Original Article

Effect of Obesity on Anti Oxidant Enzymes and Type 2 Diabetes

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ABSTRACT

Introduction: Obesity has become a major health problem in both developed and under-developed countries. Obesity represents an abnormal accumulation of adipose tissue resulting from chronic over nutrition and reduced physical activity. The aim of this study was to determine the effect of obesity on anti-oxidant enzymes and type-2 diabetes.

Aim: The present work aimed at investigating the effect of obesity on type-2 diabetes and anti-oxidant enzyme activity.

Materials and Methods: A total of 50 Subjects (32 males, 18 females) aged > 35 years were selected from Chalmeda Anand Rao Institute of Medical Sciences, Bommakal, Karimnagar. Obesity is determined by the body mass index(BMI). Lipid peroxidation was determined by measurement of thio-barbituric acid reactive substances (TBARS) in plasma of the patients. The anti-oxidant enzymes superoxide dismutase was assayed.

Results: The mean and S.D of Malondialdehyde of controls is 240 + 1.14 as compared to 619 + 0.91 in cases. The mean S.D. of superoxide dismutase of controls 100.2 + .67 and the mean S.D. of cases 50.04+4.69. The mean and S.D. of fasting serum glucose in controls is 88.33+ 6.78 as compared to 146.30 + 11.68 in cases. The mean and S.D of serum total cholesterol, triglycerides, VLDL-C LDL-C in controls are 163.28 + 5.98, 136.88 + 14.62, 26.68 + 2.32, 90.04 + 6.14 respectively as compared to 214.16 + 10.77, 190.56 + 13.77, 38.08 + 2.69, 141.36 + 11.32 in cases. The mean and S.D of serum non HDL-C and TG/HDL-C in controls are 118 + 12.88 and 2.88+ 0.40 respectively as compared to 179.4 + 11.30 and 5.85 + 0.66 respectively in cases. The mean and S.D. of TC/HDL-C in controls is 3.5 + 0.35 as compared to 6.54 + 0.69 in cases.

Conclusion: Our study was concluded that obesity even in the absence of smoking, renal or liver disease can decrease the body's protective anti-oxidants and enhance the systematic oxidative stress.

Keywords: Obesity, antioxidant enzymes, oxidative stress, type 2 diabetes

INTRODUCTION

Obesity is defined as a chronic disease affecting life span and quality in a negative manner.[1] Obesity is a complex disease that involves the interaction between environmental[2] and genetic factors.[3] Most patients with type-2 diabetes are obese and the global epidemic of obesity largely explains the dramatic increase in the incidence and prevalence of Type-2 diabetes over the past 20 years. The prevalence of Type-2 diabetes in India is highest among various Asian population. Number of deaths in adults due to diabetes among all age groups is 6.8% at global level.[4]

Obese adults demonstrate reduced glucose disposal, primarily at the level of skeletal muscle (peripheral insulin resistance), as well as impairment in insulin action on non-esterified fatty acid oxidation leading to insulin resistance and abnormal Lipolysis. [5,6] Obesity is associated with increased lipid peroxidation. [7]

Obesity can independently cause increase lipid peroxidation by progressive and cumulative cell injury resulting from pressure from the large body mass. Cell injury causes the release of cytokines especially TNF-a, which generates reactive oxygen species from the tissues which in turn cause lipid peroxidation.[8] The cell adaptation to the increase of radical production, as a consequence of obesity, consists then in the increase of SOD activity. [9] The present work aimed at investigating

the effect of obesity on type-2 diabetes and anti-oxidant enzyme activity.

MATERIALS AND METHODS

Subjects

The present study was carried out in the Department of Bio-chemistry Chalmead AnandRao Institute of Medical Sciences, Karimnagar. The cases were selected from those attended, the medicine OPD at Chalmeda Medical College and Hospital, Karimnagar. The Investigating were carried out in Bio-Chemistry laboratory Chalmeda Medical College and Hospital, Karimnagar.

Design

The total number of subjects included in the study was 50 and divided into two groups. Group-I consisted 25 normal healthy subjects as controls while Group-II consists patients with obesity as cases. This is a case control study.

Criteria for Selection

Obesity = Obese subjects with BMI in the range of =30 kg/m2 and =39.9kg/m2 BMI was calculated as weight in Kilograms divided by the square if height in meters10. Chronic diseases hepatitis, epilepsy and renal diseases, were included in the exclusion criteria.

Blood Sample Collection

A venous blood sample was obtained from every fasting volunteer into heparinized tubes (BD vacutainer system). All the blood samples were immediately carried to the Biochemistry Laboratory In a crushed ice block, Blood samples were centrifuged at 3000 rpm for 10 minutes. The Lipid profile and FBS were performed in fully automated analyzer (DS-302 vector Biotech); SOD is measured by the Worthington method using inhibiting enzyme by oxygen dependant reaction.[11]

Estimation of Malondialdehyde; The lipid per oxidation product, Malondialdehyde (MDA) in the plasma was estimated by thiobarbituric acid reactive substances (TBARS) by the method of Buege and Aust,1978.[12] MDA reacts with thiobarbituric acid (TBA) to form pink colored adduct, TBA-MDA adduct at 535nm. SOD and MDA were measured spectrophotometrically.

