

Association of Obesity and Dyslipidaemia with Type 2 Diabetes in Outpatients of Enugu State University Teaching Hospital (ESUTH) in Enugu Nigeria

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Abstract: Obesity is known to be a major risk factor of type 2 diabetes (T2D) and responsible for most lipid abnormalities associated with the disease but limited data on such association are available for diabetic patients of Igbo ethnicity in the South East region of Nigeria. A case-control study involving 72 T2D patients and 75 non-diabetic (ND) patients (control) of Igbo ethnicity was conducted. Demographic and anthropometric data were obtained followed by blood collection for the determination of fasting blood sugar (FBS), total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL). Obesity based on waist circumference (WC) was significantly higher ($p < 0.001$) in T2D patients compared to their non-diabetic counterparts. Similarly, TC, TG and LDL levels were significantly ($p < 0.001$) higher in T2D patients while HDL was significantly lower ($p < 0.001$) in T2D patients compared to the control. The proportion of dyslipidaemia characterized by high TC, high TG, high LDL and low HDL was significantly higher ($p < 0.001$) in T2D patients. BMI correlated positively ($p < 0.05$) with WC, TC, and LDL while FBS correlated positively ($p < 0.05$) with TG but negatively with HDL. In conclusion, dyslipidaemia characterised by hypercholesterolaemia, hypertriglyceridaemia, elevated LDL and reduced HDL, as well as obesity were associated with T2D and correlated with FBS in this population.

Key words: Type 2 diabetes, obesity, Igbo, dyslipidaemia, Enugu.

1. Introduction

According to World Health Organisation (WHO) global diabetes report in 2014, about 422 million adults are diabetic as against 108 million in 1980 indicating an increase from 4.7% to 8.5% [1]. In Africa, about 25 million people are affected with diabetes giving a global prevalence of 7.1% [1] with

Nigeria presenting the greatest disease burden of over 1.2 million people affected [2]. Also, Nigeria has the highest number of people with impaired glucose tolerance with an estimate of 3.85 million [3].

Diabetes is a complex metabolic disease defined by chronic hyperglycaemia and disturbances of carbohydrate, lipid and protein metabolism [4] of which about 90% of all cases are due to type 2 diabetes (T2D) [5]. T2D, commonly termed insulin resistance (IR) diabetes is characterized by impaired insulin secretion and/or insulin action as a result of increase in blood glucose level [6, 7].

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Insulin regulates both carbohydrate and lipid metabolism. It promotes lipid synthesis and suppresses lipolysis such that most cells preferentially utilize carbohydrates instead of fatty acids as the primary source of energy [8]. Hence, high calorie diet may lead to obesity thereby increasing intracellular fat and triglyceride (TG) synthesis [9]. The increase in TG promotes fatty acids to be utilized as the primary source of energy over glucose thereby promoting IR. Thus, abdominal or central obesity which is the accumulation of fat worsens IR and eventually promotes T2D [10, 11]. Several studies have shown body fat and lipid abnormalities to be responsible for increased prevalence of T2D [12, 13].

Dyslipidaemia is a lipid abnormality commonly associated with T2D [14, 15]. Diabetic dyslipidaemia constituting a triad of high levels of TG, total cholesterol (TC) and low density lipoprotein (LDL) as well as reduced level of high density lipoprotein (HDL) molecules [15,16] usually originates from the emergence of IR or insulin deficiency which impairs the actions of enzymes such as cholesteryl ester, lipoprotein lipase etc. involved in apoprotein production or lipid metabolism [17]. As such, high levels of LDL and low HDL may increase the risk of cardiovascular diseases [18, 19] and thus, LDL is considered as the main lipid marker in the estimation of cardiovascular risk and the principal therapeutic target in diabetic subjects.

Though dyslipidaemia is a major risk factor of diabetes and cardiovascular diseases, limited data are available for diabetic patients of Igbo ethnicity in the South East region of Nigeria. Hence, this study evaluated the relationship between obesity and lipid abnormality with T2D in this population.

