International Scholars Journals

International Journal of Medicine and Medical Sciences ISSN: 2167-0404 Vol. 6 (5), pp. 332-339, May, 2016. Available online at www.internationalscholarsjournals.org © International Scholars Journals



*Full Length Research Paper*

# Clinical diagnostic indices of obese patients

**[*1]Obimba, Kelechukwu Clarence, [2]Ozougwu, Jevas Chibuike, [3]Emejulu Ada A, and [4]Ihedimbu, Perpetua Chiamaka**

[*1,3,4]Department of Biochemistry, School of Biological Sciences, Federal University of Technology Owerri. Nigeria.
[2]Physiology and Biomedical Research Unit, Department of Biological Sciences, Rhema University, Aba, Abia State, Nigeria.

**Received 22 April, 2016; Accepted 13 May, 2016; Published 22 May, 2016**

**The aim of the study was to investigate the efficacy of the use of biochemical, hematological, blood pressure and anthropometric indices as diagnostic parameters of obesity. The experimental design was a single factor completely randomized design (CRD). Twenty (20) normal human subjects and thirty (30) obese patients were subjected to systolic (SP) and diastolic (DP) blood pressure, body mass index (BMI), serum glycated albumin (GA), fasting blood sugar (FBS), serum aspartate aminotransferase (AST), serum glycated hemoglobin (HbA1c), serum leptin (L), serum total cholesterol (C) and serum triglyceride (T) tests. Results recorded of the normal human subjects and obese patients, expressed as mean ± standard error (S.E) (unit) were as follows : SP (120.52 ± 9.14) and (146.87 ± 11.23) (mmHg), DP (75.6±1.7) and (90.11±0.9) (mmHg), BMI (15.3±0.7) and (30.5 ±1.7) (kg/m$^2$), GA (15.5±0.2) and (13.1±0.3) (%), FBS (68.8±4.7) and (125.8±1.7) (mg/dl), AST (9.2 ±1.9) and (22.5 ±1.0) (U/l), HbA1c (6.15 ±0.4) and (8.15 ±0.3) (%), L (10.5 ±1.0) and (31.5 ±1.0) (ng/ml.kg), C (170±1.5) and (195±2.0) (mg/dl), and T (175.51±2.3) and (110.2±0.215) (mg/dl), respectively. The mean values of SP, DP, and BMI, FBS, AST, HbA1c, L, C and T were significantly higher (p<0.05), but that of GA was significantly lower (p<0.01) in obese patients compared with normal human subjects. The statistical regression and correlation between L (ng/ml.kg) and BMI (kg/m$^2$) of obese patients were significant (p<0.05)(r = 0.963). Incidence of obesity correlated positively and significantly (p<0.05) with significant increase (p<0.05) in BMI, FBS, AST, HbA1c, L, C, T and significant decrease (p<0.01) in GA of the obese patients. These significant differences/alterations could be used as effective criteria/yardstick for the diagnosis, and management/treatment of obesity or monitoring of recovery from obesity.**

**Key words**: Leptin, glycated albumin, glycated hemoglobin.

## INTRODUCTION

Obesity is a disease condition in which excess body fat has accumulated to the extent that it impairs the normal functioning of the body, leading to reduced life expectancy and/or increased health problems [Haslam and James (2005), Rettner (2012)]. Obesity could be an issue of caloric intake in excess of body needs. A decreased capacity to generate heat by futile cycling and/or by the thermogenic action of brown fat could result in obesity (Lehninger, 1982).

The World Health Organization (WHO) formally recognized obesity as a global epidemic in 1997 (Caballero, 2007). The WHO estimates that at least 500 million adults are obese with higher rates among women than men. The rate of obesity also increases with age (age range 50 - 60 years old are very vulnerable) (Kopelman *et al*., 2009).

The tabular schematic of the description of obesity with respect to body mass index (BMI) values is shown in Table 1.

Much of the pathophysiology of obesity is associated with influence of hormonal mechanisms on the regulation of appetite and food intake, storage patterns of adipose tissue, and development of insulin resistance, the chief of which are leptin and ghrelin

Corresponding author. E mail: kechrisob@yahoo.com. Tel. 07034851899.

**Table 1.** Classification of obesity in relation to the body mass index (BMI).

| BMI ($kg/m^2$) from | up to | Classification |
|---|---|---|
| | 18.5 | underweight |
| 18.5 | 25.0 | normal weight |
| 25.0 | 30.0 | overweight |
| 30.0 | 35.0 | class I obesity |
| 35.0 | 40.0 | class II obesity |
| 40.0 | | class III obesity |

WHO (1995), WHO (2000), WHO (2004)
Body mass index is a measure of the weight of an individual (kg) per the square of the individual's height ($meters^2$).

hormonal mechanisms. Ghrelin produced by the stomach modulates short-term appetitive. Leptin is produced by adipose tissue to signal fat storage reserves in the body, and mediates long-term appetitive controls (Hamann and Matthaei, 1996). Leptin and ghrelin control appetite through their actions on the hypothalamus of the central nervous system. The hypothalamus is a region of the brain central to the regulation of food intake and energy expenditure. Leptin inhibits the neuropeptide Y (NPY)/ agouti-related peptide (AgRP) arcuate neuron which stimulate feeding and inhibit satiety, but stimulates the pro-opiomelanocortin (POMC)/cocaine- and amphetamine-regulated transcript (CART) which stimulate satiety and inhibit feeding. A deficiency in leptin signaling, either via leptin deficiency or leptin resistance, leads to overfeeding and may account for some genetic and non-genetic forms of obesity (Flier, 2004).

