

Original Research Article

Implications of Glucose and Creatinine Concentration in Relation to Obesity among the Students of Faculty of Health Science and Technology in Ebonyi State University, Abakaliki, Nigeria

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ABSTRACT

Background: The prevalence of obesity is on the rise and it's now widely recognized as a risk factor for cardiovascular diseases, chronic kidney diseases and other metabolic disorders such as diabetes mellitus type 2. The aim of this study is to know the relationship between body mass index (BMI) in respect to glucose and creatinine clearance in the body.

Materials and Methods: The mean age for the males was 25±7 years while the mean age for the females were 24±5years. The proportion of study population that was overweight and obese was 21.1% and 26.3% respectively.

Result and Discussion: The mean plasma creatinine (male, 120.29±22.14; female, 87.96±16.92; p=0.000) and WHR (male, 0.84±0.039; female, 0.81±0.6; p= 0.003) were all significantly higher in males when compared with that of the females. In the present study, the plasma creatinine level of obese subjects appeared to be insignificantly correlated with BMI and MUAC. BMI showed an insignificant but a negative correlation with plasma creatinine levels in female obese subjects whereas it showed a positive correlation in male obese subjects. However there was no correlation between fasting glucose level of obese female students and their BMI ($r = -0.03$, $p = 0.921$) but fasting glucose level was observed to be positively and insignificantly correlated with MUAC ($r = 0.236$, $p = 0.437$). The relationship was negatively and weakly correlated among the male subjects (BMI; $r = -0.244$, $p = 0.756$ and MUAC; $r = -0.213$ and $p = 0.787$).

Conclusion: This study shows that obesity is more correlated with plasma creatinine level than fasting glucose level and can be a predictor of kidney damage. When considering the entire subjects, the mean values of fasting glucose in gender were similar and insignificant.

Keywords: Glucose, Creatinine, Obesity, BMI

INTRODUCTION

Obesity is a medical condition in which excess fats has accumulated to the extent that it may have a negative effect on

the health; ^[1] or a chronic condition characterized by the accumulation of fat. ^[2] It is defined by body mass index (BMI) and further evaluated in terms of fat distribution

via the waist-hip ratio and total cardiovascular risk factors. [3] The incidence of obesity is increasing, and the number of obese individuals has grown rapidly with continuous improvements in living standards, reduced physical activity, and excessive dietary energy. Studies in Nigeria, have shown that the prevalence of obesity ranges between 6%-22.1%. [4] As the prevalence of obesity increases, so does the prevalence of its associated comorbidities. People are considered to be obese when body mass index (BMI) Kg/m^2 is greater than 30kg/m^2 and overweight when it ranges from $25\text{-}29\text{kg/m}^2$. People who are obese, compared to those with a normal or healthy weight are at increased risk of many serious diseases and health conditions. [5]

Obesity is a widely recognized risk factor for cardiovascular disease and for various metabolic disorders such as type 2 diabetes (T2DM). Serum creatinine values are highly correlated with BMI in patients with T2DM, and these values increased significantly with the increase in BMI and waist circumference. [6] Other reaction indicators of renal function like glomerular filtration rate and serum uric acid (UA) have been shown to be associated with obesity or metabolic syndrome (MetS). [7-9]

Creatinine is a breakdown product of creatine phosphate in muscle, and is usually produced at a fairly constant rate by the body (depending on muscle mass). Creatinine is an important indicator used in the diagnosis of renal health as a result of its production through muscle metabolism and it is excreted unchanged by renal organs. [10] Overweight/obese patients have an increased glomerular filtration rate (GFR) and increased renal plasma flow. [11] Renal hyperfiltration occurs through renal vasodilation in a compensatory response to overcome the increased tubular reabsorption of sodium. However, vasodilation of afferent arterioles increases the hydrostatic pressure in the glomerulus, which can lead to hypertrophy over time and renal disease, even in patients without diabetes. [12] In addition, hyperlipidaemia, leptin, and

adipocyte-derived hormones contribute to the development of glomerular sclerosis. Thus, the results of these studies indicate that obesity independently affects the filtering capacity of the kidneys over time. [12]

The concentration of glucose in the blood is regulated by a complex interplay of multiple pathways modulated by several hormones such as insulin and glucagon. Insulin fundamental effect is to increase glucose uptake into the cells. [13] Decreased biological response to normal concentrations of circulating insulin is known to be as a result of insulin resistance seen in T2DM. [13] Obesity is believed to account for 80-85% of the risk of T2DM, while recent research suggests that obese people are up to 80 times more likely to develop T2DM than those with a BMI of less than 22. It is a well-known fact that anyone that is overweight or obese is at greater risk of developing type 2 diabetes, particularly if they have excess weight around their abdomen. Studies suggest that abdominal fat causes fat cells to release 'pro-inflammatory' chemicals, which can make the body less sensitive to the insulin it produces by disrupting the function of insulin responsive cells and their ability to respond to insulin. This is known as insulin resistance - a major trigger for T2DM.

