0.98 | 6.21 ± 1.16 | 0.9239 | 1.9600 | >0.05 | NS |
| HDL (mmol/L) | 0.93 ± 0.46 | 0.96 ± 0.45 | -0.4540 | 1.9600 | >0.05 | NS |
| TG (mmol/L) | 2.56 ± 0.77 | 2.52 ± 0.84 | 0.2215 | 1.9600 | >0.05 | NS |
| LDL (mmol/L) | 3.67 ± 0.77 | 3.61 ± 0.91 | 0.3507 | 1.9600 | >0.05 | NS |

**Figure 1** Mean Serum Lipids concentration of the different study gro

HD = Hypertensive diabetic

ND = Non hypertensive diabetic

HND = Hypertensive non diabetic

**Discussion**

Diabetes and hypertension are important metabolic disorders in Nigeria [10]. The prevalence of the two disease states are on the increase worldwide [18]. Some diabetics are also afflicted with hypertension. The study of the serum lipids in affected persons showed (Table 1 and Table 2) that the mean values of systolic blood pressure, diastolic blood pressure, TC, TG and LDL-C were higher in hypertensive diabetics and hypertensive non diabetics compared with normal healthy individuals, and the HDL-C was significantly lower in the two conditions. This finding is in line with earlier report of [19]. The workers also reported that the concentration of HDL-C level was lower in hypertensive diabetics and hypertensive non diabetics compared with the control subjects. The study also showed that the mean level of TC, TG, LDL-C, and FBS were higher in non hypertensive diabetics when compared with the control subjects. The lower HDL-C may result from the inability of the liver to synthesize HDL under both conditions (of diabetes and hypertension). These observations were consistent with previous reports of [10] [19], [20], that dyslipidaemia is usually present in diabetes mellitus and in hypertension. Dyslipidaemia in diabetes mellitus is due to both insulin deficiency and insulin resistance that affects enzymes and Pathways of lipid metabolism [10]. Lipoprotein lipase (LPL) is the main enzyme for the catabolism of chylomicrons and LDL particles which enables them to form remnants that are cleared by Apo E or Apo B receptors in the liver. The LPL is an insulin dependent enzyme and insulin resistance will lead to increased triglyceride levels with decreased catabolism of triglycerides rich lipoprotein [22]. Hypertension even in the absence of diabetes mellitus is known to cause insulin resistance [7], [20]. Insulin resistance occurs when the inner wall of the artery is bombarded with an elevated pressure due to hypertension. The excessive stretching and tension on the wall leads to cracking. The cracks increase the filtration of plasma lipids in the intimal cells. The result of insulin resistance is chronic hyperglycaemia and increased lipolysis which then results in elevated levels of lipids in the plasma [18],[23]. The result of insulin resistance is chronic hyperglycaemia and increased lipolysis that result in elevated levels of lipids in the plasma [14],[18]. Moreover, the higher serum levels of TC, TG and LDL-C found among the diabetic hypertensive, than non hypertensive diabetics and non diabetic hypertensive (Tables 1, 2, 3, 4) patients as against their age matched control indicates that these patients are at a high risk of developing cardiovascular diseases such as coronary artery disease, myocardial infarction and angina pectoris [22]. The lowered concentration of HDL-C in diabetic hypertensive, non-hypertensive diabetics and non-diabetic hypertensive as shown in these Tables also points to the danger of coronary artery disease in these conditions. Values obtained for BMI as presented in the Tables showed no significant difference in the subjects studied, though the values for hypertensive diabetics are slightly higher. This may be due to the similar life style of the subject, and the fact that the subjects were at the onset of the diseases. Values obtained for BMI as presented in the Tables showed no significant difference in the subjects studied, who can be classified as overweight, though the value obtained for the hypertensive diabetes were slightly higher. This may be due to the fact that most of the subjects are living within Aba metropolis and may be having similar lifestyle.

**Conclusion and Recommendations**

This study has generated data which shows that dyslipidaemia exists in patients at Aba who suffer from both diabetes and hypertension, compared with their age matched control. Similar findings have been reported in the literature [23]. The reports from this study provides another bench mark for evaluating the risks of developing cardiovascular disease in persons from the South Eastern part of Nigeria and elsewhere who may present diabetes, hypertension or both diseases[23],[24]. Laboratory investigations to diagnose diabetes and hypertension should include lipid profile investigation as has been proposed by other authors [25]. Persons who are not suffering from any of these conditions should by mindful of the predisposing factors for both diseases and avoid and control them when they set in due to genetic factors. These include controlling life style, eating adequate food while avoiding excess calories, reducing mental and physical stress by limiting activities as age advances and reporting for regular medical check-up.

**References**

1. Lopez – Virella, M.L (1998) Cholesterol determination in high density lipoproteins separated by three different methods. *Clinical* *Chemistry* *23:455-440*

2. Austin, M. A. , Breslow J. I., Henrekaush, C. and Buring W. (2005) Low density lipoprotein. Subclass, Patterns and Risks of Myocardial infarction. *Journal of Lipid Research*. 270:2010-2020.

3. Baron D. N., Whicher, Z.T. and Lee, K. E. (1993). Carbohydrate Metabolism. *In New Short Text Book of Chemical Pathology* (5th ed). Edward Arnold, London (PP. 46-63).

