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Serum osteopontin levels in patients with psoriasis vulgaris and its relation with oxidative stress

Psoriazis vulgariste serum osteopontin düzeyi ve bunun oksidatif stres ile ilişkisi

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Abstract

Background and Design: Oxidative stress is known to play a role in the etiopathogenesis of psoriasis. Recent data suggest that osteopontin (OPN) can also play a role in the pathogenesis of psoriasis. In the current study, OPN levels and oxidative stress were evaluated in patients with psoriasis.
Materials and Methods: The study included 61 patients with psoriasis and 62 healthy controls. The OPN levels, total antioxidant status (TAS), total oxidant status (TOS), and oxidative stress index (OSI) were measured using serum. The disease severity was evaluated using the psoriasis area and severity index (PASI).
Results: No statistically significant differences in OPN, TAS, and OSI values were identified between the psoriasis and control groups. A negative correlation was found with the TAS. There was no statistically significant correlation between the PASI score and OPN, TAS, TOS, and OSI values.
Conclusion: We did not find a statistically significant correlation between OPN levels and oxidative stress in patients with psoriasis. We believe that larger and more detailed studies are needed to highlight the role of OPN and oxidative stress in the etiopathogenesis of psoriasis.
Keywords: Psoriasis, osteopontin, oxidative stress, etiology

Öz

Amaç: Psoriazis etiyopatogenezinde, oksidatif stresin rolü olduğu düşünülmektedir. Son zamanlarda, osteopontinin (OPN) psoriazis patogenezinde rol aldığını savunan çalışmalar yayınlanmaktadır. Bu çalışmada; psoriazis hastalarında, OPN ve oksidatif stres değerlendirilecektir.
Gereç ve Yöntem: Çalışmaya 61 psoriazis vulgaris hastası ve 62 sağlıklı kontrol katıldı. OPN, total antioksidan seviye (TAS), total oksidan seviye (TOS) serum düzeyleri ve oksitadif stres indeksi (OSİ) ölçüldü. Hastalık şiddeti psoriazis alan şiddet indeksi (PAŞİ) kullanılarak değerlendiririldi.
Bulgular: Psoriazis vulgaris hastaları ile kontrol grubu arasında serum OPN, TAS, OSİ düzeyleriyle ilgili istatistiksel olarak anlamlı fark saptanmadı. TOS değerlerinde ise negatif ilişkili korelasyon bulundu. PAŞİ ile OPN, TAS, TOS, OSİ arasında anlamlı istatistiksel korelasyon tespit edilmedi.
Sonuç: Psoriazis hastalarında, OPN ile oksidatif stres korelasyon ilişkisi açısından anlamlı istatistiksel fark tespit etmedik. Psoriazis etiyopatogenezinde OPN ve oksidatif stres rollerinin daha iyi aydınlatılması için geniş ve kapsamlı çalışmalara ihtiyaç duyulduğu kanısındayız.
Anahtar Kelimeler: Psoriazis, osteopontin, oksidatif stres, etiyoloji

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Introduction

Psoriasis is a chronic inflammatory hyperproliferative skin disease, and its exact etiology is currently unknown[1]. It is thought that the disease starts with the activation of T-cells and the release of cytokines involved in autoimmune pathways. Following stimulation by antigen-presenting cells, the differentiation of T-helper lymphocytes (Th) into Th1 cells, which secrete interleukin-17 (IL-17) and interferon-gamma (IFN-γ), plays a role in the pathogenesis of psoriasis.

Osteopontin (OPN) is a phosphorylated glycoprotein that is secreted by macrophages, T- and B-cells, osteoclasts and osteoblasts, hepatocytes, dendritic cells, vascular smooth muscle cells, and endothelial and epithelial cells[2,3]. There are two isoforms of OPN, namely, secreted OPN (sOPN) and intracellular OPN (iOPN)[4]. sOPN interacts with integrin and CD44, and acts as an effector of Th1 cytokines. A positive correlation between iOPN levels and macrophage migration along with IFN-γ secretion in dendritic cells has been reported. The OPN complex augments the Th17 response by blocking IL-27 secretion from dendritic cells. Overall, OPN can enhance the immune response mediated by Th1 and Th17 cells[5,6].