Less attention has been paid to the link between increased body weight and chronic kidney disease (CKD), although there is evidence that the steady rise in CKD prevalence may be closely associated with increasing obesity. Obesity causes cardiovascular and renal diseases through several mechanisms including hypertension, hyperglycaemia, dyslipidaemia, inflammation, and atherosclerosis. BMI is calculated as weight in kilograms divided by the square of the height in meters. The BMI takes into account both frame size and body composition and is considered to provide a realistically achievable range of healthy weights and is a predictor of dangers associated with obesity. A BMI less than 18.5 kg/m^2 is underweight. Normal BMI

ranges between 18.5 and 24.9 kg/m². A BMI greater than or equal to 25 kg/m² is overweight. Obesity is defined as having a BMI of 30 kg/m² or more. [2]

In a bid to understand the influence of obesity on metabolites, this research considered the serum levels of glucose and creatinine in normal weight and overweight/obese individuals and discussed the mechanism in which obesity may contribute to chronic kidney failure and T2DM.

MATERIALS AND METHODS

Study Area

The study was carried out at the Faculty of Health Science and Technology (FHST), Ebonyi State University (EBSU), Abakaliki. Analysis was done at Federal Teaching Hospital Abakaliki II (FETHA II).

Ethical Consideration

Approval for this research work was obtained from the Research Ethics Committee of Federal Teaching Hospital Abakaliki (FETHA), Ebonyi State, Nigeria.

Subject Recruitment

The individuals that participated in this study were students of FHST both from NSC and MLS departments. It cuts across students of all genders and age from 18 years and above. Greater attention was given to subject with greater body size (BMI >25Kg/m²). The sample size included 28 male and 52 female students (80 participants).

Experimental Materials/Reagents

Test tube, test tube rack, cotton wool, fluoride oxalate anticoagulant container, lithium heparin anticoagulant container, syringe, tourniquet, 70% alcohol, plain container, gloves, measuring tape, weighing scale, laboratory coat, spectrophotometer, centrifuge, Pasteur pipette, distilled water, refrigerator, Creatinine reagent, Glucose reagent.

Sample Size Determination

The formula below was used to calculate the sample size;

Sample size estimation was determined using the formula [Araoye, 2004] for

calculating minimum sample size, $n = \frac{Z^2 \cdot p \cdot q \cdot N}{e^2 (N-1) + Z^2 \cdot p \cdot q}$ where N= size of population=630, n=Minimum sample size, Z=Standard normal deviation usually set at 1.96 which corresponds to 95% confidence interval, P= Prevalence of obesity in South-eastern Nigeria = 6% [4]

(0.06), $q = (1.0 - p) = (1.0 - 0.06) = 0.94$, e = acceptable error set at 0.05.

Hence, $n = \frac{(1.96)^2 \cdot 0.06 \cdot 0.94 \cdot 630}{(0.05)^2 \cdot (630 - 1) + (1.96)^2 \cdot 0.06 \cdot 0.94} \approx 55$.

Therefore n=55. However, selected sample size of 80 participants was used based on the willingness the students to participate, cost and duration of the study.

Collection of Data

Before the subject blood sample is collected, the anthropometric measurement was first taken which includes: the subject's height and weight, head circumference, hips and abdominal circumference, mid upper-arm circumference and body mass index (BMI) of every subject was calculated to classify them into different groups such as underweight (<19kg/m²), normal (19-24.9 kg/m²), overweight (25-29.9 kg/m²) and obese (>30kg/m²).

During blood sample collection, 2 anticoagulant sample container used where made available and they are; fluoride oxalate container which is used in collection of sample for glucose estimation and lithium heparin container for creatinine estimation.

The blood sample used are through venepuncture and since we are to run two biochemical parameters, a total of 7mls of blood is collected and shared among the two anticoagulant containers to avoid double puncture of the subject causing them pain. About 80 students participated in this study as subjects i.e. 160 samples was analysed biochemically in estimation of both creatinine and glucose concentration.

Physical/Athropometric measurements

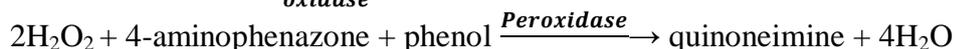
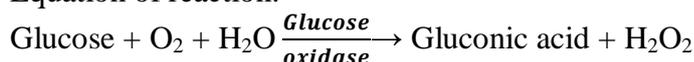
Body mass index is the most popular anthropometric index used for the assessment of obesity among individual subjects. The weight was measured in kilogram with subjects standing on bare feet

in their minimal clothing with their pockets free from what could interfere in the taking of accurate weight measurement such as mobile phones, keys wallets, wrist watches etc. I ensured that the weighing scale was free from zero error.