Obese individuals are susceptible to various diseases, particularly heart disease, type 2 diabetes, obstructive sleep apnea, certain types of cancer, and osteoarthritis, and hypertension (Haslam and James, 2005). Obesity is a major feature in several syndromes such as Prader–Willi syndrome, Bardet–Biedl syndrome, Cohen syndrome, and MOMO syndrome, among others, are syndromes of which obesity is a major feature. Obesity that is not associated with syndromes is termed "non-syndromic obesity" (Walley *et al*., 2009).

Ten (10) microorganisms (including canine distemper virus, avian adenovirus [SMAM-1] and human adenoviruses) have been linked with obesity in either humans or animals (Dhurandhar, 2011). Adenovirus 36 (Ad36) is associated with adiposity and inflammation. A meta-analysis of 10 studies revealed that Ad36 infection was associated with an increased risk for obesity (OR 1.9 [95% CI 1.01 to 3.56]; P=0.047) and weight gain (increase in body mass index of 3.19 $kg/m^2$) (Yamada *et al*., 2012). There is a positive, significant correlation between viral infection and obesity in humans and several different animal species (Falagas and Kompoti, 2006).

Obesity is associated with age-related cataract, glaucoma, age-related maculopathy, elevated intraocular pressure and diabetic retinopathy (Cheung and Wong, 2007). Obesity has also been linked to cataract by its associated complications such as diabetes, glucose intolerance, insulin resistance, hyperlipidemia and hypertension (Grundy, 2000). Oxidative stress associated with obesity may play an important pathogenic role in cataract formation.

Serum glycated albumin (GA) was inversely influenced by fat mass and visceral adipose tissue in Chinese with normal glucose tolerance (Feifei *et al*., 2012). Reduced levels of high-density lipoproteins (HDL) in non-obese and obese states are associated with increased risk for the development of coronary artery disease (Rashid and Genest, 2007). BMI and serum total cholesterol concentration were significantly higher ($p<0.05$) in obese subjects compared with normal (control) human subjects in Karachi Pakistan (Mushtaq *et al*., 2014). No significant difference in mean values of aspartate aminotransferase ($p<0.05$) was observed among normal, overweight and obese individuals in East Medinipur, India. (Das *et al*., 2015).

The levels of glycated hemoglobin were higher in obese diabetic patients (Types I and II) than in non-obese diabetic patients in Cumballa Hill Hospital and Purohit Hospital, Mumbai (Kale and Rawat, 2006). Glycated hemoglobin may be used as a reliable index for screening and assessing blood glycemic control in obese subjects who are at risk of developing type 2 diabetes mellitus (Emeribe *et al*., 2015).

Activation of the sympathetic nervous system, activation of the renin-angiotensin system, and sodium retention, characterize obesity-associated arterial hypertension (Re, 2009). Obesity is associated with increased blood flow, vasodilatation, cardiac output, and hypertension. Leptin, a hormone produced in fat that produces satiety and weight loss by diminishing caloric intake and by activating the sympathetic nervous

system to enhance thermogenesis, can cause hypertension [Hall (2000), Frohlich (2002)].

Obesity is associated with elevated systolic blood pressure (SBP) and diastolic blood pressure (DBP), dyslipidemia, and diabetes. BMI and fat percentage correlated significantly and positively with blood pressure (both systolic and diastolic blood pressure), and odds ratio showed that overweight/obese subjects were more likely to develop hypertension than normal BMI subjects in Punjabi community, Delhi, India (Dua *et al*., 2014).

Mean values of serum leptin was significantly higher ($p<0.05$) in Omani obese patients compared with the control non-obese subjects with recorded significant, positive multiple correlation of serum leptin levels with weight ($p=0.002$), body fat percentage ($p=0.0001$) and BMI ($p=0.001$) of the obese individuals (Al Maskar and Alnaqdy, 2006).

Moderately intense physical activities including fast walking and swimming for 150 to 300 minutes every week prevent weight gain, effectively. Other prophylactic measures of obesity include: making a staple of diet of low-calorie, nutrient-dense foods, such as fruits, vegetables and whole grains. Avoiding saturated fat containing foods, sweets and alcohol. Controlling ones eating behaviors and habits. Monitoring body weight regularly.

The main treatment for obesity consists of dieting and physical exercise (Lau *et al*., 2007). Orlistat (Alli; Xenixal) reduces intestinal fat absorption by inhibiting pancreatic lipase and is approved for long term use for weight loss (Rucker *et al*., 2007). Lorcaserin effects a weight loss of 5.8 kg in one year (Astrup, 2010). An effective combination therapy of obesity is a reduced calorie diet, walk briskly for 30 minutes for five days of a week and phentermine/topiramate (Qsymia) drug (FDA, 2012).

The most effective treatment for obesity is bariatric surgery, and is associated with long-term weight loss, improvement in obesity related conditions and decreased overall mortality [Chang *et al*. (2014), Colquitt *et al*. (2014)]. Use of devices that occupy space in the stomach are also effective in the treatment of obesity (Weintraub, 2014).

One-pound (lb) weight of body mass is acquired by an intake of 3500Kcal in excess of the recommended daily (dietary) allowance of 2700kcal for adult males (Lehninger, 1982). 1lb = 0.45kg. A human, adult male may acquire 25lb body mass by intake of excess calories of 25 X 3500Kcal = 87,500Kcal. If the adult male consumes excess 150Kcal of the 87,500Kcal, per day, then the accumulation of the body mass is over $\frac{87500}{150}$ days ≈ 583 days. In other words, an adult male who consumes 150Kcal in excess of the recommended 2700Kcal (ie a total of 2850Kcal) for 583 days will acquire 25lb body mass over the period. 1kcal = 4.18kJ.

Dietary therapy that would ensure that an adult male would loose 25lb (87,500Kcal) over a period of 140 days (20weeks), would require the shedding of $\frac{87500}{140}$ kcal = 625kcal per day. In other words, the adult male would consume only 2700kcal (RDA of dietary calories for adult males) - 625kcal = 2075 kcal per day.

