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Psoriazis Vulgaris' li Hastalarda Oksidatif Stres Belirteci ve İskemi Modifiye Albümin Düzeylerinin Değerlendirilmesi

Evaluation of Oxidative Stress Marker and Ischemia Modified Albumin Levels in Patients with Psoriasis Vulgaris

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ÖZ

Amaç: Sistemik inflamatuar bir deri hastalığı olan psoriazis vulgaris (PS), hücresel immün mekanizmalarıyla ilişkilidir. Çalışmamızın amacı PS' li hastalarda ve kontrollerde oksidatif stres belirteçleri ve iskemi modifiye albümin düzeylerini değerlendirmektir.

Materyal ve Metot: Eylül 2020 - Mayıs 2021 tarihleri arasında dermatoloji polikliniğine başvuran ve PS tanısı konan (18 yaş üstü) katılımcılar (n=50) çalışmaya alındı. PS hastalarında nitrik oksit (NO), malondialdehit (MDA), 8-hidroksi 2-deoksi guanozin (8-OHdG), süperoksit dismutaz (SOD), glutatyon peroksidaz (GPx) parametreleri ve iskemi modifiye albümin (IMA) seviyeleri gibi oksidatif stres belirteçleri ve kontroller belirlenerek aralarındaki ilişkiler değerlendirildi.

Bulgular: Çalışmaya 50 PS' li ve 50 sağlıklı kontrol olmak üzere toplam 100 gönüllü alındı. Oksidatif stres belirteçlerinin ölçümlerinde GPX, 8-OHdG, MDA, IMA, IMA/Albumin ve NO parametreleri hasta ve kontrol grupları arasında istatistiksel olarak anlamlı bulundu. Ayrıca iki psoriazis alanı ve şiddet indeksi (PASI) grubu arasında SOD, 8-OHdG, IMA ve NO istatistiksel olarak anlamlı bulundu.

Sonuç: PS' li hastalarda 8-OHdG, MDA, IMA, IMA/Albumin, NO düzeylerinde yükselme ve GPx düzeylerinde azalma gözlendi. Bu konuyu netleştirmek için daha fazla ve kapsamlı çalışmalara ihtiyaç vardır.

Anahtar Kelimeler: İskemi modifiye albümin, oksidatif stres, psoriazis vulgaris

ABSTRACT

Objective: As a systemic inflammatory skin disease, psoriasis vulgaris (PS) is associated with cellular immune mechanisms. We aimed to evaluate oxidative stress markers and ischemia modified albumin levels in patients with PS and controls.

Materials and Methods: The participants (over age of 18) who applied to the dermatology outpatient clinic between September 2020 - May 2021 and diagnosed with PS (n=50) were enrolled into the study. Oxidative stress markers such as nitric oxide (NO), malondialdehyde (MDA), 8-hydroxy 2-deoxy guanosine (8-OHdG), superoxide dismutase (SOD), glutathione peroxidase (GPx) parameters and ischemia modified albumin (IMA) levels in patients with PS and controls were evaluated.

Results: A total of 100 volunteers, 50 with PS and 50 healthy controls were enrolled in the study. In the measurements of oxidative stress markers, GPX, 8-OHdG, MDA, IMA, IMA/Albumin and NO parameters were found to be statistically as significant between the patient and control groups. Also, SOD, 8-OHdG, IMA and NO were found to be statistically as significant between two psoriasis severity index (PASI) groups.

Conclusion: Elevated levels of 8-OHdG, MDA, IMA, IMA/Albumin, NO and decreased levels of GPx were observed in patients with PS. To clarify this topic, further and comprehensive studies are needed.

Keywords: Ischemia modified albümin, oxidative stress, psoriasis vulgaris

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INTRODUCTION

As a systemic inflammatory skin disease, psoriasis vulgaris (PS) is associated with cellular immune mechanisms. The disease observed at a rate of 1-4% in the general population. PS is characterized by abnormal keratinocyte differentiation, elevated keratinocyte proliferation, differences in dermal vascularity, increased cellular antioxidant activity, monocytes, macrophages, and polymorphonuclear leukocytes.[1] The pathogenesis of the disease is not fully known. Reactive oxygen derivatives formed as a result of normal metabolism in healthy individuals and removed by the antioxidant system, which is the body's defense mechanism. Oxygen radicals and antioxidant defense mechanism function in a balance. When this balance is disrupted in favor of radicals, it is called as oxidative stress.