In measuring the height, it was taken from a measuring scale drawn against the wall. In taking the measurement, the subject was barefoot and without cap and stood against the calibrated wall with his Achilles, gluteus and occiput touching it.

BMI was estimated by dividing the measured weight in kilograms by height in meters squared. It was taken as operational definition of obesity (Siminnialayi *et al.*, 2008) with the following categorization: class 1 obesity (mild obesity) =BMI of 34 -

Equation of reaction:



Procedure: Using a micropipette, 10 µl of the standard and 10 µl of the test sample were added to the test tubes designated for the standard and test sample respectively. 1000 µl of the reagent were added each to the two (2) test tubes mentioned above and also to the test tube designated for the reagent blank. After which these test tubes were mixed respectively and incubated for 10 minutes at 20-25°C. After incubation, small volumes of the reagent blank, standard and test sample were transferred into the cuvettes respectively. The concentration of the test sample was read against the reagent blank at 500 nm wavelength.

Estimation of Plasma Creatinine level

– Jaffe Slot Method was used

Principle: Creatinine in the sample reacts with picrate in alkaline medium forming a coloured complex. The complex formation rate is measured in a short period to avoid interferences.

Procedure

Bring the working reagent to room temperature, after which 1000ul (1.0ml) of

34.9, class 2 obesity (moderate obesity) = BMI of 35-39.9, and class 3 obesity (severe obesity) = BMI of greater or equal to 40. Investigations were carried out for the specific primary co-morbidities of obesity such as hypertension, diabetes mellitus and chronic kidney diseases.

Estimation of Blood Glucose level

– Glucose Oxidase Enzymatic Method was used

Principle: Glucose is determined after enzymatic oxidation in the presence of glucose oxidase. The hydrogen peroxide formed reacts, under catalysis of peroxidase, with phenol and 4-aminophenazone to form red-violet quinoneimine.

this working reagent is pipette into a cuvette. Add 100ul (0.1ml) of the test sample to the cuvette. Mix and insert cuvette into the spectrophotometer immediately; initial absorbance (A_1) is recorded after 30 seconds and final absorbance at 90 seconds (A_2).

Calculation:

$$\frac{(A_2 - A_1) \text{ sample}}{(A_2 - A_1) \text{ standard} \times \text{concentration of standard}}$$

Statistical Analysis

SPSS (version 20.0) was used for all statistical analyses. Analysis of variance (ANOVA) was used to evaluate differences in mean for study groups. Continuous variables were expressed as mean \pm standard deviation. Categorical variables were expressed as number (percentage, %). Using previously described methods, 95% confidence intervals for prevalence estimates were determined [Chobanian *et al.*, 2003]. All reported *P* values are two tailed, and statistical significance was set at 0.05 levels.

RESULTS

Baseline Characteristics

The study consisted of 80 participants (28 males [35%] and 52 [65%] females) with mean age 24.4±5.88 years old. The mean age for the males was 25±7 years, while the mean age for the females was 24±5 years. Table 4.1 shows the mean and standard deviation (SD) of baseline

characteristics of participants and also their statistical significance by Gender. The proportion of study population that were overweight and obese were 21.3% and 26.3% respectively. The mean plasma creatinine and WHR were all significantly higher in males when compared with females.

Table 1: Baseline characteristics of participants and differences by gender

Characteristics	Gender			P- value ^a
	Total (n=80) (%)	Male students (n=28) (%)	Female students (n=52) (%)	
BMI (kg/m ²)				
Underweight (<18.9)	5	2.5	2.5	0.000 ^b
Normal (19-24.9)	47.5	17.5	30.0	
Overweight (25.0-29.9)	26.3	11.3	15.0	
Obese (≥30.0)	21.3	5.0	16.3	
		Mean ±SD	Mean ±SD	P- value ^a
Age		24.96±6.76	24.21±5.39	0.614
Fasting Glucose (mmol/l)		4.13±.71	4.14±.57	0.938
Plasma Creatinine (mg/dl)		120.29±22.14	87.96±16.92	0.000
Waist circumference (cm)		79.89±9.89	79.96±12.00	0.978
Waist-Hip Ratio (WHR)		.84±0.039	.81±0.06	0.003
Mid Upper Circumference		27.61±4.18	27.75±4.16	0.884

Abbreviations: BMI, body mass index;

^aDifferences determined by using 2-tailed independent *t* tests following Levene's test for equality of variances. ^bDifferences determined by using ANOVA

Correlation analysis showed that the association of fasting glucose, plasma creatinine and CGR with BMI and MUAC in Obese students differed (Table 2). However, the plasma creatinine levels were not significantly correlated with BMI and

MUAC in obese students. In addition, the correlation coefficient between of both plasma creatinine and CGR with BMI was negative in female and positive in male obese students.

