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Original Article

Evaluation of Serum Biomarkers in Patients with Sarcoidosis: Can Visfatin Be a New Biomarker for Sarcoidosis?

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Abstract

OBJECTIVES: Sarcoidosis is a chronic systemic inflammatory disease that affects multiple organ systems. The role of biomarkers in the diagnosis and prognosis of sarcoidosis is increasing. Interest in the role of adipose tissue-mediated inflammation in the pathogenesis of inflammatory diseases has increased in recent years. Visfatin is a proinflammatory adipocytokine that has been studied for several inflammatory diseases such as diabetes mellitus, obesity, and metabolic syndrome. We aimed to assess serum visfatin levels in sarcoidosis and its relationship with other markers of inflammation such as C-reactive protein (CRP), angiotensin-converting enzyme (ACE) and erythrocyte sedimentation rate (ESR).

MATERIALS AND METHODS: We enrolled 59 patients with sarcoidosis and 21 healthy controls and measured plasma levels of visfatin, along with serum CRP, ESR, and ACE using ELISA (enzyme-linked immunosorbent assay) kits (Blue Gene Biotech, Shanghai, China).

RESULTS: Visfatin levels did not differ significantly between the patients and control subjects (29.9±15.8 ng/mL for patients and 23.93±16.73 ng/mL for controls, p=0.15), and there was no correlation between visfatin and serum CRP, ACE, or ESR in patients with sarcoidosis.

CONCLUSION: Visfatin is recently being discussed as a biomarker for inflammatory diseases in several studies, and results are controversial. In our study, no differences were found in the serum levels of visfatin between patients with sarcoidosis and the control group.

KEYWORDS: Biomarkers, inflammation, sarcoidosis, visfatin

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INTRODUCTION

Sarcoidosis is a multi-system disorder with a predilection to affect the lungs. Although spontaneous remission can often occur, approximately one-third of the cases develop into a chronic disease that may be fatal [1, 2]. The etiology of sarcoidosis is yet to be fully understood. However, immunologic evidence and the geographical variation of the disease have suggested various causes, including infection, occupational exposure, and genetic factors [3]. Although the mechanisms are yet to be defined, a chronic inflammatory state is thought to produce pathophysiological effects, resulting in the formation of non-caseating granuloma in the lungs and other systems [4]. Despite the several known gene associations, there is a lack of clinically useful biomarkers [5].

Adipokines are proteins originating from adipose tissues and macrophages, and they regulate the inflammatory response in many chronic inflammatory disorders [6]. Visfatin, previously named pre-B cell colony-enhancing factor, is a pro-inflammatory adipokine [7]. It is a limiting enzyme in nicotinamide adenine dinucleotide (NAD) synthesis and is also known as nicotinamide phosphoribosyltransferase (NAMPT) [8]. Visfatin has been proposed as a new marker of inflammation in diabetes mellitus (DM), metabolic syndrome, polycystic ovary syndrome, coronary artery disease, and rheumatoid arthritis (RA) [9–13]. Visfatin reportedly induced the secretion of proinflammatory cytokines including tumor necrosis factor-α (TNF-α), interleukin (IL)-6, and IL-8, while these cytokines induced the expression of visfatin [14]. In sarcoidosis pathogenesis, the antigen recognition by CD4 positive T lymphocytes is a critical step in triggering the disease. During the inflammatory process, production of cytokines, such as TNF-α, INF-10, IL-2, transforming growth factor-β (TGF-β), IL-8, IL-12, IL-10, and IL-23, increases [15]. The presence of common inflammatory pathways suggests that visfatin could play a role in the pathogenesis of sarcoidosis. The relationship between sarcoidosis and visfatin has not been previously reported. Therefore, we aimed to assess visfatin levels in sarcoidosis patients and evaluate the relationship between the visfatin concentration and

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angiotensin converting enzyme (ACE), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), lung function tests, and the stage of sarcoidosis.

MATERIALS AND METHODS

We included 59 patients with sarcoidosis and 21 healthy controls who were admitted to the outpatient clinic of our hospital between May 2015 and January 2016. Patients with diabetes mellitus and those having a body mass index over 25 kg/m^2 were excluded. Each patient underwent routine tests, such as posteroanterior chest radiography, complete blood count (CBC), serum ACE levels, and lung function tests. In addition, venous blood samples were collected from each subject for analysis of visfatin. After centrifugation at 3000 rpm for 10 minutes, the supernatant was stored at -20°C. Serum visfatin concentrations were analyzed by ELISA kits (Blue Gene Biotech, Shanghai, China). Our study complied with the ethical principles of the Helsinki Declaration. All participating subjects gave informed consent. Our study was approved by the Ethical Committee of University of Abant İzzet Baysal Clinical Research.

Statistical Analyses

Data were analyzed with the Statistical Package for Social Sciences version 22.0 for Windows software (IBM Corp.; Armonk, NY, USA). Descriptive data were given as number of participants and frequency. Categorical variables were expressed as the number of patients and the visfatin percentage value. Categorical variables were compared using the Chi-square and Fisher's exact tests. Continuous variables were documented as mean and standard deviation, and the Shapiro-Wilk test was used to determine whether these variables were normally distributed. The Student's t-test and Mann-Whitney U test were used for continuous variables depending on the normality of their distribution. Spearman's correlation test was used to analyze the relationship between variables. A p-value of <0.05 was considered statistically significant.

