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PLASMA C - REACTIVE PROTEIN AND LIPID PROFILE IN PATIENTS WITH OBESITY AT A TERTIARY HEALTH CENTRE ABUJA, NIGERIA

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ABSTRACT

The objective of this study was to determine the levels of C-reactive Protein (CRP) and Lipid Profile in obese and normal body weight patients (controls). The study group consisted of 120 obese patients while the control group had 80 non obese clients. Highly sensitive CRP, total cholesterol CT"-C,) Triglycerides (TGs,) High Density Lipoprotein (HDL-C), and Low Density Lipoprotein (LDL-C) were measured. The levels of CRP, T-C, TGS, HDL-C and LDL-C in the obese subgroup were significantly higher than those in the non-obese controls. The elevated levels of CRP and Lipid Parameters also correspond to elevation of body mass index (BMI) and waist to hip circumference (WHR) in the obese subject.

KEYWORDS: CRP, Lipid Profile, Obesity.

INTRODUCTION

Obesity is associated with a low-grade inflammation which may play a significant role in the development cardiovascular complications.[1] C-reactive protein (CRP) is an extremely sensitive marker of systemic inflammation produced mainly by the liver under the stimulation of adipocyte-derived proinflammatory cytokines.[2]

CRP has also emerged as a powerful predictor of cardiovascular diseases.[3] Elevated levels of CRP have been associated with man's metabolic disorders such as type II Diabetes Mellitus (DM), hypercholesterolaemias and hypertriglyceridemias.[4] Some studies indicate a relationship between CRP and macrovascular complications as in coronary heart disease.[5] It has not yet been shown that the CRP level increases due to the metabolic effects of obesity and plays a direct role in promoting the inflammatory component of atherosclerosis or whether it is merely a marker of the ongoing inflammation in the vessel affected.[6] Data on elevated levels of CRP in obese patients providing a link between inflammation and the development of macrovascular complications appears to be scanty.[7] The purpose of this study was to evaluate the levels of CRP and Lipid Levels in obese patients and their non-obese counterparts.

2.0. PATIENTS AND METHODS

2.1. Study Design and Setting

This is a cross-sectional study that lasted for about two years between February 2017 and December 2019. The study group consisted of 120 obese individuals (69 men and 51 women) aged 41 - 62 years (53.5 $\pm$ 6.6 years). Patients were recruited from the metabolic Clinic of University of Abuja Teaching Hospital.

Obesity is defined as an imbalance between excessive energy intake to energy expenditure measured in BMI (Body mass index). A BMI of $\geq$ 30kg/m^2 classified over weights.

2.2. Ethical clearance

Approval of the study was obtained from the ethical committee of the University of Abuja Teaching Hospital, Gwagwalada Abuja.

Inclusion Criteria

All willing patients who consented and fall between the age of 43-65 years were included.

Exclusion Criteria

Patients with impaired glucose tolerance, diabetic mellitus were excluded using oral glucose tolerance test (75g). Persons with suspected ischemic heart disease, cerebrovascular disease, hypertension, non-consenting patients were all excluded in the study.

Informed consent was obtained for inclusion in the study and each patient was completed a bio-data form. Obesity with body mass index (BMI) of 30.0 - 39.5 kg/m2) (34.8 ± 3.9 kg/m2) treated with diet and exercise.

The control group (non-obese) comprised 80 persons (30 men and 50 women) aged 43 - 65 years) (54.0. ± 6.8) with BMI of 18.6 to 23.6 kg/m2 (2.1 ± 1.2 kg/m^2).

Physical examination and measurement of BMI and waist/hip circumference ratio (WHCR) were done. The plasma levels of CRP were estimated and the patient's blood pressure, height and body weight were all evaluated in all subjects and an additional laboratory investigations such as total, HDL and LDL cholesterol and triglycerides were performed. The concentration of CRP was estimated by Colorimetric Enzyme linked immune sorbent Assay (ELISA) (Monobind Inc kit, Accubind hs-CRP product code: 3125 - 300, USA).

Total cholesterol was estimated spectrophotometrically (manual) using modified Liebermann - Burchard reaction. The estimation of HDL- Cholesterol was performed using 2-stage procedure and shared the same method with total cholesterol. The LDL -cholesterol was calculated from Friedewald's reaction. The triglyceride was estimated using enzymatic methods. The reference interval for has CRP Acubind (R) ELISA Microplate test system based on study on apparent normal population states that, values < lug/ml are low risk, 1-3 uq/ml are considered normal and >3 uq/ml confirm high risk of coronary heart disease.[8]

Desirable, borderline and undesirable levels of total cholesterol (T-C), LDL-C, HDL-C and TG were as follows; Desirable level (mmol/L) for TC is <5.1, for LDL-C is <3.5, HDL-C is >1.4, and TGs is <1.5.[9, 10]

Borderline level (mmol/L) of T-C is between 5.1 - 6, LDL-C is between 3.3 - 3.9, HDL - C is between 1.1 - 1.4 and TGs are devoid of borderline values. Undesirable levels of these parameters (rnrnol/L) include TC of ≥ 6.0, LDL-C of ≥ 3.9 HDL-C of < 1.1, and TGs of < 1.3.[9,10]

2.5 Data Analysis

Data analysis was computed using SPSS version 21.0 computer software packages. Tests of significance was with chi-square or fisher exact test (whenever the expected frequency was less than 5) and logistic regression was used for categorical variables, and analysis of variance (ANOVA) or student's t-test for continuous variables. Differences between the groups were considered significant at $p<0.05$.

