International Journal of Biomedical Research
ISSN: 0976-9633 (Online); 2455-0566 (Print)
Journal DOI:10.7439/ijbr
CODEN: IJBRFA

Original Research Article

Evaluation of fasting lipid profile and glycated hemoglobin in obese subjects at University of Calabar teaching hospital, Nigeria

Agu Chidozie Elochukwu[1], Emeribe Anthony Uchenna[1], **Idris Abdullahi Nasir*[2]**, Effa A. Faith[1], Adamu Babayo[3] and Alphonsus Ekpe Udoh[1]

[1]Department of Medical Laboratory Science, University of Calabar, PMB 1115 Calabar, Cross River State, Nigeria
[2]Department of Medical Microbiology, University of Abuja Teaching Hospital, PMB 228, Gwagwalada, FCT Abuja, Nigeria
[3]Department of Medical Laboratory Science, College of Medical Science, University of Maiduguri, PMB 1069, Maiduguri, Borno State, Nigeria

*Correspondence Info:
Idris Abdullahi Nasir
Department of Medical Microbiology
University of Abuja Teaching Hospital
PMB 228, Gwagwalada, FCT Abuja. Nigeria
E-mail: eedris888@yahoo.com
Phone Number: +2348030522324

Abstract
Background: Obesity is a major public health issue which has been established to be a significant risk factor for several metabolic disorders such as impaired glucose tolerance, dyslipidaemia, and atherosclerosis. This study aimed to evaluate and compare the serum lipid profiles and glycatedhaemoglobin of obese and non-obese adults in Calabar, Nigeria.
Methodology: This was a prospective comparative study that involved quantifying serum lipid profile and glycated hemoglobin (HB1Ac) in seventy (70) obese subjects and thirty (30) non-obese control subjects.
Results: The mean HB1Ac, Total Cholesterol (TC), Low Density Lipoprotein (LDL) and Tryglyceride (TG) for obese subjects were 6.13±2.76%, 4.92±1.23mmol/L, 3.18±1.21mmol/L, 1.21±0.40mmol/L, 128.14±12.65mmHg and 88.56±11.87mmHg respectively. These values were significantly higher than those of the non-obese control subjects whose mean values for HB1Ac, TC, LDL and TG were 5.34±1.15%, 3.08 ± 0.63mmol/L, 1.74 ± 0.54mmol/L, 0.67±0.33mmol/L ($p<0.05$) respectively. The mean High Density Lipoprotein (HDL) value for obese subjects was 1.35 ±0.28mmol/L, this is significantly lower than that of the non-obese control subjects with a mean HDL of 1.77± 0.41mmol/L ($P<0.05$). No significant difference was found on Very Low Density Lipoprotein (VLDL) between the two groups ($P>0.05$). A positive correlation between BMI, HB1Ac, TC, LDL was observed in obese subjects (r=0.341, 0.287, 0.393, $P<0.05$), while a negative correlation was observed between BMI and HDL (r= -0.147, $P<0.05$), WHR and HDL (r= -0.289, $P<0.05$).
Conclusion: Findings from this study show that obese individuals have higher risk to develop cardiovascular related disorders and type II diabetes mellitus if appropriate interventions are not considered.
Keywords: Obese, Glycated hemoglobin, Lipid profile, cardiovascular disorder, Type-II Diabetes mellitus, Calabar

1.Introduction
Obesity as defined by World Health Organization is a condition in which there is excessive fat accumulation in the body, to the extent that the health and wellbeing of the individual is adversely affected[1]. Non-communicable diseases (such as obesity) have overtaken communicable diseases as the leading causes of morbidity and mortality in Nigeria[2][3]. Obesity is the second leading cause of preventable death after smoking worldwide with increasing prevalence in adults and children, it has been considered as one of the serious public health problems of the 21st century[4].

The prevalence of obesity is rising globally, in 2008, about 1.5 billion adults, 20 years and older

were overweight, of this 1.5 billion overweight adults, over 200 million men and nearly 300 million women were obese[5]. In overall, more than one tenth of the world's population are obese and nearly 4 million children under the age of five were overweight in 2010[6].

According to the 2010 WHO survey data on Nigeria, the prevalence of overweight was 26% and 37% in men and women respectively, while the prevalence of obesity was 3% and 8.1% in men and women respectively [7]. Data from the WHO Global InfoBase, based on individuals aged 30 years and above, shows that the prevalence of overweight and obesity together increased by 23% in men and 18% in women, while the prevalence of obesity alone increased by 47% in men and 39% in women, between 2002 and 2010, in Nigeria[7].

The fundamental cause of obesity and overweight is energy imbalance between calories consumed and calories expended[8]. More so, genetic factors, environmental factors, diet, medication, psychological factors, life style preferences and sociocultural practice seems to play a major role in the rising prevalence of obesity worldwide[9].

Obesity poses a major risk to serious diet-related non-communicable diseases including diabetes mellitus, cardiovascular diseases, hypertension, dyslipidaemia, stroke, gall bladder disease, osteoarthritis, sleep apnea and certain forms of cancer such as ovary, breast and colon cancer[5][10]. Combination of energy restriction, exercise, behavioral modifications, drugs and occasionally surgery should help in the management of obesity-related problems[10] however, for any significant progress to be made in the prevention of obesity, a public health approach is urgently needed[11].

Obesity is the leading determinant of dyslipidaemia (abnormal lipid concentrations) and diabetes mellitus[12]. Dyslipidaemia is a major risk factor associated with coronary heart disease, as elevated levels of triglycerides, total cholesterol, low density lipoprotein cholesterol (LDL-C) and low levels of high density lipoprotein cholesterol (HDL-C) are documented risk factors for atherosclerosis[13]. Medical technology has developed lipid profile testing to determine a person's risk of coronary heart disease. The tests make up the lipid panel test and include; finding out the total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides and VDL cholesterol[14].