4. Tietz, N.W., Pruden, E., Ronald, W. and Adrich, J. 2008) Lipids, Lipoproteins and Apolipoproteins. In fundamentals of *Clinical chemistry* (4th ed.). W.B Saunders, (Pp. 968-971). Philadelphia

5. Whitbey, L.G., Smith, A.F. and Beckett, E. (2008) Disorders of Plasma Lipids. In *Lecture notes* on *Clinical chemistry* (3rded.) (Pp. 223-240) Blackwell Scientific Publishers, London.

6. Bucolo, S. and David, H. (1999) Quantitative Determination of serum Triglycerides by use of enzymes. *Clinical chemistry* 25:450 – 455

7. Dokken, B. (2009). The Pathophysiology of Cardiovascular Disease and Diabetes. *American Heart Journal* 156: 361 – 368

8. Al-Shammari Khoja T., and Gad A. (2001) Community based study of obesity among children and adults in Riyadh, Saudi Arabia. *Food and Nutrition Bulletin* 22(2).

9. World Health Organization (1997) Obesity epidemics put millions at risk from diseases WHO/46. Geneva.

10. Bello-Sani, F., Bakari, A and Anumah, F. E. (2007). Dyslipidemia in persons with Type 2 diabetes mellitus in Kaduna, Nigeria. *International Journal Diabetes and Metabolism* 15: 9 -13.

11. Caballero, F. (2007) Prediabetes. *Journal of Clinical Endocrinology* 92: 15 – 16

12. American Diabetic Association (ADA) (2007) Diagnosis and Classification of diabetes mellitus. *Diabetic care* Suppl. 1, 42-47

13. Eli Lilly, A. (2006) *Long Term Complications Managing Your Diabetes*. Eli Lilly and Company, Indianapolis Pp.86 – 89.

14. DCCT Group (1993) The Diabetic Control and Complications Trial Research Group: the effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin dependent diabetes mellitus. *N. Engl J. Med*. 329 (14) 977-986.

15. Badung, L.,H, Isezuo, S.A., and Omotoso B.O. (2003). Comparative Analysis of Lipid profile Among Patients with Type 2 Diabetes Mellitus, Hypertension and Type 2 diabetic hypertensive. *Journal of the National Medical Association* 95(5):328-334

16. Carl, B., Edward, R, and David E.B (2006). Lipids and Lipoproteins In *Tietz Textbook of Clinical Chemistry and Molecular Diagnosis* (4thed.) (pp. 435-437) Saunders Elsevier: 435-437.

17. Nicholas, A. B., Nicki, R.C. and Brain, R. (2006). Diabetes Medllitus. In *Davidson’s Principles and Practice of Medicine* (20th ed.) Churchill Livingstone Elsevier: 805-817

18. Jarret, R. J. (2009). Cardiovascular Disease and Hypertension in Diabetes Mellitus. *Diabetes Metabolism* 20: 547 – 550

19. Sola, Jimoh A. K., Agboola, M., Busari O., Idowu, A. and Adedeji A. (2010). Lipid profile of Type 2 diabetic patients at a rural tertiary hospital in Nigeria. *Journal of Diabetes and Endocrinology, 4: 46-51*.

20. Idogun E., Uniugbe, L., Ogunro, P., Akimola, T. and Famodu D. (2007). Assessment of serum lipids in Nigeria with Type 2 Diabetes Mellitus Complications *Journal of Medical Science* 23: 708 – 712.

21. Rao, V., Lakshmana, D. J. and Kiran D. (2010). Study of lipid profile, serum magnesium and blood glucose in hypertension. *Biology Medicine* 2 (1):6 – 16.

22. Reckless, J. P., Betteridage D. J., Wu P., Payne, B., Galton D. J. (1978) High density and low density lipoproteins and prevalence of vascular diseases in diabetes mellitus. *Br. Med. J.* 1 (6117) :883 – 886.

23. Oyelola, O., Ajayi, A, Babalola, R., Slein, E. (2008) Plasma lipids in Nigerian diabetes mellitus, essential hypertension and hypertensive diabetics, *American Heart Journal.* 96: 1200 – 1205.

24. Al-Adsani A., Memon, A. and Suresh A. (2004) Patterns and determinants of dyslipidaemia in Type 2 diabetes mellitus. *Diabetology* 14: 129 – 135.

25. Merrin P. K. and Elkeles R. S. (1991) Treatment of diabetes: the effect of serum lipids and lipoproteins. *Postgraduate Med. J.* 67(792): 9311-937.

26. Trinder, P.(1990). Determination of blood glucose using an oxidase – peroxidase system with a non- carcinogenic chromogen. *Journal Clinical Pathology*. 40:158-161

27. Roeschlau, P., Bernt, E. and Gruber, J.W.(1974). The use of cholesterol oxidase in determination of serum cholesterol. *Clinical Biochemistry* 12:403.

28. Friedward, W.T., Levy, R.T. and Fredericton, P. S.(2000) Estimation of the concentration of low density lipoprotein cholesterol in serum, without use of Preparative Ultracentrifuge. *Clinical Chemistry* 30: 395 - 400.