RESULTS

Table 1 shows the characteristics of the study population based on age, sex, BM! and WHCR. There were 69(57.5%) males and 51(42.5%) females in the subjects while 30(37.5%) males and 50(62.5%) females were in the control group. The mean characteristics of their ages in the subjects were 51.5 ± 6.3 while 54.0 ± 6.5 was obtained in the control group. Their BMI, WHCR was 34.2 ± 3.0 (p=0.02) and 0.94 ± 0.06 (p=0.03) in the subjects and 20.04 ± 1.6 (p=0.02), 0.80 ± 0.05 (p=0.03) was respectively in the control group were statistically significant.

Table 2 shows the means biochemical parameters of the study population. The mean CRP (uq/ml) was 7.0 ± 2.0 (p=0.0l) in the experimental group while the control group on the other hand had mean CRP of 0.8 ± 0.12 uq/rn (p=0.0l) which was statistically significant. The mean values of TC, LDL-C, HDL-C and TGs respectively were 6.9 ± 1.8 (p=0.04), 4.1 ± 1.6 (p=0.02), 0.8 ± 0.02 (p=0.01), 2.2 ± 0.6 (p=0.0) in the experimental group were statistically significant. The mean values of TC, LDL-C, HDL-C and TGs were respectively, 4.6 ± 2.2 (p=0.04), 2.8 ± 1.4 (p=0.02), 1.6 ± 0.8 (p=0.0l), 1.2 ± 0.4 (p=.0.00) in the control group.

Table 1: Mean values of Characteristics of the study population according to sex.

Parameter	Subject (n-1200	Control (n-80)	P – value
Sex (M/F)	69/51	30/50	-
Age (year)	51.5 ± 6.3	54.0 ± 6.5	0.16
BMI (Kg/m^2)	34.2 ± 3.0	20.4 ± 1.6	0.02
WHCR	0.94 ± 0.06	0.80 ± 0.05	0.03

P < 0.05 – Significant

Table 2: Mean values of Biochemical Parameters of study population.

Parameter	Subject (n-1200	Control (n-80)	P – value
CRP (ug/ml)	7.0 ± 2.0	0.8 ± 0.12	0.01
Total Cholesterol (mmol/L)	6.9 ± 1.8	4.6 ± 2.2	0.04
LDL Cholesterol (mmol/L)	4.1 ± 1.6	2.8 ± 1.4	0.02
HDL Cholesterol (mmol/L)	0.8 ± 0.2	1.6 ± 0.8	0.01
Triglycerides (mmol/L)	2.2 ± 06	1.2 ± 0.4	0.00

DISCUSSION

This basic study looked at CRP and lipid parameters in asymptomatic obese subjects and normal individuals. Chronic inflammation is believed to play an important role in people with obesity mediated by cytokines that modulate inflammatory reactions. Immune cells activated by hyperglycemia and associated metabolic disorders play a key role. The purpose of having two groups, the obese and normal body weight was to enable the assessment of the extent to which CRP is determined by the presence of obesity in which adipose tissue is believed to initiate and sustain inflammation in such individuals. Higher CRP levels in these obese subjects which was not observed in the control group, suggest that obesity is a state corresponding to subminimal inflammation. This is consistent with data reported by other authors.[11, 12, 13]

It has been observed that the levels of CRP in the subjects (obese patients) were elevated correspondingly to the levels of BMI and WHCR. This is not surprising since obesity is associated with inflammation and WHCR represents visceral obesity since, according to current studies, the principal place of production of inflammatory cytokines and proteins are visceral tissue.[14, 15] Trayhurn P, Wood IS and Weisberg et al, It has also been observed that the increase in the levels of total cholesterol, triglycerides, HDL-C and LDL-C in the obese subjects also correspond to an elevation of both BMI and WHCR. It is important to remember that in order to directly determine the amount of visceral fat, it requires the use of radiological techniques (CT, MRI) which would distinguish visceral and subcutaneous fat.[16]

In addition recent reports suggest that apart from visceral tissue, the perivascular adipose tissue surrounding virtually all blood vessels, may also be a source of inflammatory cytokines.[17]

CONCLUSION AND RECOMMENDATION

The results obtained in this study indicate that the most important factor determining an increase in the concentration of CRP in obese subjects is excess body fat and existence of hyperlipidaemias. This study forms a basis for further study that may reveal the extent of cardiovascular system involvement like Hypertension or coronary heart disease, myocardial infarction.

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