2. Materials and Methods

2.1 Study Population and Design

This was a case-control study involving T2D patients and non-diabetic (ND) patients of Igbo ethnicity conducted at the Enugu State University

Teaching Hospital (ESUTH) in Enugu Nigeria. Ethical approval was sought from the ethical committee of ESUTH Enugu, Nigeria and the study was conducted in accordance with the Helsinki Declaration. Written informed consent was obtained from all willing patients before recruitment into the study. Only patients above 30 years of age without any critical or emergency health condition or complication and not admitted at the hospital were recruited for the study. Breastfeeding and/or pregnant women as well as HIV positive patients were excluded from the study. T2D patients were those with at least one year history of the disease diagnosed according to the International Diabetic Federation (IDF) criteria while the ND patients who served as the control were patients without hyperglycaemia or diabetes.

2.2 Data and Blood Collection

Demographic information of patients including age, sex, ethnicity, location and disease history was collected using a questionnaire while the systolic blood pressures (SBP) and diastolic blood pressure (DBP) were measured using an automatic sphygmomanometer. The height, weight and waist circumference (WC) of the patients were measured and the body mass index (BMI) was calculated. After interview, 4 mL of blood was collected from each patient using a syringe and transferred into fluoride tubes and EDTA free tubes. The EDTA free tubes were centrifuge at 5,000 rpm for 10 minutes to obtain serum.

2.3 Laboratory Analysis

Fasting blood sugar (FBS) was measured from whole blood in fluoride tubes using an accu-chek glucometer according to the glucose oxidase enzymatic method by Trinder [20]. Serum was used for the determination of total cholesterol (TC), TG, LDL and HDL using diagnostic Kits by Randox Laboratories Ltd, United Kingdom. The TC was determined according to the enzymatic method of

Allain and collaborators [21], TG was determined by the enzymatic method of Esders and Michira [22] and HDL by the precipitation method of Grove [23]. LDL was determined using the Freidwald's [24] formula: $\text{LDL (mg/dL)} = \text{TC} - (\text{TG}/5) - \text{HDL}$.

2.4 Definition and Classification of Obesity and Lipid Profile Indices

The following cut-off values were considered for lipid profile indices and obesity. BMI was classified according to WHO criteria [25]. BMI (kg/m^2) was classified as underweight when < 18.5 , between 18.5 and 24.9 as normal weight, ≥ 25 as overweight and > 30 as obese. Abdominal obesity (cm) was defined as $\text{WC} > 88$ in women and $\text{WC} > 102$ in men.

The various lipids were classified according to the criteria of their respective test kits. TC (mg/dL) level was classified as desirable < 200 , between 200 and 239 as borderline high and ≥ 240 as high. TG (mg/dL) value < 150 as normal, between 150 and 199 as borderline high and ≥ 200 as high. HDL (mg/dL) < 40 was classified as low, from 40 to 60 as normal and ≥ 60 as high. LDL (mg/dL) was considered most desirable when < 100 , from 100 to 129 as normal, between 130 and 159 as borderline high, 160 to 189 as high and ≥ 190 as very high level.

Dyslipidemia was defined using the national cholesterol education program (adult treatment panel III) criteria [26] as TC level greater than 200 mg/dL, LDL greater than 100 mg/dL, TG level greater than 150 mg/dl and HDL cholesterol level less than 50 mg/dL in females and less than 40 mg/dL in males.

2.5 Statistical Analysis

Data were analyzed using Statistical Package for Social Science (SPSS) version 16. Results were expressed as mean \pm standard error of the mean (S. E. M) and frequencies. Pearson chi-square (χ^2) test was used to compare proportional differences of sex, obesity (BMI and WC) and lipid profile indices (TC, TG, HDL and LDL) between diabetic and ND patients.

Parametric independent sample *t*-test was used to compare mean differences of lipid profile indices as well as demographic parameters between T2D patients and ND patients while interaction between study independent variables (patient type and sex) among T2D patients and their non-diabetic counterparts was assessed using 2 way ANOVA. Correlation between various lipid profile indices and risk factors (age, SBP, DBP and FBS) was done using pearson correlation test. A *p*-value less than 0.05 was considered to be statistically significant.

