

CORRELATION OF BODY FAT DISTRIBUTION WITH PRESENCE OF OXIDATIVE STRESS IN OBESITY

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ABSTRACT

BACKGROUND

Though, there are experimental and clinical evidences on oxidant-antioxidant disturbance in obese patients, data supporting the same in centrally and peripherally obese subjects is lacking.

The aim of the study is to study the effect of body fat distribution on oxidative stress parameters in obesity.

MATERIALS AND METHODS

A prospective randomised study was done on 300 obese subjects and compared with 100 normal subjects having BMI between 19 to 25 kg/m² in the Department of Medicine and Department of Biochemistry, G. R. Medical College, Gwalior. Patients were grouped as case (n=300) and control (n=100). Detailed physical examination and laboratory investigation including Fasting Blood Glucose (FBG) and complete lipid profile was done and recorded for each subject. The waist and hip was measured with inch tape to calculate the waist/hip ratio. Patients were divided on the basis of central and peripheral obesity. Superoxide Dismutase (SOD) activity and plasma Malondialdehyde (MDA) level were also estimated.

RESULTS

Mean age in case and control group was 46.2 ± 2.4 years and 44.5 ± 2.2 years (p>0.05) respectively with male predominance. Majority of subjects were centrally obese (82.33%) in centrally obese patients MDA levels (4.89 ± 1.26 vs. 4.06 ± 1.12 µmol/mL) were higher and SOD levels (8.62 ± 2.23 vs. 9.58 ± 1.26 units/mL) were lower compared to peripheral obesity (p<0.001). Significant (P<0.001) difference was observed for MDA level between case and control in centrally obese (4.89 ± 1.26 vs. 2.06 ± 0.76, respectively) and peripherally obese (4.06 ± 1.12 vs. 2.06 ± 0.76, respectively) subjects. Similarly, SOD level between case and control among centrally obese (8.62 ± 2.23 vs. 12.42 ± 2.18) and peripherally obese (9.58 ± 1.26 vs. 12.42 ± 2.18) subjects was significantly different (P<0.001).

CONCLUSION

The central fat deposition in abdomen or the apple-shaped obesity is associated with higher oxidative stress in obese subjects.

KEYWORDS

Oxidative Stress, Malondialdehyde, SOD, Obesity.

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BACKGROUND

Body fat distribution plays an important role in disease pathophysiology. Central obesity (fat distribution of adipose tissue at abdomen) has been linked with increased risk of Coronary Heart Disease (CAD), type 2 diabetes mellitus, cancer and all-cause mortality.¹ Fat accumulation in hips and thighs (peripheral obesity) has lower pathologic significance.²

Lipid peroxidation is increased and antioxidant enzyme activity is decreased in patients with obesity. Oxidative stress is the imbalance between reactive oxygen species

production and antioxidant enzyme defense, which may result in tissue injury.³

In lipid peroxidation, polyunsaturated fatty acids are deteriorated with the help of oxygen, which forms free radical. Malondialdehyde (MDA) is a biomarker of lipid peroxidation.⁴ In the presence of excess free radicals because of low level of antioxidant enzyme activity risk of coronary artery disease may increase. Superoxide Dismutase (SOD) is the endogenous enzymes, which diminish free radical activity hence protect the body.⁵

In obesity, protective action of SOD is decreased and lipid peroxidation is increased resulting in increased level of MDA.⁶

As the obesity is an independent risk factor for oxidative stress, the present study was done to assess the effect of fat distribution in obese subjects on the oxidative parameters. The MDA and SOD were measured in obese subjects and these parameters were compared between central and peripheral obesity.

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MATERIALS AND METHODS

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Institutional ethics committee approval and written informed consent was obtained from each subjects before starting study.

Subjects having BMI >25 kg/m², age >18 yrs. and subjects willing to give written informed consent were included in the present study. Subjects with any severe or critical illness, pregnant and lactating women and patients with history of smoking, diabetes, hypertension and liver or renal disease were excluded from the present study.

Detailed physical examination including height, weight, hip circumference, waist circumference, waist/hip ratio, BMI and laboratory investigation including Fasting Blood Glucose (FBG) and complete lipid profile, including Total Cholesterol (TC), Triglyceride (TG), Low-Density Lipoprotein Cholesterol (LDL-C), High-Density Lipoprotein Cholesterol (HDL-C) and Very Low-Density Lipoprotein Cholesterol (VLDL-C) was recorded for each subjects. BMI was calculated as weight divided by height squared. The waist and hip was measured with inch tape to calculate the waist/hip ratio. The absolute waist circumference (>102 centimetres (40 inch) in men and >88 centimetres (35 inch) in women) and the waist/hip ratio (>0.9 for men and >0.85 for women) or both were used as measures of central obesity. Central obesity, the "apple-shaped" obesity commonly referred to as belly fat is the accumulation of visceral fat deposited between the internal organs in the torso resulting in an increase in waist size. Peripheral obesity, "pear shaped" is the accumulation of excess fat in the buttocks, hips and thighs.

Venous blood (5 mL) was collected from each subject and used to estimate Superoxide Dismutase (SOD) activity and plasma Malondialdehyde (MDA) level.

All the analysis was done with IMB SPSS ver. 20 software. Results on continuous measurements are presented on mean ± Standard Deviation (SD). Unpaired t-test and Analysis of Variance (ANOVA) with post-hoc Bonferroni and Tukey test was used to find out the significance between two and more than two groups,

respectively. Pearson correlation test was used to find correlation between study parameters. Significance is assessed at 5% level.

RESULTS

Mean age of subjects in case and control group was 46.2 ± 2.4 years and 44.5 ± 2.2 years (p>0.05), respectively. In case and control group, there were 61.33% and 54% males and 38.67% and 46% female, respectively.