Dietary therapy that would ensure that the adult male would loose 25lb (87,500Kcal) over a period of 364 days (approximately one year), would require the shedding of $\frac{87500}{364}$ kcal = 240.38kcal per day. In other words, the adult male would consume only 2700kcal (RDA of dietary calories for adult males) - 240.38kcal = 2459.62 kcal per day.

Proximate analysis of a diet/meal: a balanced semi-vegan diet, high in dietary soluble fiber, low in saturated fat and sugar, but moderate in essential fatty acids (EFAs), prepared primarily from processed catfish (weight ratio: 1), processed soya bean (*Glycine max*) seeds (weight ratio: 1.8), processed groundnut (*Arachis hypogaea*) seeds (weight ratio: 1.2), fluted pumpkin leaves, low-sugar banana (*Musa acuminata*) fruit, palm oil and vitamin-dietary mineral premix, revealed that it contains: 28% crude protein, 20% carbohydrates and 15% lipids (fatty acids). The approximate energy yield per gram of nutrient fraction is : carbohydrate - 4.2 kcalories, fats - 9.5kcalories, and protein - 4.3 kcalories (Lehninger, 1982).

One hundred (100) grams of the diet/meal would contain: (28 X 4.3) + (20 X 4.2) + (15 X 9.5) kcals = 346.9kcals.

2075kcals is contained in $\frac{100 \text{ x } 2075}{346.9}$ ≈ 598.2 grams of the diet/meal.

An adult male would loose 25lb (87,500Kcal) over a period of 140 days (20weeks) by consuming only 598.2 grams (2075kcal) of the diet/meal per day.

The aim of the research was to investigate the efficacy of the use of biochemical indices : fasting blood sugar (FBS) , serum glycated albumin (GA), serum aspartate aminotransferase (AST), serum triglyceride (TG), serum total cholesterol (C), serum leptin, and hematological index: glycated hemoglobin (HbAc1), anthropometric body mass index (BMI), and systolic and diastolic blood pressure as clinical diagnostic indices of obesity and for the monitoring of recovery (rehabilitation)/treatment of obesity.

## MATERIALS AND METHODS

### EXPERIMENTAL DESIGN

The experimental design is a single-factor completely randomized design (CRD).

SPSS for windows (version 17.0, SPSS, Chicago, IL, USA) was used to perform the statistical analyses. The significance levels were $p<0.05$, $p<0.01$.

#### Selection of human subjects:

Thirty (n = 30) clinically confirmed obese patients, of age bracket 35-50 years and twenty normal or healthy subjects (n=20) of the same age bracket, voluntarily participated in this study from Owerri municipal. Inclusion criteria for the obese patients was BMI≥30kg/m$^2$. Exclusion criteria included: cancer, persons living with HIV, malaria patients, sickle cell anemia patients.

The research was carried out in compliance with the Declaration on the Right of the Patient (WMA, 2000). Blood was obtained by veni-puncture carried out by a Phlebotomist nurse.

The method described by Thavasu *et al*. (1992) was used in obtaining the serum. Whole blood was collected in a covered test tube, and allowed to clot by leaving it undisturbed for 15-30 minutes at room temperature. The clot was removed by centrifuging at 1,000-2,000 x g for 10 minutes in a refrigerated centrifuge, to obtain the blood serum. Citrate phosphate dextrose-adenine 1 (CPDA-1) stored whole blood was used for whole blood analysis.

**Determination of blood pressure**

An electronic sphygmomanometer (DM 3000, Kawamoto Corporation, Osaka Japan, digital blood pressure monitor), was used in measuring systolic and diastolic blood pressures of the hypertensive patients and normal human subjects.

**Measurement of body mass index (BMI)**

Weight and height of human subjects were measured using the weight watchers ultimate precision electronic scale and meter rule, respectively. The BMI was calculated as :

$$\frac{\text{weight}}{\text{height}^2} = \frac{\text{kg}}{\text{m}^2}$$

***In vitro* quantitative determination of serum fasting blood sugar (FBS)**

Serum was obtained from patient/individual who had not taken any victual (food or drink, except water), for an 8-hour period. Glucose oxidase catalyses the oxidative transformation of β D- glucose present in the serum to D glucono -1 ,5 - lactone with the formation of hydrogen peroxide. The lactone is slowly hydrolysed to D-gluconic acid. The hydrogen peroxide produced is broken down to oxygen and water by a peroxidase enzyme. Oxygen reacts with ortho-toluidine to produce a coloured complex, the intensity of which is proportional to the concentration of the D-glucose in the serum, and measurable at 540nm in a spectrophotometer (Thermo scientific model G10S UV-Vis).

***In vitro* quantitative analysis of aspartate amino transferase (AST)**

Quantitative *in vitro* determination of serum aspartate amino transferase (AST) was carried out using the method employed by Reitman and Frankel (1957). The test based on the reaction in which l-aspartate and α-ketoglutarate are converted to l-glutamate and oxaloacetate by the catalytic activity of AST. The oxaloacetate so formed, forms a complex known as oxaloacetate hydrazone with 2,4-dinitrophenyl hydrazine. The intensity of the colour of the hydrazone, which is measurable with a spectrophotometer (Thermo scientific model G10S UV-Vis) at 578nm is directly proportional to the AST enzyme activity.