3. Results

3.1 Baseline Characteristic of Participants

As summarized in Table 1, a total of 147 patients participated in the study of which 72 (49.0%) were T2D patients while 75 (51.0%) were ND patients. Among these patients, 53 (36.1%) were male while 94 (63.9%) were female but the differences were not significant ($p = 0.229$) between the T2D and ND patients. The age and FBS were significantly higher ($p < 0.05$) in T2D patients compared to the non-diabetic control while the height, weight, SBP and DBP were not significantly different ($p > 0.05$).

3.2 Obesity and Lipid Profile Indices of Patients

Obesity based on BMI and WC and lipid profile indices (TC, TG, HDL and LDL) were all significantly different ($p < 0.05$) among the T2D and ND patients. BMI, WC, TC, TG and LDL were significantly higher ($p < 0.05$) while the HDL was significantly lower ($p < 0.05$) in T2D patients compared to the control (Table 2). Lipid profile indices did not show any significant ($p > 0.05$) sex differences between T2D patients and their non-diabetic counterparts except for TG which showed sex to interact significantly ($p = 0.010$) with T2D as shown in Table 3.

3.3 Classification of Lipid Profile Indices

As presented in Table 4, the proportion of patients

Table 1 Demographic and clinical characteristics of participants.

	T2D patients	ND patients	Minimum	Maximum	<i>p</i> -value
Age (years)	56.83 ± 1.21	49.03 ± 1.89	26	92	0.001
Height (m)	1.58 ± 0.01	1.61 ± 0.01	1.37	1.90	0.179
Weight (kg)	78.85 ± 3.42	71.52 ± 1.95	35.00	190.00	0.063
SBP (mmHg)	132.69 ± 2.65	132.86 ± 3.23	100	213	0.967
DSP (mmHg)	78.80 ± 1.36	81.76 ± 2.24	58	151	0.240
FBS (mg/dL)	166.71 ± 11.39	65.75 ± 3.79	11.00	520.00	0.000

Results are presented as mean ± S. E.

Table 2 Comparison of obesity and lipid profile in patients.

Parameter	T2D patients	ND patients	Minimum	Maximum	<i>p</i> -value
BMI (Kg/m ²)	31.38 ± 1.41	27.81 ± 0.76	18.20	85.13	0.026
WC (cm)	100.15 ± 1.70	89.37 ± 2.25	36.00	149.00	< 0.001
TC (mg/dL)	291.78 ± 28.90	159.43 ± 7.77	5.28	1,387.07	< 0.001
TG (mg/dL)	241.33 ± 15.443	148.82 ± 7.59	22.99	792.16	< 0.001
LDL (mg/dL)	212.40 ± 29.42	78.96 ± 7.96	1.26	1,302.06	< 0.001
HDL (mg/dL)	33.55 ± 2.14	62.74 ± 5.16	1.29	239.16	< 0.001

Table 3 Sex differences of obesity and lipid profile in patients.

Parameter	T2D patients		ND patients		2 way ANOVA (<i>p</i> -value)		
	Male	Female	Male	Female	Patient type	Sex	Patient type X sex
BMI (Kg/m ²)	30.35 ± 2.06	31.25 ± 1.323	26.25 ± 1.73	29.02 ± 1.42	0.059	0.272	0.575
WC (cm)	100.47 ± 3.80	100.22 ± 2.44	92.59 ± 3.19	87.20 ± 2.62	0.001	0.358	0.403
TC (mg/dL)	205.35 ± 41.25	338.71 ± 26.51	150.80 ± 34.60	170.49 ± 28.43	0.001	0.023	0.089
TG (mg/dL)	200.38 ± 23.84	266.52 ± 15.32	169.82 ± 20.01	136.07 ± 16.43	< 0.001	0.400	0.010
LDL (mg/dL)	136.35 ± 42.44	254.27 ± 27.28	68.09 ± 35.60	91.52 ± 29.25	0.001	0.041	0.169
HDL (mg/dL)	34.07 ± 8.08	33.48 ± 5.19	59.76 ± 6.78	63.97 ± 5.57	< 0.001	0.781	0.713

