

ORIGINAL RESEARCH

Serum paraoxonase enzyme activity in obese individuals

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ABSTRACT

Background: Recently, there has been a lot of focus on the connection between serum paraoxonase enzyme activity and dyslipidemia in obese people, highlighting the interdependence of lipid profiles and enzymatic performance in the context of obesity. The present study was conducted to assess serum paraoxonase enzyme activity in obese individuals. **Materials & Methods:** 135 obese subjects of both genders were enrolled. Serum paraoxonase activity was measured using a spectrophotometric enzymatic method. **Results:** Out of 135 patients, 70 were males and 65 were females. The mean BMI was 32.6 kg/m² in males and 31.4 kg/m² in females. The mean age was 44.3 years in males and 45.1 years in females. The mean serum paraoxonase activity was 64.9 U/L in males and 62.4 U/L in females. The difference was significant (P < 0.05). There were 32% male and 16% female drinkers and 68% male and 84% female non-drinkers. There were 33% male and 24% female smokers and 67% male and 76% female non-smokers. There Physical activity was sedentary in 52% male and 68% females and active 48% male and 32% females. The difference was significant (P < 0.05). **Conclusion:** It was discovered that obese people as compared to non-obese have decreased serum paraoxonase enzyme activity.

Keywords: Enzyme, obese, serum paraoxonase

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INTRODUCTION

Recently, there has been a lot of focus on the connection between serum paraoxonase enzyme activity and dyslipidemia in obese people, highlighting the interdependence of lipid profiles and enzymatic performance in the context of obesity. An HDL-associated enzyme called paraoxonase 1 (PON1) guards against atherosclerosis and oxidative stress.¹ The risk of cardiovascular illnesses, which are associated with obesity and dyslipidemia, is inversely connected with its activity. Many people who are overweight suffer from dyslipidemia, a metabolic disease marked by high levels of bad cholesterol (LDL), triglycerides, total cholesterol (HDL), and low levels of good cholesterol (HDL).² Atherosclerosis and other cardiovascular diseases (CVDs) are accelerated as a result. The results imply that because obese people have lower PON1 activity, they may be more vulnerable to cardiovascular problems and dyslipidemia.³ Obesity is linked to decreased PON1 activity because it is believed that oxidative stress and inflammatory conditions caused by excessive adiposity have a significant effect on the PON1 enzyme. Additionally, research has linked

PON1 activity to lipid profiles, indicating that when PON1 activity is low, atherogenic profiles are made worse by detrimental lipid changes like elevated LDL and lower HDL levels.⁴ The relationship between serum paraoxonase enzyme activity and dyslipidemia in obese individuals highlights the significance of enzymatic activity in regulating lipid metabolism and averting cardiovascular disorders. Additional study in this area could yield useful information.⁵ The present study was conducted to assess serum paraoxonase enzyme activity in obese individuals.

MATERIALS & METHODS

The present study was conducted on 135 obese subjects of both genders. All were informed regarding the study and their written consent was obtained.

Data such as name, age, gender etc. was recorded. Lifestyle choices, BMI, and the blood paraoxonase enzyme activity level (U/L) were noted. All participants had fasting blood samples taken for biochemical analysis at the start and finish of the research period. Automated enzymatic assays were used to evaluate lipid profiles, and a

spectrophotometric enzymatic technique was used to measure serum paraoxonase activity. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 135		
Gender	Male	Female
Number	70	65

Table I shows that out of 135 patients, 70 were males and 65 were females.

