Relationship between blood peroxidases activity and visfatin levels in metabolic syndrome patients

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Original Article

Abstract

BACKGROUND: The observed relationships between visfatin, peroxidases activity, and metabolic syndrome (MetS) are inconsistent; therefore, this study was undertaken to understand these relationships.

METHODS: This cross-sectional study was conducted as a part of the Isfahan Healthy Heart Program, Iran. A blood sample of 90 MetS and non-MetS patients were used to estimate total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), triglycerides (TGs), fasting blood glucose (FBG), waist circumference (WC), systolic blood pressure (SBP) and diastolic blood pressure (DBP), visfatin and peroxidases activity. Data analysis for MetS group was carried out in two ways. (1) MetS with three components and with > 3 components. (2) MetS with hyperglycemia and without hyperglycemia.

RESULTS: SBP, DBP, WC, FBG, TC, TG, LDL-C, and were higher and HDL-C levels was lower in MetS patients. There was a significant correlation between visfatin levels and peroxidases activity in MetS patients with three components. Levels of visfatin were significantly higher in male as compared to female subjects in the MetS with three components group. There was a significant decrease in peroxidases activity in > 45 years old subjects in the MetS with > 3 components group. A significant correlation was observed between serum visfatin levels and FBG in the MetS without hyperglycemia group.

CONCLUSION: Peroxidases activities in MetS patients can be related to visfatin levels. Gender influences on peroxidases activity probably and was lower in female patients with MetS. Hyperglycemia does not influence peroxidases activities and visfatin levels.

Keywords: Peroxidase, Metabolic Syndrome, Visfatin

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Introduction

The prevalence of metabolic syndrome (MetS) has increased in recent decades,[1] and has been described as a cluster of multiple, partially or fully expressed, metabolic abnormalities within the single individual that increase the risk of developing cardiovascular disease and diabetes.[2,3] In recent years, there has been much interest in the role of free radicals and oxidative stress in the pathogenesis of MetS.[4] It has been shown that obesity per se may induce systemic oxidative stress and that increased oxidative stress in accumulated fat is, at least in part, the underlying cause of the dysregulation of adipocytokines and the development of MetS.[5] Adipocytokines include adiponectin, leptin, resistin, and visfatin that are secreted from adipose tissue.[6]

In human pulmonary vascular endothelial cells, visfatin was demonstrated to interact with several proteins mediating oxidative stress and inflammation leading to increased levels of reactive oxygen species.[7] Oxidative stress may be defined as

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an imbalance between the production and degradation of reactive oxygen species. Enzymatic inactivation of reactive oxygen species is achieved mainly by antioxidative enzymes.[8] The main antioxidant enzymes are glutathione peroxidase (GPx), superoxide dismutase, catalase, and myeloperoxidase.[9] Peroxidases are a family of widespread enzymes which perform distinct tasks. On one hand, they act as preventive antioxidants to detoxify damaging lipid peroxides or other peroxides from blood and organic substrates. On the other hand, these enzymes function as starters for oxidative reactions, thereby generating a source of reactive oxygen species such as hypochlorous acid (HOCl) or hypoiodous acids (HOI).[10]

Increasing the visfatin levels can be observed in atherosclerosis,[11] endothelial dysfunctions,[12,13] and renal insufficiency.[14] Evidence on possible associations between serum visfatin and metabolic parameters in patients with obesity and diabetes are contradictory.[14-18] Takebayashi et al.[14] did not find any correlation between visfatin and diabetes, and other study proved that there is a positive correlation between the decrease of visfatin and type 1 diabetes and negative correlation between glycated hemoglobin and visfatin levels.[19] Berndt et al.[16] and Hammarstedt et al.[20] have reported that serum concentration of visfatin is increased in obesity. On the other hand, Pagano et al.[21] revealed that plasma visfatin was significantly lower in obese subjects.

The effect of hyperglycemia on levels of visfatin is discussed. Alexiadou et al.[22] finding was discordance with previous study[23] demonstrating that visfatin is enhanced by hyperglycemia.

The visfatin levels and peroxidases activity are important in MetS, whereas only very few studies have been conducted to clarify the relationships between visfatin and peroxidases, and these factors and MetS. Therefore, the present study was designed to understand these relationships.