with general obesity (BMI) was insignificantly higher ($p = 0.171$) in T2D patients (21.1%) compared to ND patients (15%) while the proportion of abdominal obesity (WC) was significantly higher ($p = 0.001$) in T2D patients compared to the non-diabetic counterparts (36.1% vs 21.8%). Majority of patients in the T2D group had high level of TC (> 200 mg/dL) while few patients in the ND group had high level of TC level with a significant difference ($p < 0.001$). Similarly, the proportion of hypertriglyceridaemia and high LDL level were significantly higher ($p < 0.001$) in T2D patients compared to ND patients. On the other hand, low HDL level was more prominent in T2D patients than in ND patients (30.6% vs. 17.0%; $p < 0.001$). The proportion of dyslipidaemia (high TC, High TG, high LDL and low HDL) was higher in T2D patients compared to the ND patients as shown in Fig. 1.

3.4 Association of Study Parameters

As summarized in Table 5, BMI positively correlated significantly ($p < 0.05$) with WC, TC and LDL while FBS significantly ($p < 0.05$) correlated positively with TG and negatively with HDL. Age did not show any significant ($p > 0.05$) correlation with the lipid profile parameters.

4. Discussion

Diabetes mellitus is a metabolic disease characterized by abnormal high level of glucose in blood. Though hyperglycaemia is the principal cause of diabetes, it does not rule out other contributory risk factors such as insulin resistance, obesity, hypertension, dyslipidaemia etc. [27, 28]. Obesity which is excess weigh gain as a result of fats accumulation in the

Table 4 Classification of obesity and lipid profile disorders.

Parameter	Category	T2D (%) [n = 72]	ND (%) [n = 75]	Total (%) [n = 147]	χ^2	π	p-value
BMI	Under weight	2 (1.4%)	0 (0.0%)	2 (1.4%)	6.402	0.209	0.171
	Normal weight	14 (9.5%)	24 (16.3%)	38 (25.9%)			
	Over weight	20 (13.6.8%)	23 (15.6%)	43 (29.3%)			
	Obese	31 (21.1%)	22 (15.0%)	53 (36.1%)			
	ND	5 (3.4%)	6 (4.1%)	11 (7.5%)			
WC	Normal	15 (10.2%)	35 (23.8%)	50 (34.0%)	14.466	0.314	0.001
	Obese	53 (36.1%)	32 (21.8%)	85 (57.8%)			
	ND	4 (2.7%)	8 (5.4%)	12 (8.2%)			
Total cholesterol	Desirable or normal	13 (8.8%)	64 (43.5%)	77 (52.4%)	67.750	0.679	< 0.001
	Borderline	35 (23.8%)	9 (6.1%)	44 (29.9%)			
	High	23 (15.6%)	2 (1.4%)	25 (17.0%)			
	ND	1 (0.7%)	0 (0.0%)	1 (0.7%)			
Triglycerides	Normal	12 (8.2%)	44 (29.9%)	56 (38.1%)	32.789	0.472	< 0.001
	Borderline	14 (9.5%)	15 (10.2%)	29 (19.7%)			
	High	46 (31.3%)	16 (10.9%)	62 (42.2%)			
LDL	Most Desired	6 (4.1%)	50 (34.0%)	56 (38.1%)	59.109	0.634	< 0.001
	Good	24 (16.3%)	16 (10.9%)	40 (27.2%)			
	Borderline	16 (10.9%)	6 (4.1%)	22 (15.0%)			
	High	12 (8.1%)	2 (1.4%)	14 (9.5%)			
	Very High	13 (8.8%)	1 (0.7%)	14 (9.5%)			
	ND	1 (0.7%)	0 (0.0%)	1 (0.7%)			
	Low	45 (30.6%)	25 (17.0%)	70 (47.6%)			
HDL	Normal	21 (14.3%)	24 (16.3%)	45 (30.6%)	18.361	0.353	< 0.001
	High	6 (4.1%)	26 (17.7%)	32 (21.8%)			

ND: not determined; χ^2 : pearson chi-square; π : phi-correlation.