Table 2: Correlation of fasting glucose and plasma creatinine with BMI and MUAC in obese students

Variables		Total (n=17)		Obese Female Students (n=13)		Obese Male Students (n=4)	
		BMI	MUAC	BMI	MUAC	BMI	MUAC
Fasting Glucose	<i>r</i> -value	.048	.250	.030	.236	-.244	-.213
	<i>P</i> -value	.856	.333	.921	.437	.756	.787
Plasma Creatinine	<i>r</i> -value	.111	.219	-.126	.064	.165	.048
	<i>P</i> -value	.673	.398	.683	.834	.835	.952

BMI, Body Mass Index; MUAC, Mid Upper Arm Circumference

DISCUSSION

The prevalence of overweight and obesity in the study population was 26.3% and 21.3% respectively. The mean plasma creatinine level of male students (120.29±22.14) was significantly higher than those of the female students (87.96±16.92). However, this might be mainly due to differences in muscle mass since creatinine levels are function of muscle mass and further reinforces the effect of sex and muscle mass as factors that affects measurement of plasma creatinine.

In the present study, the plasma creatinine level of obese subjects appeared to be insignificantly correlated with BMI and MUAC. BMI showed an insignificant but a negative correlation with plasma creatinine levels in female obese subjects whereas it showed a positive correlation in male obese subjects. Previous studies have reported an association between the mechanism of renal dysfunction and obesity. Obesity is linked to several functional and structural alterations in the kidney. Renal diseases mainly proceed from hemodynamic abnormalities,

obesity-related dysfunction, metabolic abnormalities, and mast cell-related kidney inflammation mechanisms. ^[14] Obesity is clearly an independent risk factor for kidney damage. ^[15] The relationship between body fat distribution and creatinine did not differ between males and females in correlation analysis suggesting that gender was not a major modifier of the independent relationship between BMI and plasma creatinine. Also in this research, there was no correlation between fasting glucose level of obese female students and their BMI ($r = -0.03$, $p = 0.921$) but fasting glucose level was observed to be positively and insignificantly correlated with MUAC ($r = 0.236$, $p = 0.437$). The relationship was negatively and weakly correlated among the male subjects (BMI; $r = -0.244$, $p = 0.756$ and MUAC; $r = -0.213$ and $p = 0.787$). When considering the entire subjects, the mean values of fasting glucose in gender were similar and insignificant. It has been shown that weight tends to increase after a stressful condition associated with “after stress syndrome”. The Nigerian university community is such that it is characterized by much stress and it has been observed that girls (female undergraduates) tends to rest more after such strenuous periods leading to “creeping weight” due to the sensitization of hormone “ghrelin” that increases the hunger reflexes in both males and females. ^[16] This increase in dietary in-take by male is balanced by exercise but female tends to relax more and as such, there is the tendency of weight gain. Investigations into female anatomy and physiology ^[17] have shown that there is an increase in fat deposition as female increase in age especially from the age of 12 years; to enable them prepare for ovulation and pregnancy. A similar investigation into male anatomy shows that a similar increase also occurs to enable spermatogenesis. ^[17]

A limitation of the study was its cross-sectional design, which cannot be utilized to infer causality. The unwillingness of some students within the study area to participate in the study, the cost and

duration of the study also limited the number of participants recruited for the study. Another limitation was that the sample was not selected from the wider university population and it is therefore recommended that our findings be verified in other Nigerian universities. Based on these research findings of increased BMI among female undergraduates and the health risks associated with increased weight, the university management has to make concerted efforts to address the issues within the limits of what is formally accepted maybe through the introduction of courses involving physical exercise/training programs among all levels and cutting across all discipline. This will help reduce sedentary life among undergraduates especially the female gender.

CONCLUSION

The mean values of fasting glucose level of the obese students were within the normal reference values for both sexes and insignificant whereas their plasma creatinine level were still within the normal reference values and significantly different. This shows that obesity is more correlated with plasma creatinine level than fasting glucose level and can be a predictor of kidney damage. Central or abdominal obesity, a particularly high-risk form of obesity and can predispose one to a greater risk of developing type 2 diabetes. However, given the prevalence of obese individuals (21.3%), prevention of obesity among these young adults should be encouraged especially among the females to reduce the risk of coronary heart disease, diabetes and other consequences of obesity in later years.

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