Materials and Methods

Participants

This cross-sectional study was conducted in 2012 as a part of the Isfahan Healthy Heart Program, Iran, (IHHP). IHHP began in 2000 to prevent and control cardiovascular disease risk factors in the Iranian population. This program was conducted in Central Iran. A stratified multi-stage probability sampling method was used in the baseline survey (2001) and the post-intervention in 2007.[24]

Blood samples (from 90 subjects within the age range of 19-82 years) of IHHP third phase (2006-2007) were used for this study. Samples of subjects with MetS (n = 45) and also without MetS (n = 45), were selected using simple random sampling. MetS defined by the National Cholesterol Education Program Adult Treatment Panel III as the presence of 3 or more of the following criteria: abdominal obesity: waist circumference (WC) ≥ 102 cm in men and ≥ 88 cm in women and 2 or more of the following: systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic blood pressure (DBP) ≥ 85 mmHg; triglyceride (TG) ≥ 150 mg/dl; high-density lipoprotein cholesterol (HDL-C) < 40 mg/dl in men and < 50 mg/dl in women; fasting blood glucose (FBG) ≥ 110 mg/dl.[10] The study protocol was approved by the Medical Ethics Committees of the Isfahan Cardiovascular Research Institute under the Approval No. 91115.

Biochemical and anthropometric measurement

All measurements were conducted using calibrated instruments and standard protocols by a trained team of general physicians and nurses. Blood samples were collected from both groups to measure the biochemical factors following a 12-h fasting. All the blood sampling procedures were performed in the central laboratory of the Isfahan Cardiovascular Research Institute. FBG and serum lipids, including serum total cholesterol (TC), TG and HDL-C levels were detected by an enzymatic method using an Elan 2000 auto analyzer (Ependorf, Hamburg, Germany). Low-density lipoprotein-cholesterol (LDL-C) was calculated (in serum samples with TG ≤ 400 mg/dl) according to the Friedewald formula.[25]

The separated serum was stored at −70 °C until the measurement of visfatin levels. Visfatin levels were assayed by ELISA kit (BioVendor Laboratory Medicine Inc., Canada and Mexico, USA). Determination of peroxidases activity in the serum was done by the reaction of endogenous peroxidases with hydrogen peroxide, using 3,5,3',5'-tetramethylbenzidine as the chromogenic substrate.[26] A mercury sphygmomanometer with a cuff size suitable for each subject was used for measuring sitting blood pressure twice from the right arm according to World Health Organization criteria. The mean of two measurements of korotkoff phase I and phase IV was recorded for SBP and DBP, respectively. WC was determined from the point halfway between the lower border of ribs and the iliac crest in a horizontal plane.[27]

Statistical analysis

Statistical analyses were performed using SPSS for

Windows (version 15; SPSS Inc., Chicago, IL, USA). Data were presented as means ± standard deviation. Data analysis for MetS group was carried out in two ways: (1) MetS with three components and with > 3 components. (2) MetS with hyperglycemia and without hyperglycemia. Analysis of covariance was used to compare factors between groups with adjusting age and sex. Also for significant differences, Bonferroni multiple comparison was applied. For comparing visfatin levels and peroxidases activity in groups based on age and sex (without adjustment) Kruskal-Wallis test was used. Mann-Whitney tests with Bonferroni adjustment used to multiple comparisons. Investigation of correlation between visfatin levels and peroxidases activity and also between these two factors with parameters of MetS was assessed using spearman and partial correlation. P-value of < 0.05 was considered to be statistically significant.

Results

Findings on the values of the biochemical factors are summarized in table 1. The MetS subjects had significantly higher values of SBP, DBP, WC, FBG, TC, TG, LDL-C, and lower levels of HDL-C than the non-MetS subjects. There was no significant difference in serum levels of visfatin and peroxidases activity between MetS and non-MetS groups.

Table 2 shows the correlation between visfatin levels and peroxidases activity in the studied groups. There was a significant correlation between visfatin levels and peroxidases activity in MetS subjects with three components whilst non-significant correlation was observed between these factors in the other groups (non-MetS, MetS with > 3 components, and MetS with and without hyperglycemia).

