DOI:10.24041/ejmr2019.133

ASSOCIATION OF ANTHROPOMETRIC MEASUREMENTS WITH IMPAIREMENT OF BLOOD BIOCHEMISTRY ANALYTES IN OBESE SUBJECTS FROM DIFFERENT SOCIO-ECONOMIC STATUS

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Received on : 10-02-2019
Accepted on : 04-04-2019

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ABSTRACT

This case control study had approved by institutional human ethics of Era's Lucknow Medical college and hospital.Study had designed, to explore association of anthropometric measurements with impairment of blood biochemistry analytes in obesity and overweight among subjects from different socioeconomic status. As well as to detect prevalence of obesity and overweight, on criteria, a) Laid down by Govt. of India and b) Laid down by World Health Organization (WHO) in subjects of different socioeconomic status.Under graduate medical, paramedical and nursing students as well as hospital lab services (HLS) staff had included in this study. Study had carried out in Department of Biochemistry with collaboration of Department of Physiology and HLS. Results of this study had showed that, prevalence of obesity and overweight were more in high socioeconomic status with respect to students of middle socioeconomic status. Participants were investigated for serum lipid profile, Lipoprotein constituents along with levels of lipid peroxide, reduced glutathione and antioxidant enzymes. A marked impairment in levels of lipid profile accompanied with increase in the lipids and apo-protein levels of serum β lipoproteins following decrease in lipid and protein constituents of α lipoprotein, serum reduced glutathione as well as level of antioxidant enzymes in obese and morbid obese subjects with respect to overweight.

KEYWORDS: Different socioeconomic status, Anthropometric measurements, Indian & Criteria of BMI to detect Obesity.

INTRODUCTION

Obesity is defined as an accumulation of excess body fat. Obesity decreases life expectancy, increases the risk of developing type II diabetes mellitus, coronary heart disease, hypertension and also cancer of colon, breast, prostate and endometrium (1). Published evidence suggests that obesity is almost invariably associated with a chronic low rank inflammation and oxidative stress and therefore remains calamitous for health and scourge on mankind (2). The disproportionately rapid rise in the prevalence of overweight and obesity in both developed and developing nations clearly indicate that environmental changes are the major determinants of this epidemic (3). Of course, genetic disposition may act independently or may abet the environmental factors (4). A closely related parallel relationship between excessive adiposity and poor health outcome is clearly visible. The adverse effects of obesity have explicitly been linked with serious diseases like diabetes mellitus, cardiovascular disease and cancer, to name a few. Further, there is mounting evidence now that obesity also induces and/ or promotes a number of other diseases and that it increases the chances of premature death unless managed properly (5). In the recent years, however, two more parameters have been in frequent use namely waist/hip ratio and waist circumference (6). In many populations, all these three parameters have been found to be related where as in some population's waist/hip ratio and waist circumference have emerged as a better expression of obesity, the latter addressing the central obesity,a better predictor of risk. It has been stressed by WHO and other workers that these parameters should be assessed in different populations because categorization may differ from population to population owing to differences in the standards of nutrition, environmental variants, genetic disposition and finally unavoidable abdominal adiposity. Anthropometric measurements are useful in many fields. For example, athletes

understand that body size and composition are important factors in sports performance. For example, a petite man with a low percentage of body fat will be more successful as a jockey in the Kentucky Derby than he would be as a defensive lineman in the National Football League. Sports coaches can also use these measurements to monitor an athlete's body to ensure they stay in their peak physical shape (7).

Dyslipipoproteinemia is an independent risk factor for the development of coronary artery diseases, myocardial infarction, and hypertension in hyperlipidemic patients [1]. Clinically, diabetic patients are characterized by a marked increase in blood glucose level followed by normal or mild hyperlipidemia. Elevated level of low density lipoprotein (LDL) along with increased triglycerides, especially very low density lipoprotein (VLDL) and cholesterol in low density lipoprotein, lead to free radical mediated formation of modified LDL. This mechanism is recognized as a leading cause for the development of atherosclerosis in obese and morbidly obese subjects. Furthermore, hyperlipidemia may also induce abnormalities like resistance to insulin in the muscle and liver cells in obese and morbidly obese subjects (8).