Body mass index (BMI) is used to classify overweight and obesity and it is defined as a person's weight in kilograms divided by the square of his/her height in meters (kg/m^2)[15]. Based on WHO classification, a BMI of less than 18.5 kg/m^2 is classified as underweight, a BMI of 18.5-24.9 kg/m^2 is classified as normal weight, a BMI of 25.0-29.9 kg/m^2 is classified as overweight, a BMI of 30.0-34.9 kg/m^2 is classified as class I obesity, BMI of 35.0-39.9 kg/m^2 is classified as class II obesity and BMI of greater or equal to 40.0 kg/m^2 is classified as class III obesity.

Glycated hemoglobin is the term used to describe the formation of a hemoglobin compound produced when glucose (a reducing sugar) reacts with the amino group of hemoglobin (a protein)[16]. The glucose molecule attaches non-enzymatically to the hemoglobin to form a ketoamine. The rate of formation is directly proportional to the plasma glucose concentrations. Because the average red blood cell lives approximately 120days, the glycated hemoglobin level at any time reflects the average blood glucose level over the previous 2 to 3 months [14][16]. Therefore, measuring the glycated hemoglobin provides the clinician with a time-average picture of the patient's blood glucose concentration over the past 3 months[16].

Fasting plasma glucose (FPG) and the oral glucose tolerance test (OGTT) are considered to be appropriate tests for diagnosing pre-diabetes and/or diabetes while OGTT is also considered an appropriate test for assessing diabetes risk in patients with impaired fasting glucose (IFG)[17]. As an alternative to these methods, an International Expert Committee, including representatives of the American Diabetes Association (ADA), the International Diabetes Federation (IDF), and the European Association for the Study of Diabetes (EASD), recently recommended evaluating glycosylated hemoglobin (HbA1c) with a cut-off point of ≥6.5% to diagnose diabetes[18]. (The HbA1c of young, lean and healthy subjects is approximately 5.0%[17][18]. This strategy was endorsed and adopted by the ADA in 2010[17][18].

Epidemiological evidence suggests that elevated HbA1c is associated with cardiovascular and ischemic heart disease risk[19]. Both obesity and physical inactivity are considered to play important roles in the prevention and treatment of diabetes, with the ADA[20] recommending that people with HbA1c of 5.7-6.4% undergo moderate weight loss (7% of initial body mass), as well as increasing physical activity to at least 150 min/week of moderate activity. This present study aimed to determine fasting lipid profile and glycated hemoglobin in obese subjects and control subjects, to determine fasting serum lipid profile, glycated hemoglobin, in the various classes

of obesity based on body mass index (BMI) and to determine if there is a relationship between BMI, waist circumference, waist-hip ratio, lipid profile and glycated hemoglobin in obese and non-obese subjects.

2. Materials and methods

2.1 Selection of subjects

A total number of 70 obese subject (BMI $\geq$ 30kg/m^2) of which consisted of 30 males and 40 females within the age range of 20-45 years were used as the test group and 30 apparently healthy non-obese subjects (BMI 18.5-24.9kg/m^2) of which consisted of 10 males and 20 females were used as control group and were within the same age range as the test group.

A structured questionnaire was used to which get data on each individual details of his/her health, family health history, age, sex, occupation, physical activity, eating habit and whether or not on medication that may affect tests results. Subjects reported in the morning after an approximate of 12-hours overnight fast.

2.2 Ethics statement

This study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the human research ethical committees of University of Calabar Teaching Hospital All the subjects gave their written informed consent for inclusion before they participated in the study. All data were analyzed anonymously throughout the study.

2.3 Exclusion criteria

Participants with impaired glucose tolerance (or diabetes) and those with known high blood pressure were excluded.

2.4 Anthropometric measurements

Anthropometric measurements included height, weight, waist circumference and hip circumference. Weight and height were measured with the subjects wearing light clothing's and without shoes. Weight was measured to the nearest kg using a balanced scale, height was measured to the nearest meters using a wall-mounted ruler, with the subjects bare foot, standing with feet together and with head, shoulder, buttocks and heels touching the wall. Waist and hip circumference were measured to the nearest 0.1cm using a flexible but in elastic measuring tape, while the subjects were standing relaxed waist circumference was taken midway between the costal margin and the iliac crest in the mid-auxiliary line around the gluteal region.

Body mass index was calculated for each subject as the ratio of body weight (in kg) and squared height (in meters), BMI (kg/m^2) was used as the index of total (general) obesity. Waist-to-hip ratio (WHR) was calculated by dividing the measurement of the waist (cm) by that of the hip (cm) and was used together with the waist circumference as the index of central obesity.

The following definitions were used: overweight – a body mass index of $\geq$ 25kg/m^2; obese – a BMI of $\geq$ 30kg/m^2; central obesity – a waist circumference $\geq$ 88cm in women or $\geq$ 102cm in men or waist-to-hip ratio $\geq$ 0.90cm in women or $\geq$1.0 in men; normal weight – a body mass index of 18.5-24.9kg/m^2.

2.4 Sample collection

Five milliliters (5ml) of blood was drawn from the antecubital vein. Three milliliters (3ml) was dispensed into plain bottles, capped and allowed to clot at room temperature. The bottle was labeled with subject's name, number and date. After 30 minutes, the serum was separated from the red cell by centrifuging at 5, 000 revolutions per minute for 5 minutes, serum was separated and stored at about 4^{o}C until the day of analysis.

The remaining 2ml of blood was dispensed into a dipotassium ethylene diamine tetra-acetic (EDTA) bottle for glycated hemoglobin estimation. The samples were processed within 24 hours of collection.

2.5 Analytical Methods

Total cholesterol (TC) was determined using a Trider-based (CHOD-PAP) colorimetric end-point assay (CH 3810, Randox Laboratories Ltd, UK). High-density lipoprotein cholesterol (HDL-C) was determined using a direct two-point kinetic assay kit (CH 2652, Randox Laboratories Ltd, UK). Triglycerides (TG) were determined using a Trinder-based (GPO-PAP) colorimetric end point assay (TR 3823, Randox Laboratories Ltd, UK) while kits for the quantitative determination of glycated hemoglobin were from Pointe scientific Inc. Serum LDL-C was calculated according to the Friedewald formula: LDL = TC - HDL - TG/5.0 (mg/dL). All analyses were conducted in accordance with the manufacturers' instructions. More so, methods were controlled and validated using control reagents from kits manufacturers.