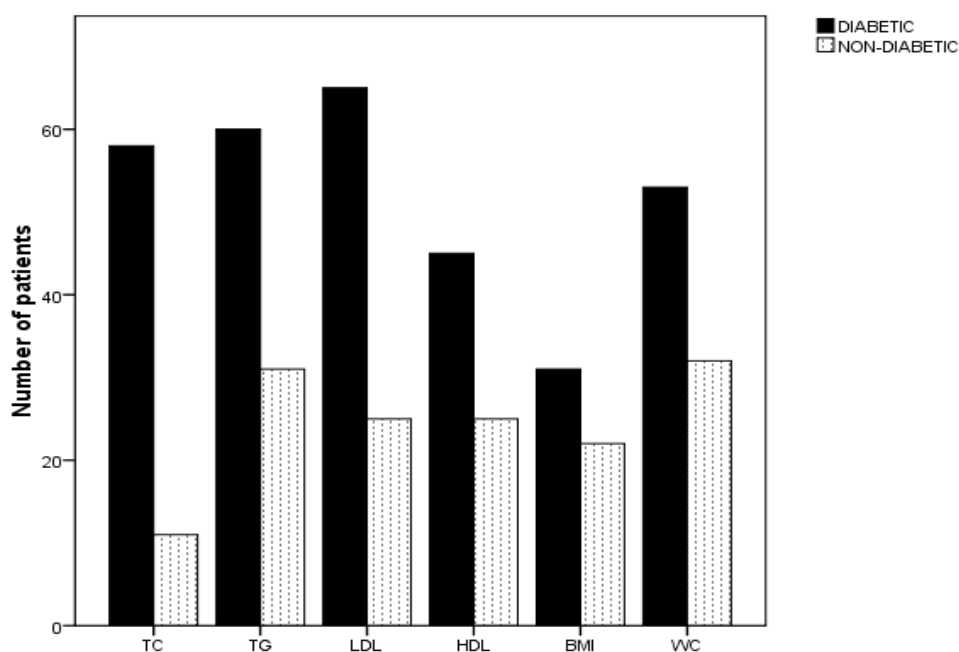


Fig. 1 Frequency of dyslipidaemia and obesity among T2D and ND patients.

Table 5 Correlation of lipid profile indices with age, BMI, WC and FBS.

Parameter	BMI		TC		TG		LDL		HDL	
	<i>r</i>	<i>p</i> -value	<i>r</i>	<i>p</i> -value	<i>r</i>	<i>p</i> -value	<i>r</i>	<i>p</i> -value	<i>r</i>	<i>p</i> -value
Age	0.036	0.680	0.132	0.117	0.105	0.211	0.130	0.122	0.003	0.969
BMI	1	-	0.212	0.014	0.111	0.198	0.215*	0.012	-0.086	0.320
WC	0.430	0.000	0.133	0.126	0.136	0.115	0.136	0.118	-0.073	0.398
FBS	0.047	0.594	0.135	0.112	0.279	0.001	0.113	0.184	-0.195*	0.020

WC: waist circumference; *r*: pearson correlation. Values in bold indicate significant difference ($p < 0.05$).

body or around the waist (abdominal obesity) has been shown as a powerful predictor of metabolic syndrome, diabetes and its complications [29, 30]. In this study, obesity was significantly higher ($p < 0.05$) in T2D patients and thus associated with T2D. This finding is consistent with other studies in Nigeria and across the globe which have shown obesity to be associated with T2D [31-33]. The confirmation of obesity as a result of excess fats particularly saturated lipid to be prevalent in T2D was further supported by our finding as TC was significant higher ($p < 0.05$) in T2D patients. High fat content can modify glucose tolerance and insulin sensitivity thereby promoting insulin resistance, the hall mark of T2D by several mechanisms which include; impaired glucose uptake, prevent glycogen synthesis and reduced proportion of glycogen synthase or increase accumulation of triglycerides in skeletal muscle, altered binding of insulin to its receptors and prevent insulin signaling, [9, 10, 34, 35]. Certain epidemiological studies have shown high saturated fat intake to be associated with insulin resistance and T2D [36, 37]. Also, high level of saturated fatty acids in muscle phospholipids and serum lipids has shown to be associated with higher fasting insulin levels, as well as lower insulin sensitivity with higher risk of developing T2D [38].