Ethics approval

The study protocol was reviewed and approved by the Institutional Ethics committee, at Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar, 2015, and written informed consent form was obtained from all participants.

RESULTS

The total number of subjects included in the study was 50 and divided into two groups. Group-I consisted 25 normal healthy subjects as controls while Group-II consists patients with obesity as cases.

DISCUSSION

Global statistics indicate that the prevalence of diabetes

Table 1: Comparison of Glucose, Malondialdehyde, Superoxide Dismutase, BMI, Waist circumference In cases and controls

	Fasting Serum Glucose	Post Prandial Glucose	Body Mass Index	Waist Circumferance	Malondiadehyde	Superoxide Dismutase
Controls	88.33±6.78	129.88±6.6	24.068±0.7	87.19±1.5	240.44±1.14	100.2±4.67
Cases	146.307±11.68	183.69±3.8	31.36±2.27	107.69±6.86	619.12±0.9	50.04 ±4.96

Table 1 shows increased Serum Glucose,increased Malondialdehyde decreased Superoxide Dismutase in obesity compared to controls.

Table 2: comparison of lipid profile in cases and controls

	CHO	TG	HDL-C	VLDL-C	LDL=C	TG/HDL-C	NON-HDL-C	TC/HDL-C
Controls	163.28	136.88	46.68	26.68	90.04	2.88	118.2	3.5
	±5.98	±14.62	±4.67	±2.32	±6.14	±0.40	±12.88	±0.33c
Cases	212.16	190.56	32.36	38.08	141.36	5.8532	179.4	6.5416
	± 10.77	± 13.77	± 2.91	± 2.69	± 11.32	± 0.66	± 11.3	± 0.69

Table 2 shows comparison of lipid profile in cases and controls. This shows Dyslipidemia in obesity compared to control group, there is elevated cholesterol, Triglyceride, LDL in obesity and HDC levels are decrease.

would nearly double by the year 2030 [13-14] obesity is increased in south Asian countries, including Nepal and Bangladesh, between 1996 and 2006 (from 1.6% to 10% and from 2.0% to 8.9% respectively). [15-18] Overweight and obese was independently associated with having hypertension, diabetes and raised serum cholestrol concentration, In the present study mean glucose level of the obese group was significantly higher.

Free radicals are very potent oxidants and react with lipids to result in lipid peroxidation.[20,21,22] Possible mechanism contributing to the obesity – associated oxidant stress include increased oxygen consumption and subsequent radical production via mitochondrial respiration, increased fat deposition and cell injury causing increased rates of radical formation which reacts with lipids result in lipid peroxidation. [23] Skrha et.al [24] also found elevated oxidative stress in obese persons. Chronic hyperglycemia in obesity may lead to continuously elevated MDA formation. [25] So, lipid peroxidation product Malondialdehyde levels are increased on the present study compared to control group.

During respiration some electrons released from mitochondrial electron transport chain incompletely reduce O2 to superoxide anion. The superoxide is ROS molecule and must be maintained at nanomolar concentration to avoid cellular damages. [26] The endogenous control of cellular superoxide levels is made by superoxide dismutase (SOD) enzymes. The increase of superoxide levels that potentially occur in obesogenic states did not trigger an increase of SOD enzyme levels obesity causes a chronic low grade inflammation process that is a perpetuating factor of oxidation stress [27] and antioxidant capacity impairment. [28]

Olusi [29] found that SOD activity was lower in very obese. Similarly, Ozata et al.[30] reported 42% lower SOD activity in obese man than in non obese. In the present study also SOD levels was lower obesity compared to normal control group.

Obesity is characterized by increased dietary fat intake, increased fat storage and excessive intracellular triglycerides and dyslipidemia. Dyslipidemic profiles in obesity include elevated triglycerides, lowered high-density lipoproteins (HDL), and elevated LDLs. When the lipid profile of both groups were compared, there were significant increase of cholesterol, TG, VLDL and LDL levels in the obese group.

CONCLUSION

It is concluded that obesity even in the absence of smoking, renal (or) liver disease can decrease the body's protective antioxidants and enhance the systemic oxidative stress. So SOD activity is decreased and MDA levels are increase. Central obesity and Diabetes are constant observations in the present study. Simple and economical measures like Life Style Modifications (LSM) and lipid lowering therapy may reduce BMI. A focus on Diabetes Mellitus and Obesity will encourage public health efforts to give more priority to the promotion of weight control and physical activities in their societies.

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CONFLICT OF INTEREST

The authors declared no conflict of interest.

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