According to previous studies with visfatin, in order to find a significant difference of 10 ng/mL between groups with the power of 0.80, the smallest sample size must be 16 patients for each group. In our study, we included 21 controls and 59 patients with sarcoidosis, and the power of our study was 0.90.

RESULTS

The sarcoidosis group included 48 women (81.4%) and 11 men (18.6%) with a mean age of 46.33±12.78 years. Based on the radiological findings, 11 (18.6%) of these cases were considered to be in stage 1, 47 (79.7%) were in stage 2, and 1 (1.7%) was in stage 3. Average serum ACE level was 69.2±52.3 U/L. ESR was 25.70±16.45 mm/h, visfatin level was 29.9±15.8 ng/mL. Patient characteristics are summarized in Table 1.

The control group included 8 women (38%) and 13 men (62%), with a mean age similar to that in the sarcoidosis group (46.33±12.78 and 41±9, respectively, p=0,07).

Visfatin levels did not differ significantly between patients with sarcoidosis and those in the control group (29.9±15.8 ng/mL and 23.93±16.73 ng/mL, p=0,15). CRP (7.09±7.72 mg/L for sarcoidosis and 2.17±1.62 mg/L for control, p<0.001) and ESR levels (25.7±16.45 mm/h for sarcoidosis and 11.26±8.97 mm/h for control, p<0.001) were higher in the sarcoidosis group than in the controls. We also evaluated sarcoidosis patients in two groups according to parenchymal involvement and found no significant difference in their serum visfatin levels (30±15.51 ng/mL and 28.9±17.64 ng/mL, respectively; p=0.826). Patients with parenchymal involvement had higher serum ACE levels and ESR compared with patients without parenchymal involvement (p=0.002 and p=0.043, respectively). The FEV1 (litre=L), FVC (L), and DLCO (mL/mmHg/min) levels were lower in parenchymal involvement but there was no statistical significance (Table 2).

Similarly, sarcoidosis patients with high-ACE levels (>52 U/L) had significantly higher ESR values (30.1±17.5 vs 19.9±13.1, p=0.019). ACE and ESR levels were positively correlated with each other (p=0.03 and r=0.274), and also with serum levels of ACE and CRP (p=0.013 and r=0.325). (Table 3).

A comparison of the visfatin and ACE levels according to previous steroid use or being steroid naive showed no statistical significance (p=0.346 for visfatin; p=0.532 for ACE). Evaluation of visfatin and ACE levels for steroid naive patients and patients on current steroid treatment showed no statistical difference (p=0.721 for visfatin; p= 0.627 for ACE).

Table 1. Characteristics of the patients with sarcoidosis

Number of patients	59
Age, years (mean±SD)	46.33±12.78
Female/Male	48/11
Stage of disease	
0 (n)	0
1 (n)	11
2 (n)	47
3 (n)	1
4 (n)	0
Follow-up period (months)	27.7±32.3
Steroid treatment (previous) (n)	20/59
Steroid treatment (current) (n)	5/59
Steroid-naive patients (n)	39/59

MAIN POINTS

- Visfatin is a proinflammatory adipokine involved in inflammation, and its upregulation results in the development of a chronic low-grade inflammatory state.
- Visfatin is recently being discussed as a biomarker for inflammatory diseases such as diabetes mellitus, obesity, and metabolic syndrome in several studies. But the findings are controversial
- No convincing evidence showing the use of visfatin as a biomarker for sarcoidosis has been found in this study.

Table 2. Comparison of sarcoidosis patients with/without parenchymal involvement

	With parenchymal involvement	Without parenchymal involvement	p
Age (years)	46.27±2.79	46.6±13.38	0.933
CRP (mg/L)	7.62±8.28	4.81±4.11	0.282
Sedimentation (mm/h)	27.08±17.67	19.81±7.69	0.043
Serum ACE level (U/L)	75.91±54.84	39.95±23.92	0.002
Serum visfatin level (ng/mL)	30±15.51	28.90±17.64	0.826
FEV_1 (Litre)	2.51±0.79	2.69±0.74	0.507
FEV_1 (%)	92.59±13.91	102.66±26.5	0. 249
FVC (Litre)	3.05±0.95	3.40±0.81	0.260
FVC (%)	92.68±18.12	109.03±25.83	0.070
DLCO (mL/mmHg/min)	20.76±7.30	21.31±6.4	0.863

CRP: c-reactive protein; ACE: angiotensin-converting enzyme; FEV_1: litre=L; FVC: L; DLCO: mL/mmHg/min

Table 3. Correlation between biomarkers

		ACE	ESR	CRP	Visfatin
ACE	p		0.03	0.013	>0.99
	r		0.274	0.325	<0.001
Visfatin	p	>0.99	0.42	0.30	
	r	<0.001	-0.109	-0.137	

ACE: angiotensin-converting enzyme; ESR: erythrocyte sedimentation rate; CRP: c-reactive protein

DISCUSSION

Serum visfatin levels did not significantly differ between sarcoidosis patients and controls, or between patients with or without parenchymal involvement. Additionally, no correlation was detected between the serum visfatin levels and other inflammatory markers, such as ACE, ESR, or CRP.