2.6 Statistical analysis

Data are presented as mean values with standard deviations and statistical significance was set at the $p \leq 0.05$ level. Comparisons between obese and non-obese were performed with a factorial ANOVA, adjusted by BMI on HbA1c and lipid profiles. Comparisons between obese and non-obese participants, with the three categories of cut-off

points of obesity were conducted with factorial ANOVA.

Associations between HbA1c and fasting lipid profiles were calculated with bivariate correlation, and with partial correlation adjusted. Data analysis was performed using SPSS v19.0 (SPSS inc, Chicago, IL, USA).

3. Results

Anthropometric parameters, fasting serum lipid profile and glycated hemoglobin were determined in 70 obese subjects (BMI 30.0 kg/M^2 and above) and 30 non obese control subject (BMI 18.5 – 24.9kg/m^2).

Table 1 shows the mean age, anthropometric parameter, glycated hemoglobin, lipid profile and blood pressure in obese subjects and control subjects. The result revealed that the mean value of BMI, waist C, Hip C, W-H ratio, HB1Ac, T-C, LDL, TG, SBP and DBP were significantly higher in obese subjects when compared to the control subjects (P<0.05). Control subject showed a significant higher mean HDL when compared to the obese subjects (P<0.05). No Significant difference was observed in mean VLDL between the two group (P>0.05).

Obese subjects were further divided into three (3) classes using BMI, class I when BMI is between 30-34.9kg/m^2, class II when BMI is between 35-39.9kg/m^2 and above. There different anthropometric parameters, lipid profile, glycated hemoglobin and blood pressure were compared alongside that of the control group using one way ANOVA. These comparisons are shown in table 2. Results from table showed that a significant different exists between the four group for BMI, WC, Hip C, WHR, HB1Ac, TC, LDL, HDL, TG, SBP, DBP (P<0.05). No significant difference was seen for VLDL between these four groups (P>0.05). Control group had significantly higher HDL than those of the three (3) obese classes (P<0.05).

Table 3 shows the comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class I and control group. BMI, WC, Hip C, WHR, HB1Ac, TC, LDL, TG, SBP, DBP were significantly higher in obese class I group when compared with that of the control group (P<0.05). Control group had significantly higher HDL than the obese class I group (P<0.05).

Table 4 shows the comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class I and obese class II groups. BMI, WC, Hip C, WHR, TC, LDL, SBP showed a significant higher difference in obese class II group when compared to class I group (P<0.05). HDL was significantly higher in obese class I than in obese class II group (P<0.05). No significant difference exist in WHR, glycated Hb, TG and DBP between these two groups (P>0.05).

Table 5 shows the comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class I and obese class III groups. Obese class III group showed significantly higher BMI, WC, HC, WHR, glycated Hb, T-C, LDL, TG, SBP,DBP when compared to obese class I group (P<0.05). Obese class I group showed significantly higher difference in HDL than obese class III group (P<0.05).

Table 6 shows the comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class II and control group. BMI, WC, HC, TC, LDL, glycated Hb, TC, SBP, DBP showed a significant higher difference in obese class II group when compared to control group (P<0.05). HDL was significantly higher in control group when compared to the obese class II group (P<0.05).

Table 7 shows the comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class II and obese class III groups. No significant difference exists in WC, WHR, HB1Ac, TC, LDL, TG, SBP, DBP between the two groups (P>0.05). There was a significant difference in BMI, HC between the two groups (P<0.05).

Table 8 shows the comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class III and control groups. BMI, WC, HC, TC, LDL, HB1AcTG, SBP, DBP showed a significant higher difference in obese class III group when compared to the control group (P<0.05). HDL was significantly higher in control group when compared to the obese class III group (P<0.05).

Correlation analysis was carried out for anthropometric parameters, glycated hemoglobin and lipid profile in obese subjects. BMI correlated positively with TC, LDL, HB1Ac. In these obese subjects (r = 0.341, 0.287, 0.393, P<0.05). There was a significant negative correlation between BMI and HDL (r = -0.147, P<0.05). Also a significant negative correlation was observed between WHR and HDL (r = 0.289, P<0.05) (fig. 1 -5).

Table 1: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese subjects and control group

Parameters	Obese n = 70 Mean ± SD	Control n =30 Mean ± SD	Calculated t – value	Critical t – value	p - value	Remark
Age (yrs)	34.53 ± 9.36	33.47 ± 3.82	0.55	1.98	>0.05	NS
Height (m)	1.62 ± 0.74	1.68 ± 0.12	3.06	1.98	<0.05	S
Weight (kg)	94.27 ± 15.3l1	64.73 ± 8.94	9.86	1.98	<0.05	S
BMI (kg/M^2)	35.69 ± 4.95	22.34 ± 0.94	14.60	1.98	<0.05	S
Waist c (cm)	106.90 ± 13.52	75.10 ± 4.37	12.57	1.98	<0.05	S
Hip c (cm)	120.09 ± 9.93	90.17 ± 4.03	15.91	1.98	<0.05	S
W-H ratio	0.87 ± 0.072	0.79 ± 0.042	5.21	1.98	<0.05	S
HB1Ac(%)	8.13 ± 2.76	5.34 ± 1.15	5.34	1.98	<0.05	S
T-C (mmol/L)	4.92 ± 1.23	3.08 ± 0.63	7.75	1.98	<0.05	S
LDL(mmol/L)	3.18 ± 1.21	1.74 ± 0.54	6.24	1.98	<0.05	S
HDL (mmol/L)	1.35 ± 0.28	1.77 ± 0.41	6.02	1.98	<0.05	S
TG (mmol/L)	1.21 ± 0.40	0.67 ± 0.33	4.36	1.98	<0.05	S
VLDL (mmol/L)	0.62 ± 0.19	0.34 ± 0.15	1.03	1.98	>0.05	NS
Systolic BP (mmHg)	128.14 ± 12.65	114.00 ± 7.24	2.47	1.98	<0.05	S
Diastolic BP (mmHg)	88.56 ± 11.87	81.67 ± 6.99	2.33	1.98	<0.05	S