Parameters	Case	Control	P value
Weight (kg)	91.57 ± 9.8	61 ± 5.2	<0.001
Height (cm)	161.6 ± 9.3	163.1 ± 8.7	NS
BMI (kg/m ²)	36.17 ± 3.4	21.24 ± 1.88	<0.001
Waist (cm)	114.7 ± 6.2	85.2 ± 1.4	<0.001
Hip (cm)	114.23 ± 17.12	97.32 ± 9.12	<0.001
W/H ratio	0.98 ± 0.22	0.86 ± 0.14	<0.001

Table 1. Anthropometric Measurements of Case and Control Groups

Data is expressed as mean ± SD, P<0.001 is considered to be highly significant, NS- Nonsignificant. BMI- Body mass index, W/H- Waist and hip.

Parameters	Case	Control	P value
FBG (mg/dL)	87.3 ± 2.6	94.4 ± 3.2	NS
TC (mg/dL)	196.0 ± 12.6	186.6 ± 6.9	<0.05
TG (mg/dL)	253.6 ± 27.3	143.4 ± 15.4	<0.001
LDL-C (mg/dL)	135 ± 47.03	95.73 ± 27.48	NS
HDL-C (mg/dL)	47.1 ± 1.2	51.6 ± 1.7	NS
VLDL-C (mg/dL)	45.8 ± 14.03	22.4 ± 10.45	<0.001

Table 2. Biochemical Parameters of Case and Control Groups

Data is expressed as mean ± SD, P<0.001 is considered to be highly significant; NS- Nonsignificant; FBG- Fasting blood glucose; TC- Total cholesterol; TG- Triglyceride; LDL-C- Low-density lipoprotein cholesterol; HDL-C- High-density lipoprotein cholesterol; VLDL-C- Very low-density lipoprotein cholesterol.

Obesity type	MDA Level		P Value	SOD Level		P Value
	Case	Control		Case	Control	
Central	4.89 ± 1.26	2.06 ± 0.76	<0.001	8.62 ± 2.23	12.42 ± 2.18	<0.001
Peripheral	4.06 ± 1.12	2.06 ± 0.76	<0.001	9.58 ± 1.26	12.42 ± 2.18	<0.001

Table 3. Distribution of Patients According to Body Fat Distribution

Data is expressed as mean ± SD, P<0.001 is considered to be highly significant, SOD- Superoxide dismutase, MDA- Malondialdehyde.

Majority of the subjects (247 (82.33%)) had central obesity as compared to peripheral obesity 53 (17.67%).

Mean MDA level in case and control group was 4.68 ± 1.72 µmol/mL and 2.06 ± 0.76 µmol/mL, respectively

(p<0.001). Similarly, mean SOD activity among case and control groups were 7.65±1.13 units/mL and 12.42 ± 2.18 units/mL, respectively (p<0.001).

MDA level was higher in centrally obese patients (4.89 ± 1.26 µmol/mL) as compared to patients with peripheral obesity (4.06 ± 1.12 µmol/mL) (P<0.001). SOD level was lower in centrally obese patients (8.62 ± 2.23 units/mL) as

compared to patients with peripheral obesity (9.58 ± 1.26 units/mL) ($p < 0.001$).

Parameters	Central Obesity	Peripheral Obesity
SOD	n=247, r=-0.043, P<0.001	n=53, r=-0.126, P<0.001
MDA	n=247, r=0.363, P<0.001	n=53, r=0.332, P<0.001

Table 4. Correlation Between Level of MDA and SOD Among Cases

Data is expressed as mean \pm SD, $P < 0.001$ is considered to be highly significant.

DISCUSSION

In India, obesity has become an epidemic. Central obesity is regarded as independent risk factors for diabetes mellitus. Central obesity also results in generation of free radical and depletes intracellular glutathione.⁷

Central or abdominal obesity is a powerful contributor to metabolic abnormalities including insulin resistance, hyperinsulinaemia and decreased insulin receptors. Measurements of central obesity can catch the additional information about the risks, which are not covered by BMI.⁸

The antioxidant defense system helps in maintaining normal cellular physiology prevents diseased condition and also promotes immunity. In present study, central obesity was more common compared to peripheral obesity. Study done by Perticone et al reported higher percentage of centrally obese patients in their study.⁹

In present study, MDA level was higher in centrally obese patients as compared to patients with peripheral obesity ($P < 0.001$), whereas SOD level was lower in centrally obese patients as compared to patients with peripheral obesity ($p < 0.001$). Sabitha et al studied 25 obese and overweight patients to study role of antioxidant enzyme and its association with obesity reported increased MDA levels ($p < 0.001$) in patients with obesity due to lipid peroxidation and SOD activity was decreased ($p < 0.001$), which shows decline of antioxidant defense capacity among obese patients.¹⁰ A study done by Nirmitha et al including 60 obese male patients reported that malondialdehyde levels were significantly high and total antioxidant capacity was significantly decreased in obese patients compared to age and sex matched healthy subjects.¹¹ The consequence of the low activity of cytoprotective enzymes in human obesity is progressive tissue damage, which may eventually lead to atherosclerosis.

There was an inverse linear relationship between central obesity and peripheral obesity with SOD level and linear relationship between central obesity and peripheral obesity with MDA. This means as level of MDA increases and SOD decreases as the central and peripheral obesity of subject is increased.

Vincent et al in their review discussed the data suggesting that obesity in particular central obesity is the independent risk factor for systemic oxidative stress.¹² Present study data is in concordance with the findings of Vincent et al.

CONCLUSION

The central obesity or the visceral fat accumulation in obese subjects poses a greater risk for development of various diseases as it is associated with higher oxidative stress than the peripheral fat accumulation.

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