

Original Research Article

Assessment of Plasma Total Homocysteine in Patients with Obesity in Nigerian Tertiary Hospital

Dalili, M. S¹, Dungus, M. M^{2*}, Aisha, S.K³, Hadiru, G. M⁴, Amali, A. O⁵, Hassan, A. A⁶, Gademi, F. M⁷, Fatima, M. L⁸, Loskurima, U⁹, Musa, A. H¹⁰, Mshelia, D. S¹¹

¹Department of Chemical Pathology, Federal University of Health Sciences, Azare, Bauchi State, Nigeria

²Department of Chemical Pathology University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

³Department of Chemical Pathology University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

⁴Department of Chemical Pathology University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

⁵Department of Internal Medicine, University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

⁶Department of Internal Medicine, University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

⁷Department of Chemical Pathology, University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

⁸Department of Chemical Pathology, University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

⁹Department of Internal Medicine, University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

¹⁰Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, University of Maiduguri, Borno State, Nigeria

¹¹Department of Chemical Pathology, Faculty of Basic Clinical Sciences, University of Maiduguri, Borno State, Nigeria

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Abstract: The objective of this study was to determine the plasma levels of homocysteine and lipid profile in obese and normal body weight patients (controls). The study group consisted of 50 obese patients while the control group had 50 non obese clients. Plasma homocysteine, total cholesterol (TC), low density lipoprotein (LDL - C), High density lipoprotein (HDL - C) and triglycerides (TGs) were measured. The levels of total homocysteine (THCY), T-C, LDL-C, HDL-C and TGs in the obese subgroup were significantly higher than those in the non-obese controls. The elevated levels of HCY and lipid parameters also correspond to elevation of body mass index (BMI) and waist to hip circumference (WHCR) in the obese subjects.

Keywords: Thcy, Lipid Profile, Obesity.

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INTRODUCTION

Homocysteine, a sulphur-containing amino acid derived from the demethylation of methionine. This amino acid may be further metabolized by the sulfuration pathway to cysteine, or remethylated in the liver have been found to confirm thrombotic tendency [1]. Recent advances have shown that hyperhomocysteinemia ($> 15\mu\text{mol/L}$) is a major and independent risk factor for cardiovascular disease [2, 3]. Some studies have postulated that homocysteine might cause atherosclerosis by damaging the endothelium by altering oxidative status and elevated plasma homocysteine may promote the production of hydroxyl radicals through autoxidation [4].

Obesity defined as a Body Mass Index (BMI) of $\geq 30\text{kg/m}^2$ is due to deposition of fat in the fat

compartments of the body [5]. It is due to growing trend of sedentary life style and increase consumption of high caloric diet [6, 7]. It is linked with metabolic complications such as hypercholesterolemia, hyperinsulinemia and dyslipidemia [8].

High levels of homocysteine are linked with obesity and the early development of heart and vascular disease. Its high level is considered an independent risk factor for heart disease. There is a link between homocysteinemia and damage to the arteries causing atherosclerosis and the formation of blood clots [5, 9].

Elevated BMI, excess use of pesticides, low consumption of folate and Vitamins are possible risk factors for hyperhomocysteinemia [9]. Genetic, metabolic errors and environmental factors have also

*Corresponding Author: Dungus, M. M

Department of Chemical Pathology University of Maiduguri Teaching Hospital, Maiduguri, Borno State, Nigeria

been found to be associated with elevated plasma homocysteine level [9]. Homocysteinuria, an autosomal recessive disorder due to a deficiency of the enzyme cystathionine β – Synthase have been found to have a thrombotic tendency [9].

MATERIALS AND METHOD

i. Subjects:

This is a case-control study comprising a total of 100 individuals with 50 obese patients and 50 healthy non-obese individuals aged 21 – 60 years as cases and controls respectively, who attend the metabolic clinic at the University of Mauduguri Teaching Hospital from February to July 2022. Both cases and controls were recruited under informed consent with approval from the institution’s ethical committee.

Patients and controls were physically examined by the researcher that included their blood pressure, body weight, height and their body mass index (BMI) was calculated using weight in kilogram (kg) divided by height in meter square (m²) (BMI = wt(kg)/height(m²). Fasting plasma homocysteine, fasting serum total cholesterol, HDL –Cholesterol, triglycerides were analysed using commercial kits while LDL-Cholesterol was calculated using Friedewald’s equation.

Patients were considered to have high plasma homocysteine if the level exceeds 15µmol/L in both sexes. The total cholesterol is said to be elevated if the level exceeds 5.7 mmol/L, the triglycerides level are said to be high if their level exceeds 1.7mmol/L. HDL-cholesterole should normally be more than 0.9mmol/L. while the LDL-cholesterol should be < 2.7mmol/L [10,11].

ii. Study Design;

Patients were excluded if they are hypertensive, Diabetic, pregnant, have cardiovascular disease or medications that affect plasma homocysteine levels and lipid lowering level drugs.

All subjects were asked to complete a questionnaire comprising of socio-demographic data that involved age, nutrition as well as medications. Subjects

were not on any medications and were on their routine diet.

A fasting blood sample of 5ml was collected from each individual using a disposable 5ml needle and syringe and transferred in to a lithium heparine bottle (Homocysteine) and plain bottle (Lipids) under aseptic procedure. The samples were transported to the chemical pathology laboratory.