Th1, Th17, and Th22 cells are known to play important roles in the pathogenesis of psoriasis[7]. Several studies have reported significantly high levels of OPN in the serum and skin lesions of patients with psoriasis[2,8-10]. OPN receptors were found to be expressed on keratinocytes and monocytes. Activation of these cells by OPN leads to the secretion of chemokines and cytokines, which triggers the polarization of T-cells to Th1 and Th17 cells. It is hypothesized that locally sOPN from lesional keratinocytes, inflammatory cells, and endothelial cells is involved in the pathogenesis of psoriasis[11]. Oxidative stress may also play a role in the etiopathogenesis of psoriasis; additionally, abnormal apoptotic activity may lead to epidermal thickening and distorted tissue structure[12]. Dysfunction in the antioxidant system against reactive oxygen species and increased reactive oxygen radicals, particularly deregulation in lipid metabolism caused by oxidative radicals, have been implicated in the pathogenesis of psoriasis[13,14]. In the current study, we aimed to investigate the relationship between oxidative stress and serum OPN levels in patients with psoriasis in order to better understand the etiopathogenesis of this disease.

Materials and Methods

Selection of the study group

The current study was carried out between 2016 and 2017. The study was approved by the Trakya University Faculty of Medicine Local Ethics Committee (approval number: 09/16, date: 10.05.2017), and subjects who agreed to participate provided written consent for inclusion in the study. A total of 123 individuals were recruited, including 61 patients with a clinical and/or histopathological diagnosis of psoriasis vulgaris (over the age of 18 years) and 62 healthy controls. Patients with a history of use of any drug that might affect oxidative stress (acitretin, cyclosporine, methotrexate, vitamin supplements, phototherapy, or other biological agents) along with smoking and/or alcohol consumption; patients with a diagnosis of rheumatoid arthritis, systemic lupus erythematosus, type 1 diabetes mellitus, malignancy, inflammatory disorders like Behçet's disease, pneumonia, urinary tract infection, infectious diseases like brucellosis, or liver and/or kidney dysfunction; and individuals who were pregnant were excluded from the study. After evaluating the history of the patients, psoriasis area and severity index (PASI) scores were calculated for those patients who were eligible to be recruited in our study. For this, 10 cc venous blood samples were taken from the patients and controls. The samples were centrifuged at 3,000 ×g for 10 minutes, and the serum was separated and kept at -80 °C until the day of analysis. Serum OPN levels were evaluated using commercial enzyme-linked immunosorbent assay kits (Elabscience, Wuhan, China) according to the manufacturer's recommendations. Serum OPN levels were calculated in ng/mL. The serum total antioxidant status (TAS) was measured using Erel's[15] method. The principle of this method relies on the reduction of the dark blue-green colored 3-ethylbenzothiazoline-6-sulfonic acid (ABTS) radical into its colorless form in the presence of antioxidants. The TAS was calculated in mmol trolox Eq/L using standard trolox. The serum total oxidant status (TOS) was also measured using Erel's[16] method. In this assay, oxidation of a ferrous ion-chelator complex in the presence of oxidants leads to the formation of ferric ions, which develops a color owing to the presence of chromogenic substances in an acidic environment. The TOS was calculated in μmol H_2O_2 Eq/L. The oxidative stress index (OSI) was calculated using the following formula:

OSI = [TOS (mmol H_2O_2 Eq/L)/TAS (mmol Trolox Eq/L)] ×100.