Insulin generally regulates both carbohydrate and lipid metabolism. Apart from metabolizing glucose, insulin promotes the mobilization of free fatty acids (FFA) to stored triglycerides in adipocytes and also inhibits lipolysis. In a state of insulin resistance, FFA is released into blood circulation. These FFAs stimulate the assembly and secretion of very-low-density lipoprotein (VLDL); the major

TG-carrying lipoprotein particle from the liver, resulting in excess circulating TG concentration [39]. As such, hypertriglyceridaemia is considered the dominant lipid abnormality in insulin resistance and plays a pivotal role in determining the characteristic lipid profile of diabetic dyslipidaemia [40]. In this study, triglyceride was significantly higher ($p < 0.05$) in T2D patients. Also, the proportion of hypertriglyceridaemia was significantly greater ($p < 0.05$) in T2D than in their non-diabetic patients. This finding concurs with previous studies which have also showed elevated triglyceride level in diabetes [41, 42].

An increase in TG-rich lipoproteins is commonly associated with a reduction in HDL and an increase in LDL levels. LDL level was significantly higher ($p < 0.05$) in T2D compared to ND patients while HDL was significantly lower ($p < 0.05$) in T2D patients. Also, the proportion of patients with high LDL and low HDL was significantly higher ($p < 0.05$) in T2D patients compared to the control. Similar findings have been observed in previous studies [43, 44]. In all, dyslipidaemia was more prevalent in diabetic patients confirming the association between dyslipidaemia and T2D. This association was further supported as FBS, the hallmark for diabetes correlated positively with TG and negatively with HDL levels.

The influence of sex differences in the pattern of serum lipids has been noticed in diabetes. A study on African-Americans showed women with diabetes to have higher LDL and HDL concentrations than their male counterparts [45]. However in an Indian diabetic population, higher level of TC, LDL and TG was found among the male subjects than female [46]. Findings from this study showed sex difference to

significantly ($p < 0.05$) influence the TG level of participants as TG was higher in female than male with T2D while it was higher in male compared to female without diabetes. This finding thus confirms to some extent, sex differences to affect the pattern of lipids in the body.

In conclusion, obesity and dyslipidaemia were associated with T2D as T2D induced hypercholesterolemia, hypertriglyceridaemia, elevated LDL and reduced HDL. The high prevalence of obesity and dyslipidaemia, which is known to be a major risk factor for the development of cardiovascular diseases among diabetic patients suggests optimum care for the management of the disease. This may include regular monitoring of blood sugar and serum lipid profile of diabetic patients for medical intervention. Also, lifestyle changes, such as physical exercise and weight reduction with quality feeding habits should be adopted.