Changes in visfatin levels and peroxidases activity based on sex and age were compared between non-MetS, MetS with three components, and MetS with > 3 components (Table 3) and between non-MetS, MetS with hyperglycemia and MetS without hyperglycemia (Table 4). There was no significant difference in serum visfatin levels between MetS and non-MetS groups in sex and age groups. For peroxidases activity, there was significant difference between non-MetS and MetS with three components groups in female subjects and also between MetS with three components and with > 3 components groups in 19-44 years old subjects. Peroxidases activity did not significantly changed between non-MetS, MetS with

Table 1. Demographic, clinical, and laboratory variables adjusted for age and sex in patient with and without metabolic syndrome

Characteristics	Non-MetS (n = 45)	MetS with three components (n = 29)	MetS with > 3 components (n = 16)	P	Non-MetS (n = 45)	MetS with hyperglycemia (n = 17)	MetS without hyperglycemia (n = 28)	P
Visfatin (mg/ml)	2.83 ± 3.43	3.02 ± 3.27	3.17 ± 3.47	0.944	2.83 ± 3.43	3.07 ± 2.10	3.07 ± 2.61	0.464
Peroxidase activity (mU/ml)	19.23 ± 30.03	3.76 ± 27.91	7.77 ± 30.03	0.050	20.03 ± 30.16	8.17 ± 18.42	8.17 ± 22.93	0.191
Total cholesterol (mg/dl)	180.57 ± 34.72**	207.08 ± 32.28*	195.87 ± 34.74	0.006	179.98 ± 34.59**	209.20 ± 33.22*	200.00 ± 33.12	0.007
Triglycerides (mg/dl)	100.19 ± 95.92$^{**,€}$	160.08 ± 89.27$^{*,€}$	261.18 ± 96.01*,**	0.001	106.20 ± 10.17$^{**,€}$	219.00 ± 9.79*	174.74 ± 9.74*	0.001
High-density lipoprotein cholesterol (mg/dl)	46.97 ± 9.74$^{€}$	41.86 ± 9.01$^{€}$	33.07 ± 9.75*,**	0.001	46.49 ± 101.35$^{€}$	40.83 ± 97.49	38.00 ± 97.15*	0.004
low-density lipoprotein cholesterol (mg/dl)	113.39 ± 29.41**	132.54 ± 27.40*	113.54 ± 29.44	0.008	112.31 ± 30.16	127.33 ± 20.03*	126.08 ± 28.89*	0.049
Fasting blood glucose (mg/dl)	84.21 ± 18.98$^{€}$	91.83 ± 17.63	104.64 ± 19.00*	0.003	85.09 ± 16.04**	112.81 ± 15.42$^{*,€}$	85.16 ± 15.36*,**	0.001
Systolic blood pressure (mmHg)	114.12 ± 17.86$^{**,€}$	125.48 ± 16.31*	130.42 ± 17.39*	0.005	114.42 ± 17.86$^{**,€}$	127.66 ± 12.56*	126.70 ± 16.59*	0.007
Diastolic blood pressure (mmHg)	73.32 ± 11.36$^{*,€}$	82.80 ± 10.39*	85.63 ± 11.07*	0.001	73.50 ± 11.30$^{**,€}$	85.18 ± 10.82*	82.82 ± 10.52*	0.001
Waist circumference (cm)	88.37 ± 11.86**	97.00 ± 10.85*	92.93 ± 11.45	0.009	88.12 ± 11.92**	96.68 ± 11.22*	95.05 ± 11.02	0.016

The results are expressed as mean values ± standard deviation (SD); P-values are significant P < 0.05; * Significant difference with non-MetS; ** Significant difference with MetS with three components or with hyperglycemia; € Significant difference with MetS with > 3 components or without hyperglycemia; Analysis of covariance was used; MetS: Metabolic syndrome hyperglycemia and MetS without hyperglycemia in sex and age groups. Levels of visfatin were significantly higher in male subjects than female in the MetS with three components. There was significant reduction in peroxidases activity in > 45 years old subjects in comparison with 19-44 years old subjects in the MetS with > 3 components.

Correlation between visfatin levels and peroxidases activity with components of MetS are provided in table 5. There was no significant correlation between serum visfatin levels and lipid profile, FBG, SBP, DBP, and WC in MetS and non-MetS groups except visfatin and FBG in the MetS without hyperglycemia subjects. No statistically significant correlation was found between peroxidases activity and studied factors.