MATERIALS AND METHODS

This study was approved by the institutional ethics of human ethics and it was carried out to detect the prevalence of obesity and excess body weight amongst the students and staff of Era's Lucknow Medical college and Era Institute of Allied Health Sciences and Research, Lucknow. Subjects from different socioeconomic status were classified on two criteria of BMI namely a. WHO criteria and b. Criteria of the Govt. of India. We also aimed to compare the relative importance of BMI, waist circumference and waist/Hip ratio for health assessment.Anxiety to curtail the obesity burden in India is very much evident by the fact that the Govt. of India has framed new guidelines to define obesity and overweight in our population. The Government has also conducted several surveys to assess the prevalence of obesity in different regions and has suggested measures to control it. Lastly, but most importantly, the criteria laid down by Government of India and WHO (Table 1 & 2)for obesity and overweight on the basis of BMI and waist circumference are different.

Criteria laid down by Govt. of India		
S. no.	**Category**	**BMI**
1.	Underweight	<18.4 Kg/m2
2.	Normal	18.5-22.9 kg / m2
3.	Overweight	23-24.99 kg / m2
4.	Obese	25-32.4 kg / m2
5.	Morbid Obese	≥ 32.50 kg / m2

Table 1: Indian Criteria Of BMI Cut Offs Were Classified In Accordance With The Revised Indian Guidelines For BMI To Detect Obesity By Union Health Ministry Of India, 2005. *(7)*

Cont. Table 1: Indian Criteria Of BMI Cut Offs Were Classified In Accordance With The Revised Indian Guidelines For BMI To Detect Obesity By Union Health Ministry Of India, 2005. *(7)*

Cutoff waist circumference

Male: 90 cm

Female: 80 cm

Selection of Subjects

All volunteering students and staff of Era's Lucknow Medical college and Era Institute of Allied Health Sciences and Research, Lucknow, from different socioeconomic status were included in this study after a written consent.

Anthropometric measurement

For anthropometric measurements (weight, height, waist and hip circumference) standard methods were used (9-10).

Study Design

Subjects were the students and staff of Era's Lucknow Medical college and hospital and were from different socioeconomic status (high, middle and low). All Subjects were divided in to 4 groups (Group 1 to 4; Table 1):

Group 1: Under graduate medical students (Age 19.07 ±1.36, batch 2009 and 2010: n=200, High socioeconomic status)

Group 2: Under graduate Nursing Students (Age 19.08±1.35, batch 2009 and 2010: n=30, Middle socioeconomic status)

Group 3: Under graduate para medical Students (Age 19.06 ±1.29, batch 2009 and 2010: n=30, Middle socioeconomic status)

Group 4: HLS Staff (Age 19.09 ±1.47, 2010:n= 30, Low socioeconomic status)

After assessment of oxidative stress and anthropometric measurements in these groups,the subjects were categorized in three final groups:

Overweight (BMI 23-24.99 kg / m2, n = 30),

Obese(BMI 25-32.4 kg / m2, n=30), and

Morbidly Obese(BMI ≥ 32.50 kg / m2,n=30)

Collection of Blood Samples

Blood samples were collected from the median cubital vein of the subjects, using disposable plastic syringes with all standard aseptic precautions. The sample was transferred immediately in toa dry clean plastic test tube with a gentle push to avoid hemolysis.

Separation of Serum

To separate the serum, the whole blood was kept in a plain (red top) vacutainer at 37 °C for 30 minutes after which this coagulated sample of blood was centrifuged at 1500 rpm for 15 minutes at 4°C in Eppendorf centrifuge machine. The supernatant was pipetted out in a new tube and kept at - 20 °C till analysis. (11)

Biochemical Analysis of Serum, Plasma and Lysate

The blood was centrifuged and plasma was separated. The fasting blood sugar (FBS) (10) was analyzed in plasma while glycosylated hemoglobin (HbA1C) (11), super oxide dismutase (SOD) (12), catalase (CAT) (13), glutathione peroxidase (Gpx) (14) and glutathione reductase (GR) (15) were estimated in RBC lysate, serum totalcholesterol (TC) (16), triglyceride (TG) (17), high density lipoprotein total cholesterol (HDL-TC) (18) were assayed by standard spectrophotometric methods. Low density lipoprotein, total cholesterol (LDL-TC) and very low density lipoprotein total cholesterol (VLDL-TC) were calculated by Friedewald's equation (19). The serum was also used for the assay of lecithin cholesterol acyl transferase activity (LCAT) (20), lipid peroxide (LPO) (21), and reduced glutathione (GSH) (22). A portion of serum was fractionated into very low density lipoprotein (VLDL), low density lipoprotein (LDL) and high density lipoprotein (HDL) by polyanionic precipitation methods (20,23) Lipoproteins were measured for their total cholesterol (TC) (16), phospholipids (PL) (24), triglyceride (TG) (17) and apoprotein (25) by standard spectrophotometric methods.