***In vitro* quantitative determination of serum glycated albumin**

Serum glycated albumin (GA) was measured according to methods described by Inaba *et al*. (2007). The in vitro quantitative determination of the serum glycated albumin was carried out by an enzymatic method using the Lucica GA-L kit (Asahi Kasei Pharma Corp., Tokyo, Japan) (Kouzuma, 2004). GA was hydrolyzed to amino acids by albumin-specific proteinase and then oxidized by ketoamine oxidase to produce hydrogen peroxide, which was measured quantitatively. The GA value was calculated as the percentage of GA relative to total albumin, which was measured with new bromocresol purple method using the same serum sample (Kouzuma, 2004). GA assay was not influenced by the physiologic concentrations of ascorbic acid, bilirubin, and up to 1000 mg/dl glucose (Nagamine *et al*., 2004).

***In vitro* quantitative determination of serum glycated hemoglobin**

Serum glycosylated hemoglobin (HbA1c) was measured using high-performance liquid chromatography, according to the method employed by Shenqi *et al*. (2013).

***In vitro* quantitative determination of serum leptin**

The method employed by Chow and Phoon (2003) was used for the quantitative measurement of leptin in serum. This was performed using a leptin enzyme immunoassay or ELISA kit (DRG Diagnostics, Marburg, Germany). Inter-assay and intra-assay reproducibility was analyzed by the manufacturer by determining the coefficients of variation, which ranged between 3.6 and 7.8 and between 4.1 and 5.4%, respectively.

**Lipid Profile Assays**

Serum total cholesterol (C), and serum triacylglycerol (TG) were determined using commercial kits (Randox Laboratory Ltd., UK), in conformity with the methods employed by Ibegbulem and Chikezie (2012); Chikezie and Okpara (2013).

**RESULTS**

Table 2 shows the results on blood pressure and body mass index (BMI) of the obese patients, and healthy human subjects. Mean values of systolic and diastolic blood pressure, and BMI observed of obese patients were significantly higher ($p<0.05$) in comparison with normal healthy patients.

Table 3 shows the results on the biochemical indices viz: serum glycated albumin (GA), fasting blood sugar (FBS), serum aspartate aminotransferase (AST),serum glycated hemoglobin (HbA1c), and serum leptin (L) of the obese patients, and normal, healthy human subjects. Significantly higher mean values ($p<0.05$) were recorded of these biochemical indices (except serum glycated albumin (GA)) of the obese patients in comparison with normal, healthy human subjects. The

**Table 2.** Results on blood pressure and body mass index (BMI) of the obese patients, and normal, healthy human subjects.

| | Systolic blood pressure (mmHg) | Diastolic blood pressure (mmHg) | Body mass index (BMI) $Kg/m^2$ |
|---|---|---|---|
| Healthy human subjects | 120.52 ± 9.14 [a] | 75.6±1.7[a] | 15.3±0.7[a] |
| Obese patients | 146.87 ± 11.23 [b] | 90.11±0.9[b] | 30.5 ±1.7[b] |

Results are expressed as mean ± standard error (S.E) (unit) ($n_1$ =sample size of Healthy human subjects 20, $n_2$ sample size of Obese patients = 30).
Values that are labeled, in the same column, with the same superscripts, are not significantly different ($p<0.05$).

**Table 3.** Results on the biochemical indices: Serum glycated albumin (GA), fasting blood sugar (FBS) and serum aspartate aminotransferase (AST), serum glycated hemoglobin (HbA1c), serum leptin (L) of the obese patients, and normal, healthy human subjects.

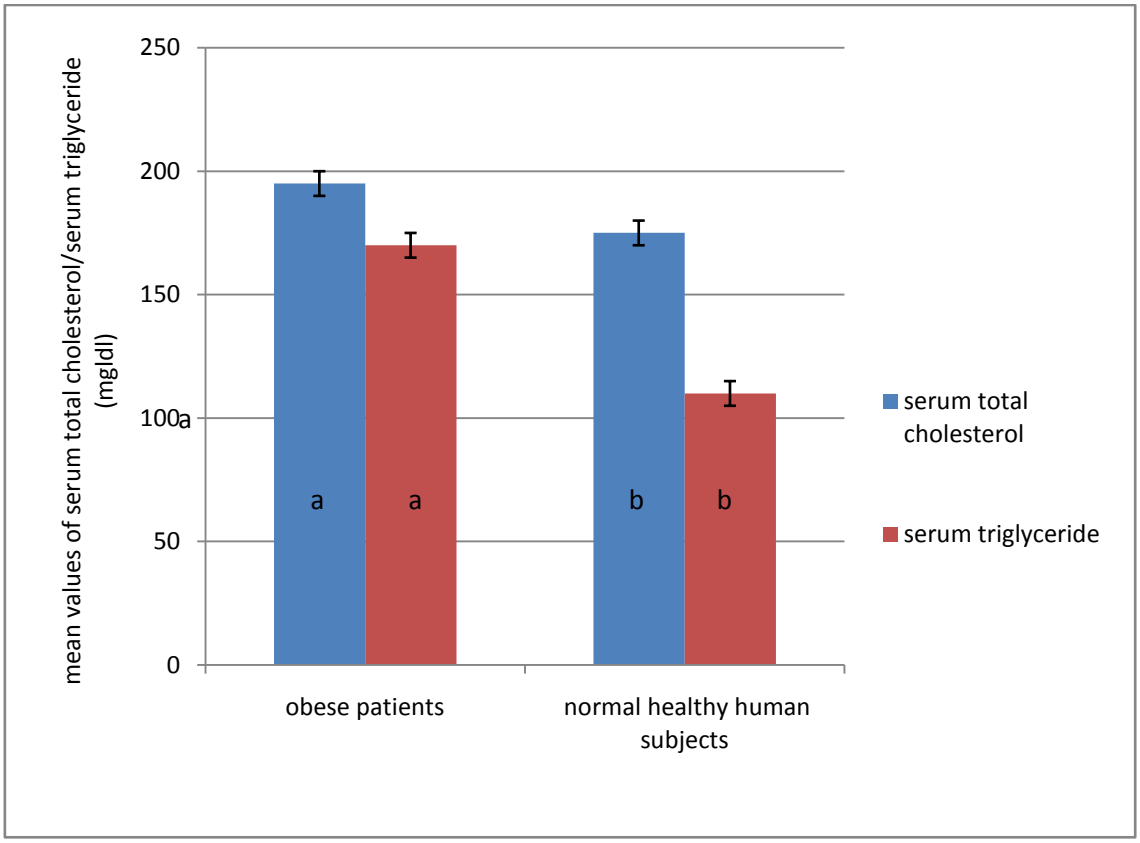
| | *Serum glycated albumin (%) | Fasting blood sugar (mg/dl) | Serum aspartate aminotransferase (U/l) | Serum glycated hemoglobin (%) | Serum leptin (L) ng/ml.kg |
|---|---|---|---|---|---|
| Healthy human subjects | 15.5±0.2[a] | 68.8±4.7[a] | 9.2 ±1.9[a] | 6.15 ±0.4[a] | 10.5 ±1.0[a] |
| Obese patients | 13.1±0.3[b] | 125.8±1.7[b] | 22.5 ±1.0[b] | 8.15 ±0.3[b] | 31.5 ±1.0[b] |