S = Significant; **NS** = Not significant

Table 2: Comparison of anthropometric parameters, lipid profile, glycated hemoglobin and blood pressure of the three classes of obesity alongside the control using one way ANOVA

Parameters	ObeseClass I n = 34 (BMI 30 – 34.9kg/m^2) Mean ± SD	ObeseClass II n = 18 (BMI 35 – 39.9kg/m^2) Mean ± SD	ObeseClass III n = 18 (BMI ≥40.0kg/m^2) Mean ± SD	Control n =30	Critical f – value	Calculated f - value	p - value	Remark
BMI (kg/M^2)	31.71 ± 9.78	36.43 ± 1.34	42.44 ± 3.69	22.34 ± 0.94	2.45	7.95	<0.05	S
Waist c (cm)	99.79 ± 11.38	110.06 ± 11.40	117.17 ± 11.74	75.10 ± 4.37	2.45	465.40	<0.05	S
Hip c (cm)	114.35 ± 5.26	118.94 ± 7.31	132.06 ± 8.63	90.17 ± 4.03	2.45	85.40	<0.05	S
W-H ratio	0.85 ± 0.077	0.89 ± 0.074	0.90 ± 0.046	0.79 ± 0.042	2.45	202.65	<0.05	S
HB1Ac(%)	6.01 ± 2.78	6.12 ±2.81	8.69 ± 2.28	5.34 ± 1.15	2.45	12.19	<0.05	S
T-C (mmol/L)	4.51 ± 0.95	5.22 ±1.35	5.39 ± 1.37	3.08 ± 0.62	2.45	13.05	<0.05	S
LDL(mmol/L)	2.79 ± 0.89	3.51 ±1.27	3.58 ± 1.50	1.74 ± 0.54	2.45	25.25	<0.05	S
HDL (mmol/L)	1.42 ±0.28	1.25 ±0.27	1.33 ± 0.26	1.77 ±0.41	2.45	17.08	<0.05	S
TG (mmol/L)	1.01 ± 0.43	1.24 ± 0.39	1.41 ± 0.46	0.67 ± 0.33	2.45	3.42	<0.05	S
VLDL (mmol/L)	0.45 ± 0.19	0.52 ± 0.19	0.48 ± 0.18	0.44 ± 0.15	2.45	0.83	>0.05	NS
SystolicBP (mmHg)	124.85 ± 10.12	130.83 ± 10.37	131.67 ± 17.33	114. 60 ± 7.24	2.45	13.31	<0.05	S
Diastolic BP (mmHg)	85.65 ± 11.89	88.28 ± 10.87	94.33 ± 11.25	81.67 ± 6.99	2.45	5.90	<0.05	S

Table 3: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class I group and control group

Parameters	Obese Class I n = 34 Mean ± SD	Control n =30 Mean ± SD	Calculated t- value	Critical t – value	p - value	Remark
BMI (kg/M^2)	31.71 ± 9.78	22.34 ± 0.94	31.24	1.98	<0.05	S
Waist c (cm)	99.79 ± 11.38	75.10 ± 4.37	11.18	1.98	<0.05	S
Hip c (cm)	114.35 ± 5.26	90.17 ± 4.03	20.45	1.98	<0.05	S
W-H ratio	0.85 ± 0.077	0.79 ± 0.042	3.27	1.98	<0.05	S
HB1Ac(%)	6.01 ± 2.78	5.34 ± 1.15	3.87	1.98	<0.05	S
T-C (mmol/L)	4.51 ± 0.95	3.08 ± 0.62	6.97	1.98	<0.05	S
LDL(mmol/L)	2.79 ± 0.89	1.74 ± 0.54	5.66	1.98	<0.05	S
HDL (mmol/L)	1.42 ±0.28	1.77 ±0.41	4.11	1.98	<0.05	S
TG (mmol/L)	1.01 ± 0.43	0.67 ± 0.33	3.45	1.98	<0.05	S
Systolic BP (mmHg)	124.85 ± 10.12	114. 60 ± 7.24	4.87	1.98	<0.05	S
Diastolic BP (mmHg)	85.65 ± 11.89	81.67 ± 6.99	1.60	1.98	>0.05	NS

S = Significant; **NS** = Not significant

Table 4: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class I group and obese class II groups

Parameters	Obese Class I (n = 34) Mean ± SD	Obese Class II (n = 18) Mean ± SD	Calculated t- value	Critical t – value	p - value	Remark
BMI (kg/M^2)	31.71 ± 9.78	36.43 ± 1.34	11.80	1.98	<0.05	S
Waist c (cm)	99.79 ± 11.38	110.06 ± 11.40	3.90	1.98	<0.05	S
Hip c (cm)	114.35 ± 5.26	118.94 ± 7.31	2.68	1.98	<0.05	S
W-H ratio	0.85 ± 0.077	0.89 ± 0.074	1.68	1.98	>0.05	NS
HB1Ac(%)	6.01 ± 2.78	6.12 ±2.81	0.83	1.98	>0.05	NS
T-C (mmol/L)	4.51 ± 0.95	5.22 ±1.35	2.21	1.98	<0.05	S
LDL(mmol/L)	2.79 ± 0.89	3.51 ±1.27	2.35	1.98	<0.05	S
HDL (mmol/L)	1.42 ±0.28	1.25 ±0.27	2.09	1.98	<0.05	S
TG (mmol/L)	1.01 ± 0.43	1.24 ± 0.39	1.15	1.98	>0.05	NS
Systolic BP (mmHg)	124.85 ± 10.12	130.83 ± 10.37	2.01	1.98	<0.05	S
Diastolic BP (mmHg)	85.65 ± 11.89	88.28 ± 10.87	2.78	1.98	>0.05	NS

S = Significant; **NS** = Not significant

Table 5: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class I group and obese class III groups