Statistical Analysis

SPSS 23.0 (IBM Corp) with serial number 10240642 was used for statistical analyses. Quantitative variables were analyzed with descriptive statistics such as mean, median, standard deviation, minimum, and maximum. The normality of the data was ascertained with the Shapiro-Wilk test. For variables that showed a normal distribution, group comparisons in independent groups were carried out using the t-test. For variables that did not show a normal distribution, the Mann-Whitney U test was used. Quantitative variables were summarized with numerical and percentage information. Group comparisons were evaluated using the chi-square test. Furthermore, distributions of the variables were presented visually with histograms, box line graphs, and dispersion graphs. For statistical significance, the type 1 error threshold was defined as 0.05.

Results

There were no statistically significant differences in the distribution of age or gender between the patient and control groups (p>0.05) (Table 1). The average OPN and OSI values were also similar between the two groups (p=0.34 and p=0.187, respectively). However, the average TOS value was significantly different between the two groups (p=0.036). The TAS value was compared between the groups using the t-test (independent groups) as the groups showed a normal distribution; however, the difference was not statistically significant (p=0.357) (Table 1).

The Spearman rho correlation coefficient between the OPN and OSI values was 0.184 (p=0.041). The dispersion graphs related to the OPN and OSI values showed no significant correlation. In the patient group, no statistically significant correlations were identified between the PASI and OPN values, and between the PASI and OSI values. The intragroup, intergender distribution, and intergender comparisons indicated that

the distributions of the TAS and TOS values between genders were significantly different in the control group (p<0.05). However, no significant differences in any of the variables as a function of gender were identified in the patient group (p>0.05). The evaluation of correlation coefficients between age and other variables also suggested the absence of any statistically significant relationship (Table 2). A statistically significant yet weak correlation was identified between age and the TOS value, and age and the OSI value in the control group; however, no statistically significant relationship was identified between age and other variables.

Discussion

OPN can contribute to the pathogenesis of psoriasis via the induction of the Th1/Th17 pathway and the release of relevant cytokines[8]. An increase in oxidative stress and free radical formation was also found to be related to the pathogenesis of psoriasis. Increased release of the superoxide anion in dermal fibroblasts is thought to play an important role in the inflammatory nature of psoriasis.

OPN receptors are expressed in keratinocytes and monocytes. Activation of these cells by OPN leads to the release of chemokines and cytokines, which, in turn, leads to the polarization of naïve Th-cells into Th1 and Th17-cells. Therefore, it is hypothesized that locally sOPN from lesional keratinocytes, inflammatory cells, and endothelial cells plays a role in the pathogenesis of psoriasis[11].

Buback et al.[5] hypothesized that increased OPN secretion after bruising of the skin or exposure to antigens can cause the induction of dendritic and Langerhans cells in the lymph nodes. These two groups of cells can cause an inflammatory response via the activation of a Th1 response. Moreover, superantigens can directly induce the release of sOPN from effector T-lymphocytes. This increase in sOPN can lead to the enhanced release of IFN-γ and IL-17 through the Th1 and Th17 pathways, respectively. iOPN can also activate the Th17 pathway, similar to sOPN. It is postulated that OPN is effective in the pathogenesis of psoriasis through the prevention of apoptosis and the triggering of keratinocyte proliferation, as well as the activation of inflammation via the mechanisms described above. OPN may also play a role in other diseases characterized by chronic inflammation, such as obesity, Crohn's disease, autoimmune disorders, some cancer types, and cardiac fibrosis[2].

Studies suggesting the presence of a relationship between OPN and psoriasis stated that OPN levels were significantly higher in the serum and/or skin lesions of patients with psoriasis[2,8-10]. In our study, the mean OPN levels in the patient and control groups were 28.531±20.590 and 24.524±19.249, respectively. Although the mean value was higher in the patient group, the difference did not reach statistical significance (p=0.34). Additionally, there was no significant difference in OPN levels as a function of age and gender in both the patient and control groups (p>0.05). The results of our study are therefore not in line with the findings of the current literature. However, it should be kept in mind that the numbers of studies and cases reported on the topics of psoriasis and OPN are very limited. Buommino et al.[17] reported a comparison of biopsy materials between patients with psoriasis and controls, and showed an increase in the expression of OPN in the skin