Results are expressed as mean ± standard error (S.E) (unit) ($n_1$ = 20, $n_2$ = 30).
Values that are labeled, in the same column, with the same superscripts, are not significantly different ($p<0.05$), *($p<0.01$).

mean value of serum glycated albumin of normal healthy subjects was significantly higher ($p<0.01$) than the corresponding value of the obese patients.
Figure 1 shows the results (graphical) on the biochemical indices serum total cholesterol and serum triglyceride. Significantly higher mean values ($p<0.05$) were recorded of these biochemical indices of the obese patients in comparison with normal, healthy human subjects.

## DISCUSSION

Hypertension results in cardiovascular damage such as left ventricular hypertrophy, arterial and ventricular arrhythmias, diastolic heart failure, systolic heart failure, and ischemic heart disease that could be associated with congestive heart failure (Sharma and Kortas, 2006). Mean values of systolic and diastolic blood pressure shown in Table 2, observed of obese patients, were significantly higher ($p<0.05$) in comparison with normal healthy patients, and reflect stage 1 hypertension in the obese patients, consistent with the postulates of Stewart *et al*. (2013), who posited that twelve percent of a population of Nepalese with high BMI [z (BMI-for-age score)= 0.42, BMI≥30kg/$M^2$] had high blood pressure (>120/80mmHg).
Obesity was assessed as the obesity index of percentage of fat and was found to be significantly related to the fasting plasma levels of glucose ($p < 0.001$) (Elahi *et al*., 1982). Non-esterified fatty acids (NEFA) also known as free fatty acids (FFA) are elevated in obesity arising from an increased adipose tissue mass (Karpe *et al*., 2011). The result is an increase in both skeletal muscle and liver insulin resistance leading to elevated fasting blood glucose levels, and subsequently, increase in glycated hemoglobin. Intentional weight loss of 10% can potentially decrease A1C% by 0.81 among patients with type 2 DM (Shantha *et al*., 2012): an indication that weight gain may lead to elevated levels of glycated hemoglobin.
The observation by Ji *et al*. (2014) that serum glycated albumin to glycated hemoglobin (GA/A1c) ratio is known to be inversely related with body mass index (BMI), lends credence to the findings in the present study which is that the mean value of serum glycated albumin of normal healthy subjects was significantly higher ($p<0.01$) than the corresponding value of the obese patients (Table 3).
Consistent with the results in Table 3 of observed significant increase ($p<0.05$) in fasting blood sugar and serum glycated hemoglobin of obesed patients compared with normal healthy subjects, is the finding by Feifei *et al*. (2012) that significantly higher ($p<0.05$) fasting plasma glucose, and glycated hemoglobin (A1c) were reported of Chinese subject with BMI ≥25.0 $kg/m^2$. High blood glucose and high BMI are independently associated with increased risk of breast cancer death in Italian women patients with oestrogen and progesterone hormone receptor-positive disease (Minicozzi *et al*., 2013).
The mean value of aspartate aminotransferase (AST) of obese patients was significantly higher ($p<0.05$) than the corresponding value of the normal human subjects (Table 3), in keeping with the postulates that aspartate aminotransferase (AST) level was significantly increased in obese males in comparison with non-obese males of a Pakistani population (Qureshi *et al*., 2006). Liver is markedly affected by obesity and is often associated with hepatomegaly,




Graphical results are expressed as mean ± standard error (mg/dl) ($n_1$ = 20, $n_2$ = 30).. Error bars represent values of standard error (1.5 – 2.3 mg/dl). Corresponding bars labeled with the same letters represent mean values of serum total cholesterol or serum triglyceride which are not significantly different ($p<0.05$).