Parameters	Obese Class I (n = 34) Mean ± SD	Obese Class III(n = 18) Mean ± SD	Calculated t- value	Critical t – value	p - value	Remark
BMI (kg/M^2)	31.71 ± 9.78	42.44 ± 3.69	15.14	1.98	<0.05	S
Waist c (cm)	99.79 ± 11.38	117.17 ± 11.74	5.18	1.98	<0.05	S
Hip c (cm)	114.35 ± 5.26	132.06 ± 8.63	2.30	1.98	<0.05	S
W-H ratio	0.85 ± 0.077	0.90 ± 0.046	2.60	1.98	<0.05	S
HB1Ac(%)	6.01 ± 2.78	8.69 ± 2.28	2.54	1.98	<0.05	S
T-C (mmol/L)	4.51 ± 0.95	5.39 ± 1.37	2.72	1.98	<0.05	S
LDL(mmol/L)	2.79 ± 0.89	3.58 ± 1.50	2.38	1.98	<0.05	S
HDL (mmol/L)	1.42 ±0.28	1.33 ± 0.26	2.41	1.98	<0.05	S
TG (mmol/L)	1.01 ± 0.43	1.41 ± 0.46	2.31	1.98	<0.05	S
Systolic BP (mmHg)	124.85 ± 10.12	131.67 ± 17.33	2.07	1.98	<0.05	S
Diastolic BP (mmHg)	85.65 ± 11.89	94.33 ± 11.25	2.55	1.98	<0.05	S

S = Significant; **NS** = Not significant

Table 6: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class II group and control groups

Parameters	Obese Class II (n = 18) Mean ± SD	Control (n =30) Mean ± SD	Calculated t- value	Critical t – value	p – value	Remark
BMI (kg/M^2)	36.43 ± 1.34	22.34 ± 0.94	42.78	1.98	<0.05	S
Waist c (cm)	110.06 ± 11.40	75.10 ± 4.37	15.13	1.98	<0.05	S
Hip c (cm)	118.94 ± 7.31	90.17 ± 4.03	17.63	1.98	<0.05	S
W-H ratio	0.89 ± 0.074	0.79 ± 0.042	5.35	1.98	<0.05	S
HB1Ac(%)	6.12 ±2.81	5.34 ± 1.15	4.82	1.98	<0.05	S
T-C (mmol/L)	5.22 ±1.35	3.08 ± 0.62	7.48	1.98	<0.05	S
LDL(mmol/L)	3.51 ±1.27	1.74 ± 0.54	6.70	1.98	<0.05	S
HDL (mmol/L)	1.25 ±0.27	1.77 ±0.41	4.50	1.98	<0.05	S
TG (mmol/L)	1.24 ± 0.39	0.67 ± 0.33	3.12	1.98	<0.05	S
Systolic BP (mmHg)	130.83 ± 10.37	114. 60 ± 7.24	6.62	1.98	<0.05	S
Diastolic BP (mmHg)	88.28 ± 10.87	81.67 ± 6.99	2.57	1.98	<0.05	S

S = Significant; **NS** = Not significant

Table 7: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class II group and obese class III groups

Parameters	Obese Class II(n = 18) Mean ± SD	ObeseClass III(n = 18) Mean ± SD	Calculated t- value	Critical t – value	p - value	Remark
BMI (kg/M^2)	36.43 ± 1.34	42.44 ± 3.69	6.49	1.98	<0.05	S
Waist c (cm)	110.06 ± 11.40	117.17 ± 11.74	1.84	1.98	>0.05	NS
Hip c (cm)	118.94 ± 7.31	132.06 ± 8.63	4.92	1.98	<0.05	S
W-H ratio	0.89 ± 0.074	0.90 ± 0.046	0.41	1.98	>0.05	NS
HB1Ac(%)	6.12 ±2.81	8.69 ± 2.28	1.55	1.98	>0.05	NS
T-C (mmol/L)	5.22 ±1.35	5.39 ± 1.37	0.38	1.98	>0.05	NS
LDL(mmol/L)	3.51 ±1.27	3.58 ± 1.50	0.17	1.98	>0.05	NS
HDL (mmol/L)	1.25 ±0.27	1.33 ± 0.26	0.96	1.98	>0.05	NS
TG (mmol/L)	1.24 ± 0.39	1.41 ± 0.46	1.10	1.98	>0.05	NS
Systolic BP (mmHg)	130.83 ± 10.37	131.67 ± 17.33	0.20	1.98	>0.05	NS
Diastolic BP (mmHg)	88.28 ± 10.87	94.33 ± 11.25	0.96	1.98	>0.05	NS

S = Significant; **NS** = Not significant

Table 8: Comparison of anthropometric parameters, glycated hemoglobin, lipid profile and blood pressure in obese class III group and control group

Parameters	ObeseClass III (n = 18) Mean ± SD	Control (n =30) Mean ± SD	Calculated t- value	Critical t – value	p – value	Remark
BMI (kg/M^2)	42.44 ± 3.69	22.34 ± 0.94	28.52	1.98	<0.05	S
Waist c (cm)	117.17 ± 11.74	75.10 ± 4.37	17.77	1.98	<0.05	S
Hip c (cm)	132.06 ± 8.63	90.17 ± 4.03	22.88	1.98	<0.05	S
W-H ratio	0.90 ± 0.046	0.79 ± 0.042	7.54	1.98	<0.05	S
HB1Ac(%)	8.69 ± 2.28	5.34 ± 1.15	8.31	1.98	<0.05	S
T-C (mmol/L)	5.39 ± 1.37	3.08 ± 0.62	7.97	1.98	<0.05	S
LDL(mmol/L)	3.58 ± 1.50	1.74 ± 0.54	6.14	1.98	<0.05	S
HDL (mmol/L)	1.33 ± 0.26	1.77 ±0.41	4.11	1.98	<0.05	S
TG (mmol/L)	1.41 ± 0.46	0.67 ± 0.33	2.18	1.98	<0.05	S
Systolic BP (mmHg)	131.67 ± 17.33	114. 60 ± 7.24	4.94	1.98	<0.05	S
Diastolic BP (mmHg)	94.33 ± 11.25	81.67 ± 6.99	4.82	1.98	<0.05	S

S = Significant; **NS =** Not significant

Fig 1: Correlation plot of total cholesterol against body mass index in obese subjects