Samples were spun using a swinging bucket centrifuge at 3,000 revolution per minute (rpm) for 10 minutes. The cells were then separated from the plasma using a Pasteur pipette. The plasma and serum were stored-frozen at -20⁰ until time of analysis.

iii. Analytical Methods

Plasma total cholesterol was estimated using modified Liebermann-Burchard reaction spectrophotometrically. HDL-Cholesterol evaluation involved a two-staged procedure using the same method as in total cholesterol. LDL-Cholesterol was calculated by Friedewald’s equation while triglycerides were estimated using enzymatic methods on commercial kits.

The plasma homocysteine was estimated using human homocysteine (HCY) Elisa kit that applies the competitive enzyme immunoassay technique (MyBiosource.com).

Statistical Analysis

Data collected were subjected to estimation of mean ± standard deviation (±SD). Student’s t-test was used for non-paired group analysis. P- Values < 0.05 were considered statistically significant. The SPSS/PS statistical package was used by a biostatistician from the college.

RESULTS

Table I shows the characteristics of the study population based on age, BMI and WHCR. The mean characteristic of their ages in the subjects were 40.5 ± 4.8 while 43.0 ± 5.2 was obtained in the control group. Their BMI, WHCR was 33.10 ± 2.90 (P=0.02) and 0.93 ± 0.04 (P=0.03) in the subject and 19.04 ± 1.3 (P=0.02), 0.78 ± 0.05 (P=0.03) was respectively in the control group were statistically significant.

Table I: Shows the characteristics of the study population based on age, BMI and WHCR

Parameters	Subjects (n = 50)	Control (n = 50)	P – Value
Age (years)	40.5 + 4.8	43.0 + 5.2	-
BMI (kg/m ²)	33.10 + 2.90	19.04 + 1.3	0.02
WHCR	0.93 + 0.04	0.78 + 0.05	0.03
P<0.05 – Significant			

Table II shows the mean biochemical parameters of the study population. The mean values of homocysteine (µmol/L) was 29.20 ± 8.10, 7.30 ± 3.40 were statistically significant (P – 0.02). The mean values

of total cholesterol (TC), LDL-cholesterol, HDL – cholesterol and triglycerides (TGs) respectively were 6.2 ± 1.5 (P – 0.03), 3.9 ± 1.3 (P – 0.02), 0.60 ± 0.02 (P – 0.03), 2.4 ± 0.4 (P – 0.01) in cases were statistically

significant. The mean values of T - C, LDL - C, HDL - C and TGs were respectively, 4.2 ± 2.1 (P - 0.03), $2.6 \pm$

1.3 (P - 0.02), 1.4 ± 0.6 (P - 0.03), 0.8 ± 0.2 (P - 0.01) in the control group.

Table II: Shows the mean biochemical parameters of the study population

Parameter	Cases (n=50)	Control (n=50)	P - value
Homocysteine (µmol/L)	29.20 + 8.1	7.30 + 3.4	0.02
Total Cholesterol (mmol/L)	6.2 + 1.5	4.2 + 2.1	0.03
LDL Cholesterol (mmol/L)	3.9 + 1.3	2.6 + 1.3	0.02
HDL Cholesterol (mmol/L)	0.60 + 0.02	1.4 + 0.6	0.03
Triglycerides (mmol/L)	2.4 + 0.4	0.8 + 0.2	0.01
P < 0.05 - Significant			

DISCUSSION

In a meta-analysis that looked at 14 studies that assessed homocysteine and obesity by Jinxiang Wang *et al.* they found remarkable lower concentrations of homocysteine in the controls than obese patients [10]. This is in agreement with our study which also indicates lower control values of homocysteine than in the cases. The assessment of Jinxiang Wang *et al.*, is regardless of nutritional status, dietary habit, insulin resistance status, specific disease history, history of medications and so on. Homocysteine concentrations in non-obese patients with polycystic ovarian syndrome (PCOS) were lower than obese patients with PCOS [12].

According to a study by Siegfried *et al.*, plasma total homocysteine level have been shown to be associated with obesity, insulin resistance in obese children and adolescents. They investigated the role of changes in body composition, Markas of insulin resistance, folate and Vitamin B12 to changes in homocysteine during a weight reduction program in obese children and adolescents. During the weight reduction BMI, Fat Mass (FM), percentage fat mass, insulin and C - Peptide decreased significantly, whereas homocysteine and Vitamin B12 showed significant increase. Folate and lean body mass (LBM) remain unchanged [13]. Total homocysteine (tHCY) concentration before weight reduction was a function of age, folate, and C-peptide, whereas tHCY concentration after weight reduction was a function of folate and base line LBM. Changes in tHCY during weight reduction correlated significantly with baseline LBM and were related inversely to changes in LBM during weight reduction.

Children with increase LBM showed lower increases in tHCY compared with children with low LBM. Their study suggest that LBM has a significant impact on tHCY metabolism during weight reduction [13]. This study therefore agrees proportionately with our study.

However, a measure limitation particularly to the developing country like ours is the high cost of running homocysteine.

CONCLUSION/RECOMMENDATION

The results obtained in this study indicate that patients with obesity have been found to have hyperhomocysteinemia, a known risk factor for cardiovascular disease and independent risk factor for heart disease. In this regard, obese patients as a routine should have their homocysteine plasma levels evaluated.

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