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References

- [1] WHO. 2016. *Global Report on Diabetes*. Geneva: WHO.
- [2] IDF (International Diabetes Federation). 2013. *IDF Diabetes Atlas, Sixth Edition*. Brussels. www.idf.org/diabetesatlas.
- [3] IDF. 2016. "International Working Group on the Diabetic." Foot. http://www.idf.org/webdata/docs/background_info_AFR.pdf.
- [4] Abou-Seif, M. A., and Youssef, A. A. 2004. "Evaluation of Some Biochemical Changes in Diabetic Patients." *Clinical Chemistry Acta* 346: 161-70.
- [5] Shaw, J. E., Sicree, R. A., and Zimmet, P. Z. 2010. "Global Estimates of the Prevalence of Diabetes for 2010 and 2030." *Diabetes Research and Clinical Practice* 87: 4-14.
- [6] Das, S. K., and Elbein, S. C. 2006. "The Genetic Basis of Type 2 Diabetes." *Cell Science* 2: 100-31.
- [7] Maitra, A., and Abbas, A. K. 2005. "Endocrine System." In: Kumar, V., Fausto, N., Abbas, A. K. Robbins and Cotran In *Pathologic Basis of Disease*. 7th ed. Philadelphia, Saunders. 1156-226.
- [8] Le Roith, D., and Zick, Y. 2001. "Recent Advances in Our Understanding of Insulin Action and Insulin Resistance." *Diabetes Care* 24: 588-97.
- [9] Saltiel, A. R., and Kahn, C. R. 2001. "Insulin Signaling and the Regulation of Glucose and Lipid Metabolism." *Nature* 414 (6865): 799-806.
- [10] Shulman, G. I. 2000. "Cellular Mechanisms of Insulin Resistance." *Journal of Clinical Investigation* 106: 171-6.
- [11] HU, F. B., Manson, J. E., Stampfer, M. J. 2001. "Diet, Lifestyle, and the Risk of Type 2 Diabetes Mellitus in Women." *New England Journal of Medicine* 345 (11): 790-7.
- [12] Elinasri, H. A., and Ahmed, A. M. 2008. "Patterns of Lipid Changes among Type 2 Diabetes Patients in Sudan." *Eastern Mediterranean Health Journal* 14 (2): 314-24.
- [13] Risérus, U., Willett, W. C., and HU, F. B. 2009. "Dietary Fats and Prevention of Type 2 Diabetes." *Progress in Lipid Research* 48 (1): 44-51.
- [14] Mooradian, A. D. 2009. "Dyslipidemia in Type 2 Diabetes Mellitus." *Nature Clinical Practice Endocrinology Metabolism* 5: 150-9.
- [15] Goldberg, I. J. 2001. "Diabetic Dyslipidemia: Causes and Consequences." *Journal of Clinical Endocrinology Metabolism* 8 (3): 965-71.
- [16] Smith, S., and Lall, A. M. 2008. "A Study on Lipid Profile Levels of Diabetics and Non-diabetics among Naini Region of Allahabad." *India Turkish Journal Biochemistry* 33 (4): 138-41.
- [17] Taskinen, M. R. 2002. "Diabetic Dyslipidaemia." *Atherosclerosis* 3: (1): 47-51.
- [18] Haffner, S. M. 2004. "Dyslipidemia Management in Adults with Diabetes." *Diabetes Care* 27: 68-71.
- [19] Kannel, W. B., and McGee, D. L. 1979. "Diabetes and Cardiovascular Risk Factors: The Framingham Study." *Circulation* 59: 8-13.
- [20] Trinder, P. 1969. "Determination of Blood Glucose Using 4-Aminophenazone as Oxygen Acceptor." *Journal Clinical Pathology* 22 (246): 1-6.
- [21] Allain, C. C., Poon, L. S., Chan, C. S., and Richmond, W. 1974. "Total Cholesterol Assay." *Clinical Chemistry* 20: 470-1.
- [22] Esders, T. N., and Michira, C. A. 1997. "Triglyceride Estimation." *Journal Biology Chemistry* 254: 2710-2.
- [23] Grove, T. H. 1979. "Grove's Method of High Density Lipoprotein Estimation." *Clinical Chemistry* 25: 560-2.
- [24] Friedwald, W. T., Levy, R. I., and Fredrickson, D. S. 1972. "Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma without Use of