Statistical Analysis

One-way-analysis of variance (ANOVA- Newman's student test) was performed by comparison of values for CAD with T2DM group with control. All hypothesis testing were two-tailed. P <0.05 was considered statistically significant and the results were expressed as mean ± SD. The graph pad INSTAT 3.0 software was used to carried out the statistical analysis 26.

RESULTS

Experimental schedule	Fasting Blood sugar	Glycosylated Hemoglobin (g%)	Serum LCAT (mmol/ L/hr)	Serum lipid profile				
				TC (mg/dl)	TG (mg/dl)	LDL-TC (mg/dl)	VLDL-TC (mg/dl)	HDL-TC (mg/dl)
Overweight (BMI23-24.99 kg/m2, n = 30)	92.26 ±9.05	4.98 ± 0.53	80.54 ± 14.97	200.26 ± 21.67	110.06 ± 20.18	127.24 ±24.57	21.89 ±7.56	49.80± 9.17
Obese (BMI 25-32.4 kg / m2, n=30)	145.40 ± 10.86* (+ 57%)	6.95 ± 0.78* (+ 40%)	65.78 ± 13.18* (-17%)	268.53 ± 11.36* (+34 %)	175.00 ±28.01* (+59 %)	201.17 ± 14.42* (+58%)	35.00 ±5.60* (+60%)	30.53 ±4.44* (-39%)
Morbidly Obese (BMI ≥ 32.50 kg / m2,n=30)	165.40 ± 10.86* (+ 57%)	7.33 ±0.99* (+ 40%)	62.33± 11.78* (-17%)	291.83 ±12.26* (+34 %)	181.10 ±41.11* (+59 %)	212.17± 14.42* (+58%)	36.97 ±5.60* (+60%)	29.13 ±5.44* (-39%)

Table 2: Association Of Blood Sugar Fasting, HBA1C, LCAT And Lipid Profile In Obese And Morbidly Obese Participants With Respect To Overweight (Different Socioeconomic Status)

Values are expressed as mean SD of 30 subjects, obese group was compared with overweight group and morbidly obese group was compared with obese group *p<0.001.

Experiment schedule	VLDL				LDL				HDL			
	TC (mg/dl)	PL (mg/dl)	TG (mg/dl)	Apo-protein (mg/dl)	TC (mg/dl)	PL (mg/dl)	TG (mg/dl)	Apo-protein (mg/dl)	TC (mg/dl)	PL (mg/dl)	TG (mg/dl)	Apo-protein (mg/dl)
Overweight (n=30)	20.00 ±9.56	35.00 ±3.97	36.00 ±3.97	13.00 ±2.44	130.00 ±16.47	38.00 ±3.18	37.12 ±2.19	27.13 ±1.62	56.80 ±9.17	88.55 ±9.35	19.40 ±1.79	179.00 ±11.34

Table 3: Association Of Lipoprotein Profile In Obese And Morbidly Obese With Respect To Overweight (different Socioeconomic Status)

Obese (n=30)	33.00 ±6.60* (+65%)	82.00 ±8.48* (+134%)	59.77 ±5.70* (+64%)	19.00 ±0.76* (46 %)	200.17 ±14.42* (+54%)	70.59 ±8.42* (+86%)	63.21 ±7.27* (+70%)	40.55 ±1.46* (49%)	49.98 ±8.47* (-46%)	52.53 ±6.28* (-41%)	13.55 ±1.28* (-30%)	119.44 ±14.33* (-34%)
Morbidly Obese (n=30)	39.00 ±6.60* (+65%)	88.99 ±9.87* (+134%)	65.00 ±5.70* (+64%)	23.44 ±0.76* (46 %)	210.85 ±17.92* (+54%)	74.59 ±8.42* (+86%)	67.28 ±7.27* (+70%)	44.95 ±1.33* (49%)	30.93 ±5.47* (-46%)	48.53 ±6.28* (-41%)	12.00 ±1.28* (-30 %)	111.77 ±13.77* (-34%)

Cont. Table 3: Association Of Lipoprotein Profile In Obese And Morbidly Obese With Respect To Overweight (different Socioeconomic Status)

Values are expressed as mean SD of 30 subjects, obese group was compared with overweight and morbidly obese group was compared with obese *p<0.001.