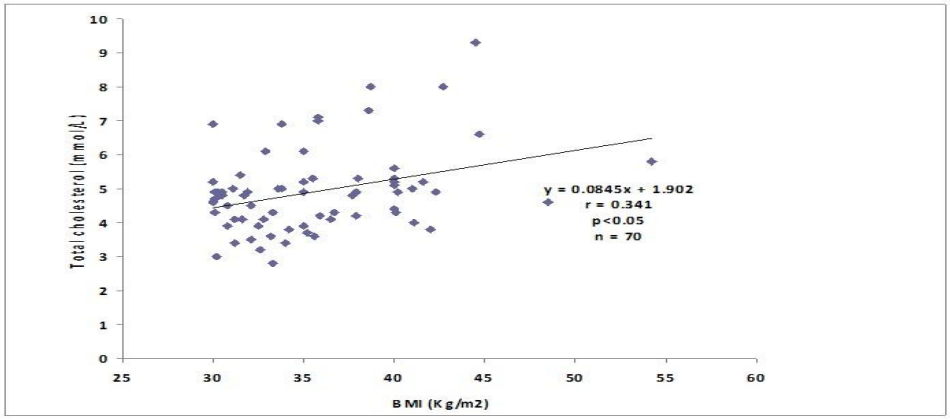


Fig 2: Correlation plot of LDL-cholesterol against body mass index in obese subjects

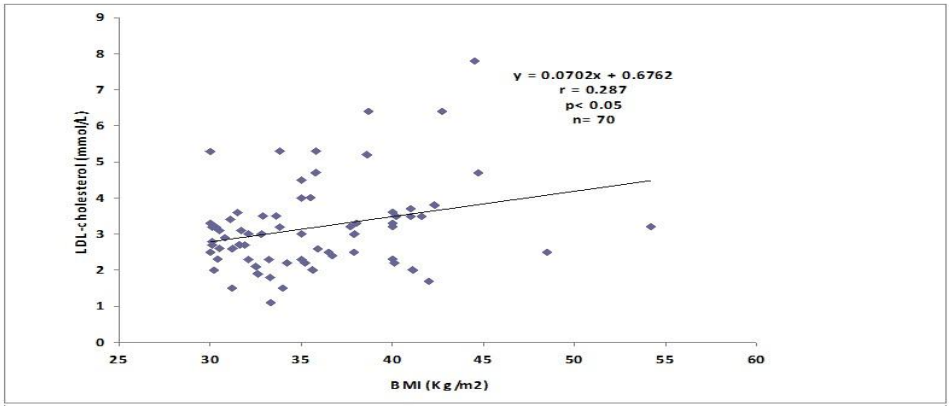


Fig 3: Correlation plot of glycatedhaemoglobin against body mass index in obese subjects

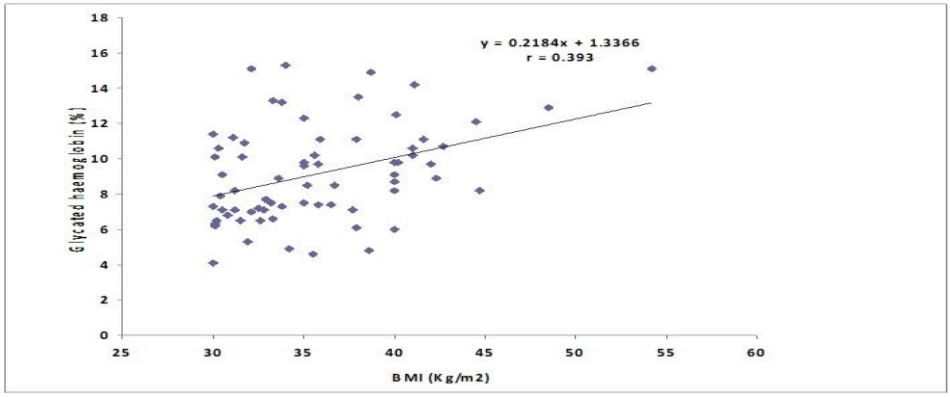


Fig 4: Correlation plot of HDL-cholesterol against body mass index in obese subjects

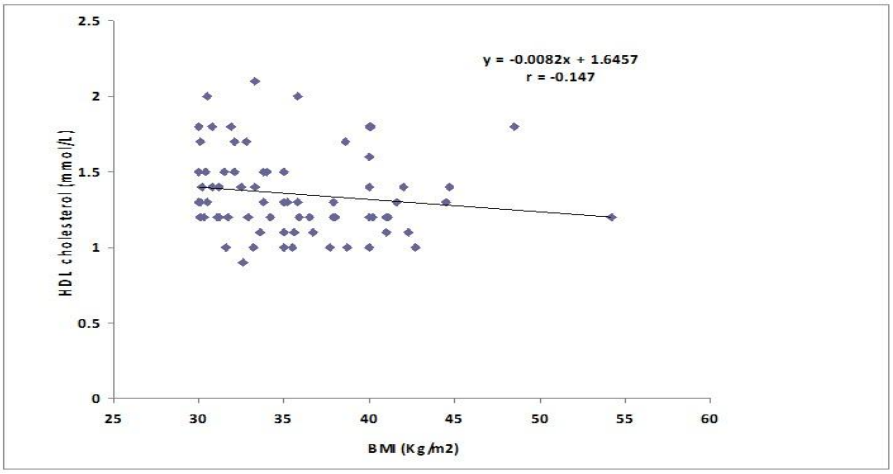
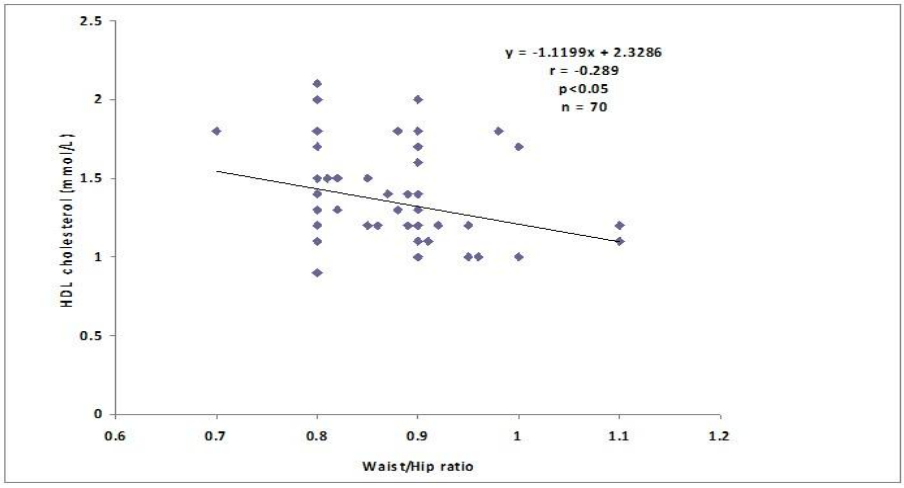