Table 2. Correlation between visfatin levels and peroxidase activity in two non-metabolic syndrome and metabolic groups

Biochemical factors	Groups	Spearman's correlation with visfatin levels (mg/ml)	P
Peroxidase activity (mU/ml)	Non-MetS (n = 45)	0.094	0.581
	MetS with three components (n = 29)	0.769	0.001
	MetS with > 3 components (n = 16)	0.315	0.253
	MetS with hyperglycemia (n = 17)	−0.244	0.328
	MetS without hyperglycemia (n = 28)	0.189	0.345

P-values are significant P < 0.05; Spearman correlation was used; MetS: Metabolic syndrome

Table 3. Visfatin levels and glutathione peroxidase activity based on sex and age in non-metabolic syndrome and metabolic syndrome (with three components, with > 3 components of metabolic syndrome) groups

Variable	Non-metabolic syndrome (n = 45)	Metabolic syndrome with three components (n = 29)	Metabolic syndrome with > 3 components (n = 16)	P
Visfatin (mg/ml)				
Sex				
Female (n = 49)	2.80 ± 3.51	1.70 ± 1.10	3.14 ± 2.41	0.206
Male (n = 41)	2.85 ± 2.17	4.33 ± 5.12	3.00 ± 1.76	0.841
P	0.435	0.022	0.842	
Age				
19-45 year (n = 56)	2.93 ± 3.29	3.11 ± 4.52	2.80 ± 2.94	0.963
> 45 year (n = 34)	2.40 ± 2.09	2.73 ± 2.84	3.16 ± 1.58	0.277
P	0.716	0.968	0.389	
Peroxidase activity (mU/ml)				
Sex				
Female (n = 49)	20.46 ± 35.35**	1.64 ± 1.53*	12.57 ± 28.89	0.043
Male (n = 41)	23.71 ± 41.90	4.21 ± 6.80	14.23 ± 30.76	0.376
P	0.314	0.060	0.272	
Age				
19-45 year (n = 56)	22.91 ± 37.80	3.72 ± 6.58 £	43.06 ± 45.31**	0.022
> 45 year (n = 34)	16.62 ± 37.38	1.90 ± 1.24	2.03 ± 1.67	0.102
P	0.598	0.853	0.005	

The results are expressed as mean values ± standard deviation (SD); P-values are significant P < 0.05; Kruskal-Wallis test and Mann-Whitney tests (for multiple comparison) was used; * Significant difference with non-MetS; ** Significant difference with MetS with three components or with hyperglycemia; £ Significant difference with MetS with > 3 components or without hyperglycemia

Table 4. Visfatin levels and glutathione peroxidase activity based on sex and age in non-metabolic syndrome and metabolic syndrome (with hyperglycemia and without hyperglycemia) groups

Variable	Non metabolic syndrome (n = 45)	Metabolic syndrome with hyperglycemia (n = 17)	Metabolic syndrome without hyperglycemia (n = 28)	P
Visfatin (mg/ml)				
Sex				
Female	2.80 ± 3.51	2.00 ± 1.27	2.25 ± 2.05	0.838
Male	2.85 ± 2.17	2.50 ± 1.60	4.41 ± 4.79	0.778
P	0.435	0.489	0.065	
Age				
19-44 years	2.93 ± 3.39	1.66 ± 1.32	4.00 ± 5.16	0.103
> 45 years	2.40 ± 2.09	2.63 ± 2.36	3.12 ± 2.84	0.399
P	0.716	0.078	0.804	
Peroxidase activity (mU/ml)				
Sex				
Female	20.46 ± 35.35	4.83 ± 15.65	8.87 ± 22.12	0.052
Male	23.71 ± 41.90	1.79 ± 1.64	9.28 ± 23.78	0.581
P	0.314	0.077	0.108	
Age				
19-44 years	22.91 ± 37.80	5.38 ± 8.67	16.89 ± 32.63	0.514
> 45 years	16.62 ± 37.38	1.95 ± 1.63	1.97 ± 1.34	0.052
P	0.598	0.394	0.333	

The results are expressed as mean values ± standard deviation (SD); P-values are significant P < 0.05; Kreskas-Wallis test and Mann-Whitney tests (for multiple comparison) was used