To minimize this oxidative damage, low molecular weight compounds such as beta-carotene, ascorbate, tocopherols, uric acid, glutathione, coenzyme Q10, proteins such as metallothionine and ferritin play important role in the body. Various enzymes such as thioredoxin/thioredoxin reductase, superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT), ischemia modified albumin (IMA) and glutathione reductase (GR) take part significant roles in the antioxidant system. Oxidative damage occurs as a result of the failure of these control mechanisms. Biomolecular damage due to increased reactive oxygen metabolites results in lipid peroxidation, DNA mutation or breakage, enzyme activation or inactivation, protein oxidation or degradation.[2] It is thought that reactive oxygen derivatives may be one of the factors that play a role in the onset or chronicity of PS. In a study, it was reported that low SOD activity in PS indicates insufficiency in the antioxidant system.[3] In another study, it was shown that serum malondialdehyde levels increased in proportion to the severity of PS and regressed to normal levels with treatment.[4] PS is a common chronic dermatological disease. There are few studies evaluating its relationship with antioxidant mechanisms, which are thought to be effective in the etiopathogenesis of the disease. However, most of the studies were conducted with a limited number of biomarkers and a small number of participants.

The purpose of the study is to evaluate and compared the blood levels of nitric oxide (NO), malondialdehyde (MDA), 8-hydroxy 2-deoxy guanosine (8-OHdG), superoxide dismutase (SOD), glutathione peroxidase (GPx) parameters, which are frequently used as markers of oxidative stress, and ischemia modified albumin levels in patients with PS and controls, and to reveal it's possible role in the etiopathogenesis of PS.

MATERIALS AND METHODS

Ethics Committee Approval: This study was approved by the Necmettin Erbakan University Faculty of Medicine Non-Invasive Clinical Research Ethics Committee (Date: 30/07/2021, decision no: 2021/3356). All the participants signed their consents prior the study.

The participants (over age of 18) who applied to the dermatology outpatient clinic between September 2020-May 2021 and diagnosed with PS (n=50) were enrolled into the study. Volunteers (n=50) were selected from participants without disease as control group. Pregnancy, lactation, smoking and alcohol users, liver or kidney dysfunction, history of systemic diseases such as diabetes, obesity, atherosclerotic heart disease, metabolic disease, those taking anti-inflammatory and immunosuppressive therapy, received topical treatment in the previous month or systemic treatment in the last three months, history of major trauma or surgery in the last three months and those who regularly used antioxidants were excluded from the study. The clinical evaluation of the patients included in the study was performed with the psoriasis area and severity index (PASI) scoring system. The patients were randomized into 2 groups according to the PASI score, as those with a PASI score of 10.00 and/or less (mild disease) and those with a PASI score greater than 10.00 (severe disease).

For the measurement of SOD, GPx, 8-OHdG, MDA, IMA, IMA/albumin and NO, approximately 4 ml of venous blood samples were taken from the antecubital region in the morning after 10-12 hours of fasting, from patients and healthy volunteers. After the blood was transferred to a tube containing ethylenediaminetetraacetate (EDTA), it was centrifuged at +4°C and 1500 rpm for 12 minutes, and the samples were separated and stored at -80°C until the assay.

Measurement of SOD, GPx, 8-OHdG, MDA, IMA and NO: For SOD activity, commercially kit (Cayman Chemical Co., MI, USA, kit catalog no: 706002) was used. The amount of superoxide was stained with nitrobluetetrazolium (NBT), and the color intensity was measured as spectrophotometrically. For GPx determination, commercially kit (Cayman Chemical Co., MI, USA, kit catalog no: 703102) was used. Enzyme activity was determined by the decrease in the optical density of the reaction content formed as a result of the oxidation of nicotinamide adenine dinucleotide phosphate to nicotinamide adenine dinucleotide (NADPH to NADP) at 37°C. Measurement of 8-OHdG was performed with commercially kit (Cayman Chemical Co., MI, USA, kit catalog no: 589320). The formed yellow color measured at 412 nm. Commercially kit was used for MDA determination (Cayman Chemical Co., MI,

USA, kit catalog no: 10009055). It was measured by the colorimetric method in the 530-540 nm spectrum of the MDA-TBA adduct, which is formed as a result of the interaction of MDA and thiobarbituric acid (TBA) in an acidic (90-100°C) environment. The IMA measurement was performed by a rapid colorimetric method depends on albumin cobalt binding. The amount of albumin bound cobalt was measured spectrophotometrically at 470 nm in comparison with a serum cobalt blank without dithiothreitol (DTT). Measurement of NO was performed with commercially kit (Cayman Chemical Co., MI, USA, kit catalog no: 589320). The formed color was measured as colorimetrically.