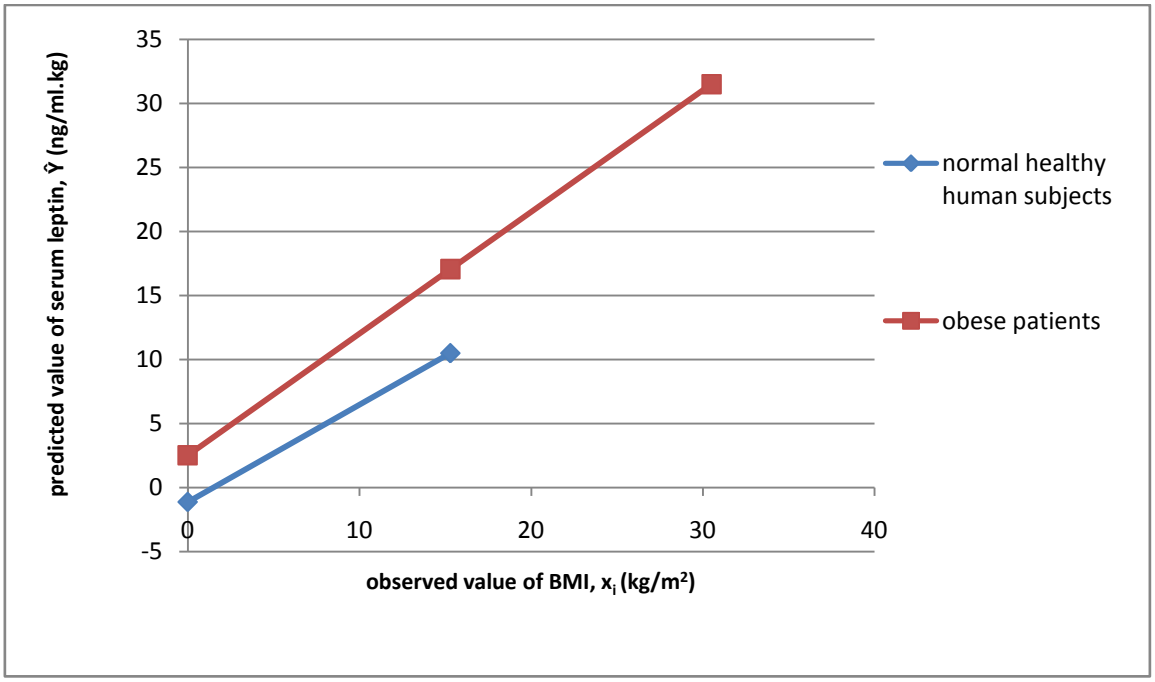
**Figure 1.** Results (graphical) on the biochemical indices : serum total cholesterol and serum triglyceride of the obese patients, and normal, healthy human subjects.

alterations in liver histology described by macrovesicular steatosis, steatohepatitis, fibrosis and cirrhosis (Mokdad *et al*., 2001).

Serum leptin levels shown in Table 3 were significantly higher ($p<0.05$) in obese patients compared with normal human subjects, a finding corroborated by the observation that serum leptin levels are elevated in obesity due to increase amount of adipose tissue which is the main source of the hormone and possibly secondary to some degree of central resistance to its action. Serum leptin is positively correlated with systolic and diastolic blood pressure in both obese and non-obese individuals [Kunz *et al*. (2000), Schutte *et al*. (2005)]. Obese hypertensives have been shown to have higher leptin levels compared with obese normotensive individuals (Bravo *et al*., 2006).

Significantly higher mean values ($p<0.05$) were recorded of the serum total cholesterol and serum triglyceride of the obese patients in comparison with normal, healthy human subjects (figure 1), in conformity with the findings of Mushtaq *et al*. (2014). Serum concentration of triglycerides in obese subjects is increased by dual metabolic defects viz: increased secretion (due to increased liver and subcutaneous abdominal fat) and severely impaired clearance of triglyceride-rich very low-density lipoprotein particles (associated with increased plasma levels of apolipoprotein) (Taskinen *et al*., 2011). Obesity enhances cholesterol synthesis very potently and reduction of weight effectively normalizes cholesterol production (Miettinen, 1971).

Multiple regression studies revealed that BMI (a function of obesity) regressed significantly ($p<0.05$) with systolic and diastolic blood pressure (a function of hypertension), serum total cholesterol (a function of cardiovascular diseases) and fasting blood sugar (a function of diabetes). Obesity increases the risk of cardiovascular diseases, hypertension and diabetes (Lehninger, 1982). Incidence of obesity correlated positively and significantly ($p<0.05$) with significant increase ($p<0.05$) in BMI, fasting blood sugar (FBS), serum aspartate aminotransferase (AST), serum glycated hemoglobin (HbA1c), serum leptin (L), serum total cholesterol, serum triglyceride and significant decrease ($p<0.01$) in serum glycated albumin of the obese patients. Observed values of BMI could be used with high precision to predict serum leptin levels of obese patients from the regression curve [$\hat{Y}$ (predicted value of serum leptin) = $\hat{Y}$ (ng/ml.kg) = 2.525 + 0.95xi ($kg/m^2$)], xi is observed value of BMI ($kg/m^2$) (figure 2).




(obese patients): Ŷ (ng/ml.kg) = 2.525 + 0.95xi (kg/m$^2$). (normal healthy human subjects): Ŷ (ng/ml.kg) = -1.13 + 0.76xi (kg/m$^2$).

**Figure 2.** Regression curve of serum leptin (ng/ml.kg) vs body mass index (BMI) (kg/m$^2$).

The correlation statistical analysis of BMI (an anthropometric index) and serum leptin (a hormonal biochemical index) of the obese patients was significant ($p<0.05$), and positive with a Pearson's product moment correlation coefficient of 0.963.

## CONCLUSION

Incidence of obesity correlated positively and significantly ($p<0.05$) with significant increase ($p<0.05$) in BMI, fasting blood sugar, serum aspartate aminotransferase (AST), serum glycated hemoglobin (HbA1c), serum leptin (L), serum total cholesterol, serum triglyceride and significant decrease ($p<0.01$) in serum glycated albumin of the obese patients. There was a significant ($p<0.05$), positive, association between serum leptin and BMI of obese patients. Observed values of BMI could be used with high precision to predict serum leptin levels of obese patients.

## ACKNOWLEDGEMENT

The authors acknowledge the technical contributions of the Department of Biochemistry, Federal University of Technology Owerri, and also wish to express our profound gratitude to the human subjects who participated in this research.