Table 1. Demographics of the study groups and statistics related to OPN, TAS, TOS, and OSI values

Variable	Patient group	Control group	p-value
Age (years)	40.43 (19-74±14.507)	40.61 (18-80±14.598)	0.907*
Gender	**Female 24 (39.3%)**	**Female 30 (48.4%)**	0.365**
	Male 37 (60.7%)	**Male 32 (51.6%)**	
OPN	28.53 (10.945-69.907) ±20.590	24.524 (1.455-79.275) ±19.249	0.340*
TAS	0.665 (0.290-1.000) ±0.166	0.638	p=0.357***
TOS	7.411 (5.101-11.000) ±1.351	7.997 (3.758-11.946) ±1.718	**0.036***
OSI	1.222 (0.696-2.361) ±0.403	1.381 (0.495-5.139) ±0.706	0.187*

*Mann-Whitney U test, **Pearson chi-square test, ***t-test in independent groups, OPN: Osteopontin, TAS: Total antioxidant status, TOS: Total oxidant status, OSI: Oxidative stress index

Table 2. Correlation coefficients between different variables (antioxidant status, OPN levels, and age) in patients with psoriasis

		PASI	OPN	TAS	TOS	Age
PASI	r*		-0.123	-0.195	0.157	0.044
	p		0.347	0.132	0.227	0.739
OPN	r*	-0.123		-0.195	0.157	0.044
	p	0.347		0.132	0.227	0.739
TAS	r*	0.047	-0.195		0.168	0.076
	p	0.722	0.132		0.195	0.558
TOS	r*	0.073	0.157	0.168		-0.070
	p	0.575	0.227	0.195		0.589
Age	r*	0.165	0.044	0.076	-0.070	
	p	0.205	0.739	0.558	0.589	

*Spearman rho correlation coefficient, OPN: Osteopontin, TAS: Total antioxidant status, TOS: Total oxidant status, PASI: Psoriasis area and severity index

of patients with psoriasis, both with and without lesions. In light of these findings, it can be postulated that evaluation of OPN from skin biopsy samples may provide more robust data. Przepiórka-Kosińska et al.[18] recently reported serum OPN and IL-17 levels in 107 patients with psoriasis, as well as the correlations of OPN and IL-17 levels with disease severity. These authors reported that psoriasis severity was not correlated with OPN levels. Supporting these findings, we also did not observe a statistically significant correlation between PASI and OPN values.

A shift in the equilibrium between prooxidants and antioxidants toward prooxidants in biological systems is called oxidative stress. In response to oxidative stress, cells generally activate endogenous antioxidant systems. If such activations are not sufficient to mitigate oxidative stress, the cells may start accumulating reactive oxygen species and free radicals that can harm the cells and cellular macromolecules like proteins, carbohydrates, and lipids via different mechanisms[19]. Oxidative stress is among the main causes of the distorted tissue structure and thickened epidermis observed in psoriasis; additionally, a deregulation in lipid metabolism has been implicated in this process[12,14]. A decrease in the TAS value and an increase in the TOS value have been reported in patients with psoriasis[14,20]. Rajappa et al.[21] reported that the OSI value was significantly higher in patients with psoriasis than in controls, while Usta et al.[22] reported the absence of significant differences in the TAS and TOS values between patients with psoriasis and controls. Severin et al.[23] also reported the lack of a significant difference in the TAS value in patients with psoriasis. In the current study, the TAS, TOS, and OSI values were 0.665±0.151, 7.411±1.351, and 1.222±0.403, respectively, in patients with psoriasis and were 0.608±0.151, 7.997±1.718, and 1.381±0.706, respectively, in controls. Although the TAS and OSI values were not significantly different ($p>0.05$), the TOS values were significantly lower in patients with psoriasis than in controls ($p=0.036$). The results of the current study therefore support some of the findings of available literature on the relationship between OPN and psoriasis, but not others, indicating the need for further studies. Based on our data and the currently available literature, the use of skin biopsy samples may provide more accurate outcomes since partial diffusion of antioxidants and oxidants to the serum can lead to contradictions. Peluso et al.[24] found that peroxidation markers were more sensitive than the TAS when determining oxidative stress in psoriasis. Thus, in the measurement of oxidative stress in patients with psoriasis, the evaluation of more sensitive parameters rather than the TAS, TOS, and OSI can be suggested.