Table 5. Correlation of visfatin levels and glutathione peroxidase activity adjusted for age and sex with parameters of metabolic syndrome

Characteristic	TC (mg/dl)	TG (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	FBG (mg/dl)	SBP (mmHg)	DBP (mmHg)	WC (cm)
Visfatin (mg/ml)								
Non-MetS (n = 45)	−0.078	−0.056	−0.198	−0.013	0.036	−0.027	0.054	0.094
P	0.648	0.740	0.240	0.937	0.832	0.879	0.759	0.596
MetS with three components (n = 29)	0.193	0.291	−0.034	0.046	−0.348	0.134	0.058	0.257
P	0.308	0.118	0.857	0.809	0.060	0.489	0.766	0.186
MetS with > 3 components (n = 16)	−0.321	0.198	0.054	−0. 007	−0.100	0.284	0.448	−0.231
P	0.458	0.480	0.848	0.979	0.722	0.305	0.094	0.408
MetS with hyperglycemia (n = 17)	0.286	0.386	−0.144	−0.002	0.151	0.221	0.338	0.373
P	0.250	0.113	0.570	0.994	0.549	0.395	0.185	0.186
MetS without hyperglycemia (n = 28)	0.175	0.327	−0.010	−0.015	−0.512	0.145	0.065	−0.032
P	0.382	0.096	0.960	0.939	0.006	0.469	0.749	0.876
Peroxidase activity (mU/ml)								
Non-MetS (n = 45)	−0.117	−0.206	−0.184	−0.033	−0.220	−0.183	−0.256	−0.130
P	0.491	0.221	0.277	0.847	0.190	0.292	0.138	0.464
MetS with three components (n = 29)	−0.093	−0.093	0.084	−0.086	−0.302	0.058	−0.002	0.024
P	0.617	0.618	0.655	0.646	0.099	0.759	0.991	0.903
MetS with > 3 components (n = 16)	−0.321	−0.313	−0.070	0.012	0.164	0.168	0.106	−0.037
P	0.225	0.238	0.796	0.966	0.543	0.535	0.696	0.890
MetS with hyperglycemia (n = 17)	−0.291	−0.363	−0.007	0.069	0.169	−0.082	−0.367	−0.117
P	0.241	0.138	0.980	0.784	0.503	0.755	0.147	0.653
MetS without hyperglycemia (n = 28)	−0.113	0.071	−0.204	−0.114	−0.247	−0.183	−0.256	0.062
P	0.560	0.715	0.289	0.555	0.197	0.292	0.138	0.755

P-values are significant P < 0.05; Partial correlation was used; MetS: Metabolic syndrome; TC: Total cholesterol; TG: Triglycerides; HDL: High-density lipoprotein cholesterol; LDL: Low-density lipoprotein cholesterol; FBG: Fasting blood glucose; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; WC: Waist circumference

Discussion

The findings of the current study provide evidence-based information about the impacts of visfatin levels and peroxidases activity on parameters of MetS. There was positive correlation between visfatin levels and peroxidases activity in MetS subjects with three components.

Peroxidases activity was higher in MetS with three components than the non-MetS in people female subjects. Peroxidases activity reduced with increasing age in the MetS with > 3 components group and visfatin levels enhanced in male subjects in the MetS with three components group.

Activities of antioxidant enzymes protect against oxidative stress in MetS.[28] Oxidative stress is associated with many of the components of the syndrome, leading to the concept that the amelioration of risk factors comprising MetS, including insulin resistance, elevated blood pressure, elevated lipid levels, inflammation and endothelial dysfunction may ameliorate oxidative stress and thus curtail the progression of metabolic disease complications.[29]

The results by Vavrova et al.[28] implicated an increased oxidative stress in MetS and a decreased antioxidative defense that correlated with some laboratory (TG, HDL-C) and clinical (WC, BP) components of MetS.