Statistical Analysis: Data analysis was performed with SPSS for Windows 22 package program. The distribution of continuous and discrete numerical variables was performed with the Kolmogorov Smirnov test, and the homogeneity of variances was investigated by Levene's test. Descriptive statistics were presented as mean±standard deviation for continuous and discrete numerical variables, and as number of cases and (%) for categorical variables. The significance of the difference between the groups was determined by Student's t test. The significance of the difference between the groups was determined with Mann Whitney U test, when the number of independent groups was two, and the significance of the difference between more than two groups was investigated with the Kruskal Wallis test. If the Kruskal Wallis test statistic results were found to be significant, the difference were determined using Conover's non-parametric multiple comparison test. Categorical variables were evaluated with Pearson's Chi-Square test. PASI scores were investigated using Spearman's correlation test. For $p<0.05$, the results were considered as statistically significant.

RESULTS

A total of 100 volunteers, 50 with PS and 50 healthy controls were included in the study. As shown in Table 1, patient and control groups had a normal distribution in terms of age, gender and BMI. There were no significant differences as statistically.

Average PASI was 9.2 in patients with psoriasis vulgaris and mean disease duration was 8 years as shown in Table 2.

Table 1. Demographic characteristics of psoriasis vulgaris patient and control group.

Parameter	Patient group (n=50)	Control group (n=50)	p value
Age, (year)†	34.9±9.9	38.4±9.3	0.150
Gender, (M/F)‡	28/22	26/24	0.428
Body Mass Index, (BMI)¶	24.22 (12.76–44.92)	23.23 (16.90–34.37)	0.692

†: Student's t test; ‡: Pearson's Chi-Square test; ¶: Mann Whitney U test.

Table 2. Clinical characteristics of the psoriasis vulgaris patients group.

Parameter		n=50
Disease Duration (years)		8 (1-35)
Family History		16 (32 %)
Nail involvement		14 (28 %)
Average PASI		9.2 (5.6-20.4)
PASI	≤10	32 (64 %)
	>10	18 (36 %)

PASI: Psoraisis area and severity index.

In the measurements of oxidative stress markers, GPX, 8-OHdG, MDA, IMA, IMA/Albumin and NO parameters were found to be statistically as significant between the patient and control groups. SOD shows no significant differences as statistically (Table 3).

As shown in Table 4, SOD, 8-OHdG, IMA and NO were found to be statistically as significant between PASI groups.

DISCUSSION AND CONCLUSION

As a chronic and systemic skin disease, PS may cause comorbidities including cardiovascular diseases, diabetes mellitus, psoriatic arthritis, obesity, inflammatory bowel diseases[5] and psychiatric problems.[6] The disease is an erythematous, scaly, inflammatory skin disease that frequently affects the knees, elbows, scalp and genital areas. It can be limited to certain areas or cause plaque lesions or erythroderma, which can affect a significant part of the skin,

Table 3. Measurements of oxidative stress markers of psoriasis vulgaris patient and control group.

Parameter		Mean ± SD	p value*
SOD, (nmol/ml)	Patient Group	5.2±1.7	0.209
	Control Group	5.8±2.0	
GPx (U/gr Hemoglobin)	Patient Group	212.4±75.8	<0.001*
	Control Group	373.2±189.3	
8-OHdG, (nmol/L)	Patient Group	32.3±14.9	0.002*
	Control Group	23.6±7.5	
MDA, (µmol/L)	Patient Group	0.19±0.20	0.015*
	Control Group	0.09±0.10	
IMA, (ABSU)	Patient Group	0.22±0.06	<0.001*
	Control Group	0.18±0.05	
IMA/Albumin, (AU)	Patient Group	0.053 ±0.016	<0.001*
	Control Group	0.042 ±0.012	
NO, (µM/L)	Patient Group	41.5±32.4	0.022*
	Control Group	30.7±16.2	

*Mann Whitney U test, results for p<0.05 were considered statistically as significant; SOD: superoxide dismutase; GPx: glutathione peroxidase; 8-OHdG: 8-hydroxy 2-deoxy guanosine; MDA: malondialdehyde; IMA: ischemia modified albumin; NO: nitric oxide; ABSU: absorbance unit; AU: arbitrary unit.