Emre et al.[14] reported the lack of a significant relationship between the PASI and TAS, as well as between the TOS and OSI. We also observed the lack of a significant correlation between the PASI and the parameters TAS, TOS, and OSI ($p>0.05$). To our knowledge, case-control studies investigating the relationship between OPN and oxidative stress in patients with psoriasis have not been reported in the literature.

Studies carried out in humans and mice showed that OPN can play a role in the development of atherosclerosis. Plasma OPN levels were reported to be higher in patients with coronary artery disease, and a positive correlation was shown between OPN levels and disease severity. The role of OPN in the incidence of cardiovascular diseases in patients with psoriasis was therefore considered. Chen et al.[25] detected significant positive correlations between plasma OPN levels and hypertension in patients with psoriasis regardless of age. Georgiadou et al.[26] reported the presence of a positive correlation between OPN and malonylaldehyde levels in patients with coronary artery disease. This correlation emphasizes the crosstalk between OPN and oxidative stress. Of note, OPN was shown to increase the production of nicotineamide adenine dinucleotide phosphate oxidase and reactive oxygen species in myofibroblasts and smooth muscle cells. An association between oxidative stress induced by angiotensin-2 and elevated OPN levels was also reported. Partridge et al.[27] showed an increase in the mRNA expression of OPN with oxidative stress in smooth muscle cells. The authors reported that the presence of oxidized low-density lipoprotein increased both intracellular and extracellular levels of OPN via the induction of oxidative stress in smooth muscle cells, fibroblasts, endothelial cells, and keratinocytes. Very recently Bartosińska et al.[28] reported that serum concentration of OPN in psoriasis patients was negatively correlated with total cholesterol and triglyceride. Overall, although the currently available data suggest the presence of a significant correlation between oxidative stress and OPN, we were not able to corroborate this relationship as we did not observe statistically significant correlations between OPN levels and TAS, TOS, and OSI values, or between PASI values and OPN, TAS, TOS, and OSI values. Our data on TAS, TOS, and OSI values in patients with psoriasis and controls support some but not all findings of studies available in the current literature. This highlights the need for further investigations on both serum and tissue samples to firmly establish the presence of a relationship among oxidative stress, OPN, and psoriasis.

Study Limitations

The most important limitation of our study is the low number of cases. Further, environmental factors, such as smoking, socioeconomical structure, stress factors, and diet, as well as exposure to passive smoke, which might affect the results of our study, were not considered in patients with psoriasis or controls. We evaluated OPN, TAS, and TOS values only in the serum, which can be considered an additional limitation.

Conclusion

Larger and more comprehensive studies are needed to better understand and establish the roles of OPN and oxidative stress in the etiopathogenesis of psoriasis.

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Ethics

Ethics Committee Approval: The study was approved by the Trakya University Faculty of Medicine Local Ethics Committee (approval number: 09/16, date: 10.05.2017).

Informed Consent: Subjects who agreed to participate provided written consent for inclusion in the study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: C.K., S.P., Concept: C.K., S.P., Design: C.K., S.K., S.K.F., Data Collection or Processing: C.K., G.S.Ö., Analysis

or Interpretation: C.K., S.K., S.K.F., S.P., G.S.Ö., Literature Search: C.K., Writing: C.K.

Conflict of Interest: The authors declared that they have no conflict of interest.

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