Here, we showed a higher serum visfatin levels in patients with MetS however this elevation was no significant. Consistent with our findings, studies have shown elevated serum visfatin levels patients with MetS when compared to individuals without MetS.[30-32] Primary investigation on visfatin showed the insulin-mimicking effect of this hormone.[33] Hence, one would conclude that an elevated visfatin levels in patients with MetS is due to insulin resistance. Cekmez et al.[34] suggested visfatin as a marker of insulin resistance. Inconsistently, Esteghamati et al.[32] showed a higher visfatin concentration, independent of insulin resistance, in type 2 diabetes. Furthermore in two other separate studies, Berndt et al.[16] and Haider et al.[35] showed that visfatin levels were not correlated with insulin resistance and lipid parameters in patients with type 2 diabetes and obesity. A study by Kaminska et al.[36] revealed elevated levels of visfatin in obese subjects did not correlate with the majority of anthropometric parameters. They suggested that elevated visfatin levels are associated with the distribution of adipose tissue characteristic of gynoid rather than visceral obesity.

Yen et al.[37] reported, the subjects suffering from MetS might be under higher oxidative stress, resulting in low levels of antioxidant enzyme activities. MetS is a type of metabolic disorder rather than a disease. Subjects with MetS might be under higher oxidative stress; antioxidant enzymes are the first line of defense against reactive oxygen species and may decrease to adjust to higher levels of oxidative stress.[38] In addition, MetS subjects in general were typically abdominally obese. Obesity is also an oxidative burden that may lead to the reduction of antioxidant enzymes activities.[39] Oxidative stress associate with advancing age.[40] Therefore, the findings of our study confirm the previous study's results.

Mecocci et al.[41] concluded that senescence seems be associated with a decline in nutritional antioxidants together with an increase in antioxidant enzyme activity; the latter understood as an adaptive response to an increased level of oxidation products.

In our study, peroxidase activity decreased with age increase in all of groups, especially MetS with > 3 components.

Because inhibition of cholesteryl ester transfer protein increases HDL-C level and decreases LDL levels,[42,43] one explanation of visfatin in cholesterol homeostasis may be via inhibition of cholesteryl ester transfer protein. The sex difference of correlation between visfatin and cholesterol levels may be due to estrogen effect. Estrogen may modulate visfatin to inhibit cholesteryl ester transfer protein in cholesterol homeostasis.[44]

Some studies had examined the relationship between plasma visfatin concentration and age in different populations. However, the results were inconsistent. A negative correlation was found in women with gestational diabetes mellitus,[45] but a positive correlation in patients with MetS.[46] The obtained results by Dogru et al.[15] were consistent with this study.

Decrease of oxidative stress association with elevating the expression of antioxidant enzymes, superoxide dismutase, catalase, glutathione, and GPx in addition to lowering LDL-C, TG, and CRP and elevating HDL-C.[47] Chen et al.[48] reported the value of WC was significantly correlated with and GPx activities in MetS patients.

We did not find any correlations between visfatin and lipid profile, glucose, and other measured parameters. Our results are different from previous reports. Contrary to our results, in multiple step-wise regressions analysis by Zhong et al.[49] LDL-C was identified as the independent factor that influences serum visfatin. They concluded visfatin may correlate with the metabolism of cholesterol. Furthermore in the study by Chen et al.,[45] serum visfatin correlated negatively with LDL-C in women with MetS. Fukuhara et al.[33] identified visfatin as an adipocytokine predominantly secreted from visceral adipocytes. Computed tomographic scan demonstrated that plasma visfatin levels correlated strongly with the visceral fat area and weakly with the subcutaneous fat area in 101 male and female human subjects.[33] One of the study revealed visfatin levels correlate with WC and waist-hip ratio.[44] However, previous reports[16,49,50] and this study have not found this correlation. The discrepancy between the studies may be explained by differences in patient populations or different methods of sample collection[51] and detection.[52]

Conclusion

Peroxidases activities in MetS patients can be related to visfatin levels. Gender influences on GPx activity probably and was lower in female patients with MetS. Hyperglycemia does not influence peroxidases activities and visfatin levels.

Suggestions

Further study needs to be done to clarify the exact role of visfatin in MetS, especially homeostasis of lipid. According to the menstrual cycle influences on levels of visfatin and peroxidases activity and thus it should be considered. The correlation between other antioxidant enzymes such as superoxide dismutase, catalase, and glutathione with visfatin is investigated in the future.

Study limitations

Our study had some limitations. First, the number of participants of each both groups was small. Second, this study was a cross-sectional study, and therefore, no causal relationship could be defined. Third, age range of the participants was wide that may be influencing on peroxidases activity and plasma visfatin levels.

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Conflict of Interests

Authors have no conflict of interests.

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