- Preparative Ultracentrifugation." *Clinical Chemistry* 18 (6): 499-502.
- [25] WHO. 1999. *Report of a WHO Consultation: Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complication: Part 1. Diagnosis and Classification of Diabetes Mellitus*. Geneva: WHO.
- [26] Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. 2001. "Executive Summary of the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)." *Journal of American Medical Association* 285: 2486-97.
- [27] HU, F. B. 2011. "Globalization of Diabetes: The Role of Diet, Lifestyle, and Genes." *Diabetes Care* 34: 1249-57.
- [28] CHAN, J. C., Malik, V., and JIA, W. 2009. "Diabetes in Asia: Epidemiology, Risk Factors, and Pathophysiology." *Journal of American Medical Association* 301: 2129-40.
- [29] Yoon, K. H., Lee, J. H., and Kim, J. W. 2006. "Epidemic Obesity and Type 2 Diabetes in Asia." *Lancet* 368: 1681-8.
- [30] Despres, J. P. 2001. "Health Consequences of Visceral Obesity." *Annals of Medicine* 33: 534-41.
- [31] Edo, A. E., and Edo, G. O. 2012. "Prevalence of Obesity in Nigerians with Type 2 Diabetes Mellitus Seen in a Secondary Medical Center." *Annals of Biomedical Sciences* 11: 44-50.
- [32] Fasanmade, O. A., and Okubadejo, N. U. 2007. "Magnitude and Gender Distribution of Obesity and Abdominal Adiposity in Nigerians with Type 2 Diabetes Mellitus." *Nigerian Journal of Clinical Practice* 10: 52-7.
- [33] Hillier, T. A., and Pedula, K. L. 2001. "Characteristics of an Adult Population with Newly Diagnosed Type 2 Diabetes: The Relation of Obesity and Age of Onset." *Diabetes Care* 24: 1522-7.
- [34] Grundleger, M. L., and Thenen, S. W. 1982. "Decreased Insulin Binding, Glucose Transport, and Glucose Metabolism in Soleus Muscle of Rats Fed a High Fat Diet." *Diabetes* 31: 232-7.
- [35] Hedekov, C. J., Capito, K., Islin, H., Hansen, S. E., and Thams, P. 1992. "Longterm Fat-Feeding-Induced Insulin Resistance in Normal NMRI Mice: Postreceptor Changes of Liver, Muscle and Adipose Tissue Metabolism Resembling Those of Type 2 Diabetes." *Acta Diabetologica* 29: 14-9.
- [36] HU, F. B., van Dam, R. M., and LIU, S. 2001. "Diet and Risk of Type II Diabetes: The Role of Types of Fat and Carbohydrate." *Diabetologia* 44: 805-17.
- [37] van Dam, R. M., Willett, W. C., and Rimm, E. B. 2002. "Dietary Fat and Meat Intake in Relation to Risk of 2 Diabetes in Men." *Diabetes Care* 25 (3): 417-24.
- [38] Storlien, L. H., Jenkins, A. B., Chisholm, D. J., Pascoe, W. S., Khouri, S., and Kraegen, E. W. 1991. "Influence of Dietary Fat Composition on Development of Insulin Resistance in Rats. Relationship to Muscle Triglyceride and Omega-3 Fatty Acids in Muscle Phospholipid." *Diabetes* 40 (2): 280-9.
- [39] Ginsberg, H. N. 1996. "Diabetic Dyslipidemia: Basic Mechanisms Underlying the Common Hypertriglyceridemia and Low HDL Cholesterol Levels." *Diabetes* 45 (3): 27-30.
- [40] Adiels, M., Olofsson, S. O., and Taskinen, M. R. 2008. "Overproduction of Very Low-Density Lipoproteins Is the Hallmark of the Dyslipidemia in the Metabolic Syndrome." *Arteriosclerosis and Thromb Vascular Biology* 28 (7): 1225-36.
- [41] West, K. M., Ahuja, M. M., and Bennett, P. H. 1983. "The Role of Circulating Glucose and Triglyceride Concentrations and Their Interactions with Other 'Risk Factors' as Determinants of Arterial Disease in Nine Diabetic Population Samples from the WHO Multinational Study." *Diabetes Care* 6: 361-9.
- [42] Tsutsumi, K., Iwamoto, T., Hagi, A., and Kohri, H. 1998. "Streptozotocin-Induced Diabetic Cynomolgus Monkey Is a Model of Hypertriglyceridemia with Low High Density Lipoprotein Cholesterol." *Biological and Pharmaceutical Bulletin* 21: 693-7.
- [43] Dixit, A. K., Dey, R., Suresh, A., Chaudhuri, S., Panda, A. K., Mitra, A., and Hazra, J. 2014. "The Prevalence of Dyslipidemia in Patients with Diabetes Mellitus of Ayurveda Hospital." *Journal of Diabetes Metabolism Disorders* 13 (58): 1-6.
- [44] Smith, S., and Lall, A. M. 2008. "A Study on Lipid Profile Levels of Diabetics and Non-diabetics among Naini Region of Allahabad." *India Turkish Journal of Biochemistry* 33 (4): 138-41.
- [45] Werk, E. E., Gonzalez, J. J., and Ranney, J. E. 1993. "Lipid Level Differences and Hypertension Effect in Blacks and Whites with Type II Diabetes." *Ethnicity Disease* 3: 242-9.
- [46] Sapna, S., and Alok, M. L. 2008. "A Study on Lipid Profile Levels of Diabetics and Non-diabetics among Naini Region of Allahabad India." *Turkish Journal of Biochemistry* 33 (4): 138-41.