Visfatin is a proinflammatory adipokine involved in inflammation, and its upregulation results in the development of a chronic low-grade inflammatory state. This biomarker has been studied for several inflammatory diseases, such as obesity, metabolic syndrome, type 2 DM, polycystic ovary syndrome, and RA, but the findings are controversial [10-13, 16-18]. Subsequent studies described visfatin upregulation in several immune cells, including monocytes, lymphocytes, dendritic cells, and macrophages [19–21]. Visfatin was shown to activate pro-inflammatory pathways and induce other proinflammatory cytokines including TNF-α, IL-1β, and IL-6. Noteworthy high visfatin levels were detected in overweight/obese patients, whereas weight loss induced by both physical exercise and bariatric surgery lowered the circulating levels of visfatin [22]. Similarly, a strong clinical association has been demonstrated between visfatin and type 2 DM, independent of BMI [23-25]. El-Suhaimi et al. [17] suggested that an increased, decreased, or unchanged level of visfatin-induced endothelial angiogenesis is mediated by the vascular endothelial growth factor (VEGF), matrix metalloproteinases (MAPK), and PI3K/Akt signaling pathways. Syrbe et al. [26] reported higher visfatin levels in patients with ankylosing spondylitis (AS) than in controls, with the elevated levels predicting the subsequent progression of radiographic damage when compared with the baseline values. In another study, patients with asthma were found to have significantly higher serum visfatin levels than controls [6]. Inflammatory bowel disease was another condition that was associated with high basal serum visfatin levels [27]. Additionally, a strong correlation has been reported between visfatin and inflammatory markers, such as CRP and IL-6 [22, 28]. However, visfatin levels of the patients in our study were not significantly different from those of the controls. We also detected no correlation between the serum visfatin level and other inflammatory markers, such as ESR or CRP. Similarly, Celap et al. [29] reported that visfatin was not associated with inflammatory markers, such as CRP, in hemodialysis patients. In a study evaluating the correlation of elevated serum visfatin levels and inflammatory markers (hs-CRP, IL-6, TNF-α, waist circumference, triglycerides, and BMI) in obese children, visfatin was only correlated with BMI and IL-6; no significant correlation with hs-CRP was observed [30]. Furthermore, serum visfatin levels, CRP, and ESR showed no correlation in patients with chronic viral hepatitis B and metabolic syndrome [31, 32]. We found no statistical difference in the visfatin levels between steroid-naive patients and patients currently receiving steroid treatment. Our results suggest that although inflammatory pathways are present in sarcoidosis pathophysiology, the visfatin level is not increased. The general role of visfatin in inflammatory diseases is still unclear because studies have shown inconsistent results.

Under different conditions, including metabolic syndrome, type 2 DM, rheumatic disease, and hemodialysis, visfatin had a positive correlation with the presence and severity of the disease [33, 34]. Circulating visfatin levels and disease activity had similar correlation in patients with RA [35]. In the present study, we found no significant difference with regard to the inter-group visfatin levels between groups with or without parenchymal involvement or stages. Patients with pulmonary involvement had significantly higher serum ACE levels and ESR. We found no association between CRP levels and parenchymal involvement. Our findings support those of Dirican et al. [36]. Based on these results, we suggest that disease severity may be associated with the serum ACE levels and ESR rather than CRP.

Our study had several limitations. First, the number of patients was limited as it was a single center study. Cohort studies with larger sample sizes are needed to investigate this possibility. Second, considering the cross-sectional nature of our study, we only evaluated and compared one sample of blood that was collected at a single visit for visfatin from all patients with different durations of sarcoidosis. Periodic serum visfatin level monitoring during the follow-up period in newly diagnosed patients with sarcoidosis may yield different results. More data from the same patient could also

help in calculating a base level for visfatin and studying the use of visfatin as a biomarker for changes in inflammation. Third, we did not exclude patients with hypertension. However, Dogru et al. [37] reported that visfatin plasma levels were not correlated with blood pressure in patients with uncomplicated hypertension and that adipokine dysregulation has no apparent role in new-onset hypertension. Moreover, we included patients receiving steroid treatment. However, previous studies have reported that visfatin levels were not affected by systemic glucocorticoid treatment [38, 39]. We also found that the serum visfatin levels were similar between the groups that were receiving current or had received steroid treatment. Even though studies have not demonstrated a relationship between visfatin and steroid use or hypertension, future studies should still take into account that proinflammatory pathways can be affected.

In conclusion, our study was possibly the first to evaluate serum visfatin levels in sarcoidosis patients. We found no convincing evidence indicating the use of visfatin as a biomarker for sarcoidosis. In order to better elucidate the role played by visfatin in chronic inflammatory disorders, such as sarcoidosis, large prospective cohort studies will be required.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of University of Abant İzzet Baysal (2015-49).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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