## REFERENCES

Al Maskar MY, Alnaqdy AA (2006). Correlation between Serum Leptin Levels, Body Mass Index and Obesity in Omanis. Sultan Qaboos Univ Med J. **6**(2): 27–31.

Astrup A. (2010). "Drug management of obesity--efficacy versus safety". N Engl J Med **363** (3): 288–90.

Bravo PE , Morse S, Borne DM, Aguilar EA, Reisin E (2006). Leptin and Hypertension in Obesity.Vasc Health Risk Manag. **2**(2): 163–169.

Caballero B (2007). "The global epidemic of obesity: An overview". Epidemiol Rev **29**: 1–5.

Chang SH, Stoll CR, Song J, Varela JE, Eagon CJ, Colditz GA (2014). "The effectiveness and risks of bariatric surgery: an updated systematic review and meta-analysis, 2003-2012". JAMA Surgery (Meta-analysis, Review) **149** (3): 275–87.

Cheung N, Wong TY (2007). Obesity and eye diseases. Surv Ophthalmol. 52(2):180-95.

Chikezie PC, Okpara RT (2013). Serum Lipid Profile and Hepatic Dysfunction in Moderate Plasmodium Falciparum Infection. Global J. of Med. Res. Dis. **13** (4): 14-20.

Chow VTK, Phoon MC (2003). Measurement of serum leptin concentrations in University Undergraduates by Competitive Elisa reveals correlations with body mass index and sex. Advances in Physiology Education. **27** (2): 70-77.

Colquitt JL, Pickett K, Loveman E, Frampton GK (2014). "Surgery for weight loss in adults". The Cochrane database of systematic reviews (Meta-analysis, Review) **8**: CD003641.

Das AK, Chandra P, Gupta A, Ahmad N (2015). Obesity and the levels of liver enzymes (ALT, AST & GGT) in East Medinipur, India, Asian J. Med. Sci. 6 (1):

40-42.
Dhurandhar NV (2011). A framework for identification of infections that contribute to human obesity. Lancet Infect Dis.**11**:963–9.
Dua S, Bhuker M, Sharma P, Dhall M, Kapoor S (2014). Body Mass Index Relates to Blood Pressure Among Adults. N Am J Med Sci. **6**(2): 89–95.
Elahi D, Muller DC, Tzankoff SP, Andres R, Tobin JD (1982). Effect of age and obesity on fasting levels of glucose, insulin, glucagon, and growth hormone in man. J Gerontol. **37**(4):385-91.
Emeribe AU, Elochukwu AC, Nasir IA, Bassey IE, Udoh EA (2015). Clinical significance of glycated hemoglobin testing in obese subjects attending a tertiary hospital at Calabar, Nigeria . Sub-Saharan Afr J Med. **2**:134-41.
Falagas ME, Kompoti M (2006). "Obesity and infection". Lancet Infect Dis (Review) **6** (7): 438–46.
FDA (Food and drug administration) (2012). "FDA approves weight-management drug Qsymia". Food and Drug Administration.
Feifei W, Xiaojing M, Yaping H, Rong Y, Jie N, Yunfeng X, Junling T, Yuqian B, Weiping J (2012). Serum Glycated Albumin Is Inversely Influenced by Fat Mass and Visceral Adipose Tissue in Chinese with Normal Glucose Tolerance. PLoS ONE **7**(11): e51098.
Flier JS (2004). "Obesity wars: Molecular progress confronts an expanding epidemic". Cell (Review) **116** (2): 337–50.
Frohlich ED (2002). Clinical management of the obese hypertensive patient. Cardiol Rev. **10**(3):127–138.
Grundy SM (2000). Metabolic complications of obesity. Endocrine.**13**:155–65.
Hall JE (2000). Pathophysiology of obesity hypertension. Curr Hypertens Rep. **2**(2):139–147.
Hamann A, Matthaei S (1996). "Regulation of energy balance by leptin". Exp. Clin. Endocrinol. Diabetes (Review) **104** (4): 293–300.
Haslam DW, James WP (2005). "Obesity". Lancet (Review) **366** (9492): 1197–209.
Ibegbulem CO, Chikezie PC (2012). Serum lipid profile of rats (Rattus norvegicus) fed with palm oil and palm kernel oil-containing diets. Asian J. Biochem. **7**(1): 46-53.
Inaba M, Okuno S, Kumeda Y, Yamada S, Imanishi Y, Tabata T, Mikio Okamura, Okada S Yamakawa T, Ishimura E, Nishizawa Y and the Osaka CKD Expert Research Group (2007). Glycated Albumin Is a Better Glycemic Indicator than Glycated Hemoglobin Values in Hemodialysis Patients with Diabetes: Effect of Anemia and Erythropoietin Injection. J. Am. Soc. Nephrol. **18**: 896–903.
Ji HH, Kwang JK, Byung-Wan L, Dong WK, Eun SK, Bong SC,Hyun CL (2014). The Relationship between BMI and Glycated Albumin to Glycated Hemoglobin (GA/A1c) Ratio According to Glucose Tolerance Status. PLoS One. **9**(2): e89478.
Kale K, Rawat DK (2006). Effect of obesity and insulin resistance on diabetic control. Indian J Clin Biochem. **21** (1) :83-88.
Karpe F, Dickmann JR, Frayn KN (2011).Fatty Acids, Obesity, and Insulin Resistance: Time for a Re-evaluation. Diabetes **60** (10). 2441-2449.
Kopelman PG, Caterson ID, Stock MJ, Dietz WH (2009). Epidemiology: Definition and Classification of Obesity. In: Clinical obesity in adults and children. 3rd ed. Wiley-Blackwell Publishing. UK. 3-14.
Kouzuma T (2004). Study of glycated amino acid elimination for an improved enzymatic glycated albumin measurement method. Clin. Chim. Acta. **346** : 135 –143.
Kunz I, Schorr U, Klaus S, Sharma AM (2000). Resting metabolic rate and substrate use in obesity hypertension. Hypertension.**36**:26–32.
Lau DC, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E, Douketis, Morrison, Hramiak, Sharma, Ur, Obesity Canada Clinical Practice Guidelines Expert Panel (2007). "2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]". CMAJ **176** (8): S1–13.
Lehninger AL (1982). Human Nutrition. In: Principles of Biochemistry. Fox J (Ed.). CBS Publishers. India. :753-789.
Miettinen TA (1971). Cholesterol production in obesity. Circulation. **44**:842-851.
Minicozzi P, Berrino F, Sebastiani F, Falcini F, Vattiato R, Cioccoloni F, Calagreti G, Fusco M, Vitale MF, Tumino R, Sigona A, Budroni M, Cesaraccio R, Candela G, Scuderi T, Zarcone M, Campisi I, Sant M (2013). High fasting blood glucose and obesity significantly and independently increase risk of breast cancer death in hormone receptor-positive disease. Eur J Cancer. **49**(18):3881-8.
Mokdad AH, Ford E, Bowman BA (2001). Prevalence of obesity, diabetes, and obesity related health risk factors. J. Am. Med. Assoc. **289**: 76-79.
Mushtaq R, Mushtaq R, Akram A, Khwaja S, Ahmed S (2014). Study of Serum Cholesterol Level in Adult Obese Population of Karachi, Pakistan. Open Journal of Endocrine and Metabolic Diseases. **4**: 238-243.
Nagamine Y, Mitsui K, Nakao T, Matsumoto M, Fujita C, Doi T (2004). Evaluation of the enzymatic method for glycated albumin with liquid type reagent (Lucia GA-L) [in Japanese]. Jpn. J. Med. Pharm. Sci., **51**: 737 –745,
Qureshi IZ, Shabana A, Fareeha A (2006). Effect of Overweight and Obesity on Liver Function in a Sample From Pakistani Population. Pakistan J. Zool. **38**(1) : 49-54.
Rashid S, Genest J (2007). Effect of obesity on high-density lipoprotein metabolism. Obesity (Silver Spring). **15**(12):2875-88.
Re RN (2009). Obesity-Related Hypertension. Ochsner J. **9**(3): 133–136.
Reitman S, Frankel S (1957). A colorimetric method for the determination of serum glutamic oxaloacetate and glutamic pyruvic transaminases. Am. J. Clin. Pathol. **28**: 56-62.