Experiment schedule	Status of markers used for oxidative stress in Serum		Status of Antioxidant Enzymes in RBC Lysate			
	GSH (mg/dl)	Lipid peroxide (nmol MDA/ml)	SOD (Unit/minute/ mg protein)	Catalase (Unit/minute/ mg protein)	Gpx(n mole NADPH Oxidized/min/ mg protein)	GR(n mole NADPH Oxidized/ min/mg protein)
Overweight (n=30)	30.00 ± 5.76	2.27 ± 0.56	4.00 ± 0.19	3800 ± 252.00	344.38±170.00	240.00±38.88
Obese (n=30)	15.79 ±3.63* (-47%)	7.00 ±2.36* (+208%)	2.00 ± 0.18* (-50%)	2934 ± 267.08* (-21%)	333.00 ± 97.56* (-24%)	135.00 ± 40.13* (-45%)
Morbidly Obese (n=30)	17.13 ±5.42* (-47%)	13.99 ± 2.36* (+208%)	2.76 ± 0.98* (-50%)	2877 ± 267.43* (-21%)	299.00 ± 97.96* (-24%)	134.00 ± 80.13* (-45%)

Table 4: Association Of Oxidative Stress And Antioxidant Enzymes In Obese And Morbidly Obese With Respect To Overweight (Different Socioeconomic Status)

Values are expressed as mean SD of 30 subjects, obese group was compared with overweight, morbid obese group was compared with obese *p<0.001.

DISCUSSION

When fat droplets are overloaded in adipocytes, TG becomes extracellular. Such TG cannot be metabolically reutilized and forms the dead bulk in obese individuals. Results of this study are showing that prevalence of obesity was more in subjects of high socioeconomic status with respect to the middle socioeconomic status. On the other hand, prevalence was more in the middlestrata when compared with the low socioeconomic status. It may be due to the sedentary life style of the participants in the high and middle socioeconomic status as well over eating and over nutrition. Our observation is dependable with frequent previous studies which have documented a strong correlation between sedentary life style, decreased GSH and increased MDA with obesity. Our data suggests a possible role of GSH and MDA in the pathophysiology of obese individuals. The World Health Organization (WHO) estimated that approximately half a million people in North America and Europe died from obesity related chronic diseases in 2002, and this is set to increase by one third over the next 20 years if nothing was done. As current intervention strategies are failing to result in sufficient weight loss to reduce the levels of obesity, it is now thought that rather than concentrating on weight loss as a sole success endpoint, reduction in the risk of obesity related chronic disease should also be an important consideration. Modest weight loss has been associated with reduction in total mortality, reduction in total cholesterol, obesity related cancers, diabetes related deaths and hypertension (20-26). The Coronary Artery