Fig 5: Correlation plot of HDL-cholesterol against waist-hip ratio in obese subjects



4. Discussion

The global incidence of obesity is on increase especially in adults. It was once considered a problem of developed countries; this epidemic now also affects developing countries. Obesity has been established to be risk factor for hypertension, type 2 diabetes and dyslipidemia. Multiple modifications of serum lipids and lipoproteins are frequently noted in overweight/obese individuals. The most common modifications are hypertriglyceridemia and decreased HDL-C levels[21]. More so, elevated glycated haemoglobin (≥ 6.5%) has been used to diagnose type-2 diabetes mellitus especially in obese individuals. This study was carried out to determine the fasting lipid profiles and glycated hemoglobin in obese and non-obese control subjects, in other to establish the effect of obesity on these biochemical parameters.

The findings of the study showed that BMI, HC, WC, WHR, HB1Ac, TC, LDL, TG, systolic and diastolic blood pressure were significantly higher in obese subjects than in the control group. This is in agreement with the findings of McGill *et al*[22] who in their study showed that BMI, WC, WHR, HB1Ac, TC, LDL and TG where higher in obese subjects when compared to the control subjects. McGill *et al*[22] also found that obese subject with elevated WC and WHR were prone to serious health conditions such as diabetes, prostate cancer and testicular cancer. Higher levels of TC, LDL and TG in these obese subjects are major risk factors for the development of coronary heart disease and atherosclerosis[23][24]. Higher levels of HB1Acin obese subjects is also a risk factor for the development of diabetes mellitus because glycated hemoglobin is used to monitor blood glucose control over a period of time (6-8weeks).

The findings of the study also revealed that HDL cholesterol was significantly higher in the control subjects than those of the obese subjects. This is also in agreement with the findings of Despres[25]and Kimberly *et al*[26] who showed that there is a strong negative correlation between obesity and HDL-C levels. HDL-C levels were significantly lower than those of the control subjects. Lower HDL

in these obese subjects can contribute to the cardiovascular risk factors facing obese subject because HDL is known as the "good cholesterol" helps mop up cholesterol. HDL removes extra cholesterol from the peripheral tissues and transports it to the liver for degradation and storage.

The findings of the study showed that class III obese subjects had significantly higher HB1Ac, TC, LDL and TG when compared to class 1 and class II subjects also class II had significantly higher values of TC and LDL when compared to class I. It was also observed that class III obese groups had significantly lower HDL cholesterol when compared to class I and class II obese classes. This is in consonance with the findings of Freedman *et al*[27]. This indicates that class III obese groups are at the highest risk of developing atherosclerosis and diabetes mellitus since this group showed the highest level of total cholesterol, LDL cholesterol, glycated hemoglobin and the lowest levels of HDL cholesterol. This therefore shows that increase in severity of obesity exposes the individual to more complications and health related problems of obesity especially atherosclerosis and diabetes mellitus.

Dyslipidemia characterized by elevated TG and low HDL-C has been associated with insulin resistance[28], even with low LDL-C, and may provide clinically relevant information related to the cardiovascular risk. There is literature associating poor HbA1c levels with atherogenic dyslipidemia, specifically with the TG/HDL-C ratio[29]. Other studies have found associations with cardiovascular disease in patients with hypercholesterolemia, suggesting that the control of HbA1c, independently of lipid management, is necessary in order to reduce the cardiovascular risk particularly in diabetic patients with elevated HbA1c[30].

The presence of the HbA1c in the model of diabetes assessment could help identify participants at high risk, with the predictability being improved by inclusion of lipid profile[31]. Therefore, evaluating the relationship between HbA1c and lipid profile might be expected to help in the identification of people at cardiovascular risk. These findings justify the need to encourage modalities to promote healthy body mass, frequent physical activities and dietary controls to prevent or minimize risks developing disorders associated with obesity.

Competing interest

Authors declare that there are no competing interests associated with this manuscript.

References

[1] World Health Organization. The World Obesity Reports, 2010.

[2] Sani MU, Wahab KW, Yusuf BO, Gbadamosi M, Johnson OV, Gbadamosi A. Modifiable cardiovascular risk factors among apparently healthy adult Nigerian population – a cross sectional study. *BMC Res Notes*, 2010; 3:11.

[3] Oladapo OO, Salako I, Sodiq O, Shoyinka K, Adedapo K, Falase AO. A prevalence of cardiometabolic risk factors among a rural Yoruba southwestern Nigerian population: a population-based survey. *Cardiovasc J Afr.* 2010; 21(1):26–31.

[4] Metcalf BS, Hosking J, Fremeaux AE, Jeffery AN, Voss LD, Wilkin TJ. BMI was right all along: taller children really are fatter (implications of making childhood BMI independent of height). *International Journal of Obesity*, 2011; 35(4):541-7.

[5] Lieb W, Sullivan LM, Harris TB. Plasma leptin levels and incidence of heart failure, cardiovascular disease, and total mortality in elderly individuals. *British Medical Journal* 2009; 32(4):612-6.

[6] Martinelli CE, Keogh JM, Greenfield JR. Obesity due to Melanocortin 4 Receptor (MC4R) Deficiency Is Associated with Increased Linear Growth and Final Height, Fasting Hyperinsulinemia, and Incompletely Suppressed Growth Hormone Secretion. *Journal of Clinical Endocrinology Metabolism*, 2011; 96 (1): E181-8.

[7] Ono T, Guthold R, Strong K. WHO Global Comparable Estimates: Global Infobase data for saving lives 2005; 2012. https://apps.who.int/infobase/Index.aspx.