Rettner R (2012). "Is Obesity a Disease? Doctors Debate," www.livescience.com.

Rucker D, Padwal R, Li SK, Curioni C, Lau DC, Padwal, Li, Curioni, Lau (2007). "Long term pharmacotherapy for obesity and overweight: Updated meta-analysis". BMJ **335** (7631): 1194–99.

Schutte R, Huisman HW, Schutte AE, Malan NT (2005). Leptin is independently associated with systolic blood pressure, pulse pressure and arterial compliance in hypertensive African women with increased adiposity: the POWIRS study. J Hum Hypertens.**19**:535–41.

Shantha GP, Kumar AA, Kahan S, Cheskin LJ (2012). Association between glycosylated hemoglobin and intentional weight loss in overweight and obese patients with type 2 diabetes mellitus: a retrospective cohort study. Diabetes Educ. **38**(3):417-26.

Sharma S, Kortas C (2006). Hypertension. eMedicine. http://www.emedicine.com. 1-25. (12-03-16).

Shenqi W, Xuhong H, Yu L, Huijuan L, Li W, Yuqian B, Weiping J (2013). Serum electrolyte levels in relation to macrovascular complications in Chinese patients with diabetes mellitus. Cardiovasc Diabetol. 12:146.

Stewart CP, Christian P, Lee SFW, LeClerq SC, Khatry SK, West KP (2013). Prevalence and Risk Factors of Elevated Blood Pressure, Overweight, and Dyslipidemia in Adolescent and Young Adults in Rural Nepal.Metab Syndr Relat Disord. 11(5): 319–328.

Taskinen MR, Adiels M, Westerbacka J, Söderlund S, Kahri J, Lundbom N, Lundbom J, Hakkarainen A, Olofsson S, Orho-Melander M, Borén J (2011). Dual metabolic defects are required to produce hypertriglyceridemia in obese subjects. Arterioscler Thromb Vasc Biol. **31**: 2144-2150.

Thavasu PW, Longhurst S, Joel SP, Slevin ML, Balkwill FR (1992). Measuring cytokine levels in blood. Importance of anticoagulants, processing, and storage conditions. J. Immunol. Methods **153**:115-124.

Walley AJ, Asher JE, Froguel P (2009). "The genetic contribution to non-syndromic human obesity". Nature Reviews Genetics (Review) 10 (7): 431–42.

Weintraub K (2014) . "New allies in war on weight". The Boston Globe.

WMA. (2000). World Medical Association declaration of Helsinki ethical principles for medical research involving human subjects. 52nd WMA General Assembly, Edinburgh, Scotland.

World Health Organization (WHO) (1995). Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva.

World Health Organization (WHO) (2000). Obesity: preventing and managing the global epidemic. Report of a WHO Consultation. WHO Technical Report Series 894. Geneva.

World Health Organization (WHO) (2004). Expert consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. The Lancet. 157-163.

Yamada T, Hara K, Kadowaki T (2012). Association of adenovirus 36 infection with obesity and metabolic markers in humans: A meta-analysis of observational studies. PLoS One.**7**:e42031.

Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM, Anand SS, INTERHEART Study Investigators. (2005). Obesity and risk of myocardial infarction in 27,000 participants from 52 different countries: A case control study. Lancet.366:1640–9.