Table II Assessment of parameters

Parameters	Male	Female	P value
BMI (kg/m ²)	32.6	31.4	0.94
Mean age (years)	44.3	45.1	0.67
Serum Paraoxonase Activity (U/L)	64.9	62.4	0.75

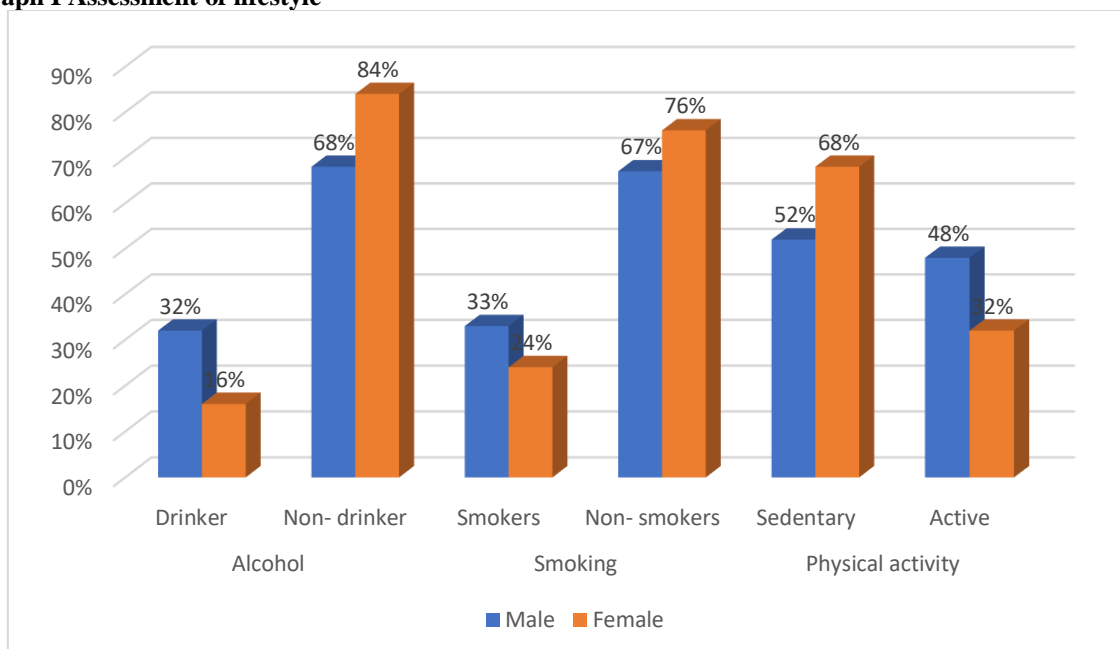
Table II shows that mean BMI was 32.6 kg/m² in males and 31.4 kg/m² in females. The mean age was 44.3 years in males and 45.1 years in females. The mean serum paraoxonase activity was 64.9 U/L in males and 62.4 U/L in females. The difference was significant (P< 0.05).

Table III Assessment of lifestyle

Parameters	Variables	Male	Female	P value
Alcohol	Drinker	32%	16%	0.01
	Non- drinker	68%	84%	
Smoking	Smokers	33%	24%	0.05
	Non- smokers	67%	76%	
Physical activity	Sedentary	52%	68%	0.74
	Active	48%	32%	

Table III, graph I shows that there were 32% male and 16% female drinkers and 68% male and 84% female non- drinkers. There were 33% male and 24% female smokers and 67% male and 76% female non-smokers. There Physical activity was sedentary in 52% male and 68% females and active 48% male and 32% females. The difference was significant (P< 0.05).

Graph I Assessment of lifestyle



DISCUSSION

Numerous changes in lipid metabolism linked to obesity result in variations in the quantity and makeup of lipoproteins.⁶ Obese people had greater than three

relative risk factors for diabetes, hypertension, dyslipidemia, insulin resistance, dyspnea, and apnea.⁷ Numerous studies have shown that obese patients experience higher levels of oxidative stress, and that

their isolated LDL is more susceptible to lipid peroxidation than that of healthy subjects.^{8,9}The present study was conducted to assess serum paraoxonase enzyme activity in obese individuals.