[8] Abbasi A, Corpeleijn E, Postmus D, Gansevoort RT, De Jong PE, Gans RO. Plasma procalcitonin is associated with obesity, insulin resistance, and the metabolic syndrome. Journal of Clinical Endocrinology and Metabolism, 2010; 95 (9):26-31.

[9] Reinehr T, Kleber M., Sousa G. Leptin concentrations are a predictor of overweight reduction in a lifestyle intervention. *International Journal of Pediatric Obesity*, 2009; 1-9.

[10] Nedeltcheva AV, Kilkus JM, Imperial J, Schoeller DA, Penev PD. Insufficient sleep undermines dietary efforts to reduce adiposity. *Annual International Journal of Medicine* 2010; 153 (7):435-41.

[11] Murray PG, Banerjee I. Reduced appetite and body mass index with delayed puberty in a mother and son: association with a rare novel sequence variant in the leptin gene. *European Journal of Endocrinology*, 2011; 164(4):521-7.

[12] Brehm A, Pfeiler G, Pacini G, Vierhapper H, Michael Roden M. Relationship between serum lipoprotein ratios and insulin resistance in obesity. *Clin. Chem.* 2004; 50:122316–2322.

[13] Anderson AJ, Sobocinski KA, Freedman DS, Barboriak JJ, Rimm AA, Gruchow HW. Body fat distribution, plasma lipids and lipoproteins. *Arteriosclerosis*, 1998; 8:88-94.

[14] Burtis CA, Edward R, Ashwood David, Bruns E. Lipids, lipoproteins, Apolipoproteins and other cardiovascular risk factors. Tietz Fundamentals of Clinical Chemistry. 6th edition, 2008; 23:402-420.

[15] Wijga AH, Scholtens S, Bemelmans WJ. Comorbidities of obesity in school children: a cross-sectional study in the PIAMA birth cohort. *Medical Sport Science Journal* 2010; 10 (1):184.

[16] Bishop ML, Edward P, Tody L, Schoeff E. Carbohydrates, lipids and lipoproteins. Clinical chemistry, techniques, principles and correlations, 2010; 13:324-326, 14: 328-351.

[17] American Diabetes Association: Standards of medical care in diabetes-2009. *Diabetes Care* 2009; 32:S13-S61.

[18] Pani LN, Korenda L, Meigs JB, Driver C, Chamany S, Fox CS, Sullivan S, D'Agostino RB, Nathan DM: Effect of aging on A1C levels in individuals without diabetes. *Diabetes Care* 2008; 31:1991-1996.

[19] Gao L, Matthews FE, Sargeant LA, Brayne C, MRC CFAS: An investigation of the population impact of variation in HbA1c levels in older people in England and Wales: from a population based multi-centre longitudinal study. *BMC Publ Health*, 2008; 8:54.

[20] American Diabetes Association: Standards of medical care in diabetes-2011. *Diabetes Care* 2011; 34:S11-S61.

[21] Elsawi NM, Omar HM, Hassan AA, Ahmed AM. Study of C- peptide and glycoslated hemoglobin in obese and obese with type 2 diabetes and its relation with lipid profile from Sohag Governarate-Egypt. *Global Advanced Research Journal of Medicine and Medical Science* 2014; 3(12):430-436.

[22] MacGill, HCJ, McMahan CA, Herderich EE, Zieshe AW, Malcom GT, Tracy RE. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation*, 2002; 105(8): 2712 – 2718.

[23] Gaziano JM, Hennekens CH, O'Donnell CJ, Breslow JL, Burring JE. Fasting triglycerides, high-density lipoprotein, and risk of myocardial infarction. *Circulation*, 1997; 96:2520–2525.

[24] Onat A, Sari I, Yazici M, Can G, Hergenc G, Avci GS. Plasma triglycerides, an independent predictor of cardiovascular disease in men: a prospective study based on a population with prevalent metabolic syndrome. *Int. J. Cardiol*., 2006; 108:89–95.

[25] Despres JP. Obesity and lipid metabolism: relevance of body fat distribution. *Curr. Opin. Lipidol*., 1991; 2: 5-15.

[26] Kimberly AE, Timothy SM, Hao W, Nathalie P, Barbara AH, Mark TC, Phuong-Oanh TM, John FO, Chongren T, Renée CL. Obesity and weight loss result in increased adipose tissue ABCG1 expression in db/db mice. *Biochimicaet Biophysica Acta* 2012; 1821: 425–434.

[27] Freedman DS, Jacobsen SJ, Barboriak JJ, Sobocinki KA, Anderson AJ, Thissebah AH, Sasse EA, Gruchow HW. Body fat distribution and male/female differences in lipids and lipoproteins. *Circulation*, 2001; 81:1498-1506.

[28] Fruchart J-C, Sacks F, Hermans MP, Assmann G, Brown WV, Ceska R, Chapman MJ, Dodson PM, Fioretto P, Ginsberg HN, Kadowaki T, Lablanche J-M, Marx N, Plutzky J, Reiner Z, Rosenson RS, Staels B, Stock JK, Sy R, Wanner C, Zambon A, Zimmet P: The Residual Risk Reduction Initiative: a call to action to reduce residual vascular risk in patients with dyslipidemia. *Am J. Cardiol*., 2008; 102:S1-S34.

[29] Hermans MP, Ahn SA, Rousseau MF:log(TG)/HDL-C is related to both residual cardiometabolic risk and b-cell function loss in type 2 diabetes males. *Cardiovasc Diabetol*., 2010; 9:88.

[30] Nishimura R, Nakagami T, Sone H, Ohashi Y, Tajima N: Relationship between hemoglobin A1c and cardiovascular disease in mild-to moderate hypercholesterolemic Japanese individuals: subanalysis of a large-scale randomized controlled trial. *Cardiovasc Diabetol* 2011; 10:58.

[31] Chien KL, Lin HJ, Lee BC, Hsu HC, Chen MF. Prediction model for high glycated hemoglobin concentration among ethnic Chinese in Taiwan. *Cardiovasc Diabetol*., 2010; 9:59-67.