Disease (CAD) progress in Young Adults Study, the Atherosclerosis Risk in Communities and the prevention of weight gain may be the easiest way to prevent the development of undesirable changes in Cardiovascular Disease (CVD) risk factors including, increased Low Density Lipoprotein (LDL) cholesterol, total cholesterol, triglycerides, fasting glucose, and decreased High Density Lipoprotein (HDL) cholesterol (27-34). Thus this type of study will be very useful at national and International Levels.Interestingly the results are very stirring. In the present study the average glycosylated hemoglobin (HbA1c) was significantly higher in obese and morbid obese with respect to overweight (Table 6, $p < 0.001$) and so was the fasting blood sugar level, total cholesterol, LDL cholesterol, VLDL cholesterol and triglycerides levels. On the contrary, HDL cholesterol level and lecithin cholesterol acyl transferase activity (LCAT) were significantly lower. These observations clearly indicated that in these obese subjects, lopsided dyslipidemia also existed. In another exercise, constituent (total cholesterol, phospholipids, triglycerides and apoprotein) of VLDL, LDL and HDL were examined. Lipid fractions were adversely affected in both obese and morbidly obese subjects. The most important features needing focus are low HDL cholesterol, low LCAT levels (Table 2), low HDL apoprotein fraction and low reduced glutathione (GSH), super oxide dismutase(SOD), catalase(CAT), glutathione peroxidase(GPx) and glutathione reductase(GR) (Table 4) (35-38). There is ample evidence that HDL cholesterol is a potent predictor of cardiovascular events, independent of other parameters. The cardio protective effect of HDL is attributed to its role in reverse cholesterol transport. It removes excess cholesterol from peripheral tissues towards the liver for excretion in to bile or else for steroid hormone synthesis in steroidogenic organs. Further effects of HDL are proteotropic as it also exerts as antioxidant and anti-inflammatory agent. Lecithin cholesterol aceyl transferase is a vitally important enzyme helping in the reverse cholesterol transport. It transfers 2 acyl groups of lecithin to cholesterol resulting in the generation of cholesterol esters which are retained in the core of HDL particle for final scavenging. Incidentally, glycosylated Hb negatively correlates with LCAT activity in obesity. Apoprotein-1 is quantitatively a major component of HDL (39). Glycation of apoprotein A-1 in HDL alters and reduces LCAT activity in proportion to the extent of apoprotein A-1 glycation. Indeed there is convincing evidence that obesity induced hyperglycemia impaired several pathways and led to the formation ofmore ROS. These ROS increased the glycation potentialthis clinical study, apoprotein-1 significantly decreased and concomitantly OS also increased. Further more, in both VLDL and LDL components, total cholesterol and triglycerides levels were consistently and considerably higher in obese and morbidly obese subjects indicating dyslipidemia. It is now widely accepted that dyslipidemia is a cardinal feature in obese subjects. Although cells usually exist with reductive environment, but oxidation and reduction reactions are essential and crucial phenomenon for every cell (40). In normal cells, at any given time, such oxidative processes yield reactive oxygen species (ROS), the latter beings lightly more than the reduction processes. This oxidative potential is termed as OS. ROS and antioxidants are the major determinant of oxidative stress (OS) as other cellular oxidative reductive processes are in balance. OS is raised in obese and morbidly obese subjects through numerous pathologies. Most ROS are generated in cells by the mitochondrial respiratory chain. Under normal metabolic conditions, these ROS are eliminated rapidly in normal cells by a wide variety of enzymatic and nonenzymatic antioxidant defences. The imbalance between production of reactive oxygen species (ROS) and their elimination by antioxidant defence system results in oxidative stress (41). Our study indicates the pivotal role of oxidative stress in the pathogenesis and progression of obesity. Although the role of OS in the origin of CAD with T2DM is still controversial issue, it definitely abets T2DM and plays a central role in the development of diabetic complications. One of the major oxidant is super oxide anion, that too with predominance in endothelial cells of both large and small arteries and myocardium. In association with dyslipidemia it increases the risk of cardiovascular events by several folds. It is also postulated that O_2 inactivates 2 critical anti atherosclerotic enzymes. endothelial nitric oxide synthase and prostacyclin Synthase (30). In the present study, LPO, an accepted marker of OS in CAD with T2DM patients was significantly raised. The average increase was more than three folds to that of controls. This clearly alluded and signified to provoke OS in CAD with T2DM patients. Consequently, this must be disturbing the redox box. The raised OS was accompanied with reduction in GSH level and lower SOD, Catalase, GPx and GR activities in obese and morbid obese subjects (42).

CONCLUSION

To evade obesity, obese people should reduce calorie intake, increase physical exercise and prefer fat restricted, fiber rich diet. Blood status of lipotropic factors should be in normal limit.Lipotropic factors are required for the normal mobilization of fat from liver. Therefore, deficiency of these factors may result in fatty liver due to obesity. These are choline, lecithin and methionine. They help in the synthesis of

apoprotein and choline formation. The deficiency on methyl groups for carnitine synthesis may also hinder fatty acid oxidation.Vitamin E and selenium give protection due to their anti-oxidant effect.Omega 3 fatty acids present in marine oils have a protective effect against fatty liver due to obesity.Further, such type of studies will specifically help health workers and clinicians to suggest health and therapeutic regimen in a particular population.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflict of interest.

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How to cite this article : Anwer E., Kumar V., Ahmad S., Mahdi F., Gogoi S., Farheen Z. Association Of Anthropometric Measurements With Impairement Of Blood Biochemistry Analytes In Obese Subjects From Different Socio-economic Status. Era J. Med. Res. 2019; 6(2): 71-77.