We found that out of 135 patients, 70 were males and 65 were females. Aslan et al¹⁰evaluated PON1 and arylesterase enzyme activities and lipid hydroperoxide (LOOH) levels, and to investigate whether there is increased susceptibility to atherogenesis in obese subjects, which might be reflected by increased oxidative stress and decreased PON1 activity. We also aimed to investigate the association between PON1 activity and body mass index (BMI) in this patient group. The study involved 25 obese subjects and 23 controls. Serum PON1 and arylesterase activity was measured spectrophotometrically. LOOH levels were measured by the FOX-2 assay. Serum basal/salt-stimulated PON1 and arylesterase activities were significantly lower in obese subjects than in controls ($P < 0.001$ for both enzymes), while LOOH levels were significantly higher ($P < 0.001$). BMI was significantly correlated with PON1, arylesterase and LOOH levels ($P < 0.001$, $r = -0.720$; $P < 0.001$, $r = -0.634$; $P < 0.001$, $r = 0.491$; respectively). Serum high-density lipoprotein (HDL) levels were positively correlated with PON1 activity ($r = 0.347$, $P < 0.05$).

We found that mean BMI was 32.6 kg/m² in males and 31.4 kg/m² in females. The mean age was 44.3 years in males and 45.1 years in females. The mean serum paraoxonase activity was 64.9 U/L in males and 62.4 U/L in females. In atherosclerotic patients, Rosenblat et al¹¹ examined the distribution of serum paraoxonase 1 (PON1) between HDL and lipoprotein-deficient serum (LPDS) and contrasted the biological roles of PON1 in these fractions. Serum HDL and LPDS fractions were extracted from individuals with hypercholesterolemia, diabetes, and healthy controls. Measurements were made of PON1 protein and activity in HDL/LPDS, as well as its capacity to prevent lipid peroxidation and promote HDL/LPDS-mediated macrophage cholesterol export. PON1 protein and a notable paraoxonase activity were detected in LPDS from controls, although arylesterase and lactonase activities were much lower than HDL, by 78% and 88%, respectively. When compared to HDL from controls, PON1 protein and paraoxonase activity in HDL were dramatically reduced in diabetes patients by 2.8 and 1.7 times, respectively. Concurrently, as compared to the LPDS of controls, the PON1 protein and paraoxonase activity in these patients' LPDS were significantly elevated by 3.7 and 1.7 times, respectively. PON1 in HDL (but not PON1 in LPDS) markedly enhanced macrophage cholesterol efflux by 31% and reduced AAPH-induced lipid peroxide production by 33%.

We found that there were 32% male and 16% female drinkers and 68% male and 84% female non- drinkers. There were 33% male and 24% female smokers and 67% male and 76% female non- smokers. There Physical activity was sedentary in 52% male and 68%

females and active 48% male and 32% females. Audikovszky et al¹²assessed the effects of orlistat therapy combined with diet on body mass index (BMI), waist circumference, lipid parameters, blood pressure, serum glucose level and PON1 activity. 139 otherwise healthy, obese subjects were divided in to two groups: 78 persons received orlistat (120 mg three times a day) combined with diet while 61 persons were kept on diet only. Anthropometrical parameters, serum lipid levels and PON1 activity were measured at baseline and after 6 months of treatment. BMI and waist circumference were reduced more pronouncedly in the orlistat group than in the control group. Patients receiving orlistat also had significantly greater improvements in fasting blood glucose levels and blood pressure. The orlistat-treated group showed a greater reduction in total cholesterol and triglyceride levels. In addition, the serum PON1 activity in these patients was significantly increased compared to the diet-only group.

The shortcoming of the study is small sample size.

CONCLUSION

It was discovered that obese people as compared to non- obese have decreased serum paraoxonase enzyme activity.

REFERENCES

1. Mackness MI, Harty D, Bhatnagar D, Winocour PH, Arrol S, Ishola M, et al. Serum paraoxonase activity in familial hypercholesterolaemia and insulin-dependent diabetes mellitus. *Atherosclerosis* 1991;86:193-9.
2. Sentí M, Tomás M, Fitó M, Weinbrenner T, Covas MI, Sala J, et al. Antioxidant paraoxonase 1 activity in the metabolic syndrome. *J Clin Endocrinol Metab* 2003;88:5422-6.
3. Van Gaal LF, Vertommen J, De Leeuw IH. The in vitro oxidizability of lipoprotein particals in obese and non-obese subjects. *Atherosclerosis* 1998;137 (suppl):S39-44.
4. Tack CJ, Smits P, Demacker PN, Stalenhoef AF. Troglitazone decreases the proportion of small dense LDL and increases the resistance of LDL to oxidation in obese subjects. *Diabetes Care* 1998;21:796-9.
5. Prazny M, Skrha J, Hilgertova J. Plasma malondialdehyde and obesity: Is there a relationship? *Clin Chem Lab Med* 1999;37:1129-30.
6. Halliwell B, Gutteridge JMC, Cross CE. Free radicals, antioxidants, and human disease: Where are we now? *J Lab Clin Med* 1992;119:598-620.
7. Maniscalco M, de Laurentiis G, Zedda A, Faraone S, Giardiello C, Cristiano S, et al. Exhaled nitric oxide in severe obesity: Effect of weight loss. *Respir Physiol Neurobiol* 2007;156:370-3.
8. Mather KJ, Lteif A, Steinberg HO, Baron AD. Interactions between endothelin and nitric oxide in the regulation of vascular tone in obesity and diabetes. *Diabetes* 2004;53:2060-6.
9. Gruber HJ, Mayer C, Mangge H, Fauler G, Grandits N, Wilders-Truschnig M. Obesity reduces the bioavailability of nitric oxide in juveniles. *Int J Obes (Lond)* 2008;32:826-31.

10. Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. *Am J Cardiol* 1999;83:25F-9.
11. Rosenblat M, Karry R, Aviram M. Paraoxonase 1 (PON1) is a more potent antioxidant and stimulant of macrophage cholesterol efflux, when present in HDL than in lipoprotein-deficient serum: relevance to diabetes. *Atherosclerosis*. 2006 Jul 1;187(1):74-e1.
12. Audikovszky M, Pados G, Seres I, Harangi M, Fülöp P, Katona E, Illyés L, Winkler G, Katona ÉM, Paragh G. Orlistat increases serum paraoxonase activity in obese patients. *Nutrition, metabolism and cardiovascular diseases*. 2007 May 1;17(4):268-73.
13. Nainani P, Singh HP, Paliwal A, Nagpal N. A rare case report of clear cell variant of oral squamous cell carcinoma. *J Clin Diagn Res*. 2014 Dec;8(12):QD07-9. doi: 10.7860/JCDR/2014/11536.5339.
14. Singh HP, Yadav M, Nayar A, Verma C, Aggarwal P, Bains SK. Ameloblastomatous calcifying ghost cell odontogenic cyst - a rare variant of a rare entity. *Ann Stomatol (Roma)*. 2013 Mar 20;4(1):156-60. doi: 10.11138/ads.0156.
15. Singh HP, Kumar P, Goel R, Kumar A. Sex hormones in head and neck cancer: Current knowledge and perspectives. *Clin Cancer Investig J*. 2012;1(1):2-5. <https://doi.org/10.4103/2278-0513.95011>.
16. Sharma A, Singh HP, Gupta AA, Garg P, Moon NJ, Chavan R. Granulocytic sarcoma in non-leukaemic child involving maxillary sinus with long term follow up: A rare case report. *Ann Maxillofac Surg* 2014;4:90-5.
17. Puri N, Rathore A, Dharmdeep G, Vairagare S, Prasad BR, Priyadarshini R, et al. A clinical study on comparative evaluation of the effectiveness of carbamazepine and combination of carbamazepine with baclofen or capsaicin in the management of Trigeminal Neuralgia. *Niger J Surg* 2018;24:95-9.
18. Singh HP, Yadav M, Nayar A, Verma C, Aggarwal P, Bains SK. Ameloblastomatous calcifying ghost cell odontogenic cyst - a rare variant of a rare entity. *Annali di Stomatologia* 2013; IV (1): 156-160