Table 4. Plasma SOD, GPX, 8-OhdG, MDA, NO, IMA levels between PASI Groups.

Parameter		Mean ± SD	p value*
SOD	PASI ≤10	5.8±1.6	0.019*
	PASI >10	4.4±1.5	
GPX	PASI ≤10	220.8±93.0	0.760
	PASI >10	200.9±43.9	
8-OHdG	PASI ≤10	36.9±16.4	0.016*
	PASI >10	26.2±10.0	
MDA	PASI ≤10	0.22±0.22	0.271
	PASI >10	0.13±0.15	
IMA	PASI ≤10	0.18±0.04	<0.001*
	PASI >10	0.21±0.05	
NO	PASI ≤10	49.6±39.8	0.007*
	PASI >10	30.6±12.6	

*Mann Whitney U test, results for p<0.05 were considered statistically as significant; SOD: superoxide dismutase; GPx: glutathione peroxidase; 8-OHdG: 8-hydroxy 2-deoxy guanosine; MDA: malondialdehyde; IMA: ischemia modified albumin; NO: nitric oxide; PASI: Psoraisis area and severity index.

and may cause to a serious health problem.[7] The complexion of PS is along with remission and exacerbations. Moreover, it can be triggered by various factors such as trauma, infections or drugs in genetically susceptible individuals.[8] Although it may be observed in all age groups, it peaks in two periods as 20-30 years and 50-60 years. The majority of patients present with the condition before 35 years old.[9]

Although it's etiology has not been fully clarified yet, genetic, immunological, physical and psychological traumas, biochemical changes, environmental factors, infections, smoking and alcohol play a role in the etiology of PS.[10] In recent studies, it has been reported that climate changes, hypocalcemia, pregnancy and cardiovascular diseases may also be factors that facilitate the emergence of PS.[11,12] Oxidative stress is described as the deterioration of the balance between prooxidants and antioxidants in the biological system and turning them in favor of prooxidants. As a result of oxidative stress, human cells usually activate antioxidant systems. When defense systems are not sufficient, free radicals that increase with the balance shifting from antioxidant systems to reactive oxygen compounds can damage cellular macromolecules such as deoxyribonucleic acid (DNA), protein, lipid and carbohydrate and cells by various mechanisms.[13]

From the studies, it has been reported that increased epidermal thickening and impaired tissue structure in PS may occur as a result of oxidative stress and abnormal apoptotic activity. Dysfunction in the antioxidant system and increased reactive oxygen radicals are involved in the pathogenesis of PS.[14] In some of the studies, it was found that increased total antioxidant status (TAS) levels and decreased total oxidant status (TOS) levels were determined in patients with PS.[15] Asha et al.[16] and Paul et al.[17] declared that MDA levels were increased in patients with PS when compared to the control group, as consistent with our study.

A recent study declared that SOD levels were decreased in patients with PS.[18] As shown in Table 3, SOD levels between patient and control group show no significant differences. The main source of endogenous DNA damage is reactive oxygen species (ROS) which are generated from normal cellular metabolism and may leads to the damage product.[19] As the most sensitive and oxidative DNA damage marker, 8-hydroxy-2-deoxyguanosine (8-OHdG) has been established as an important biomarker of oxidative stress.[20] A recent study reported that 8-OHdG levels of the PS patients and control groups did not show differences (as 16781.2±5918.95 and 15276,13±6084.95, respectively (p=0.26)).[21] However, a significant increase in 8-OHdG levels were observed in PS patients with compared to the control group in our study (p=0.002).

Pektas et al.[22] reported that serum IMA levels were significantly elevated in patients with PS compared to the healthy group (0.6±0.1 and 0.4±0.1, respectively p=0.001). In the presented study, IMA and IMA/Albumin show a significant increase in patients with PS when compared to the healthy subjects (Table 3). Also, IMA levels were found to be significant in PASI groups (Table 4). Another study performed by Kaur et al.[23] reported that levels of SOD and GPx were found to be low in PS patients (168.46±51.89 U/ml and 4121.63±1812.53 U/ml respectively) as compared with the controls (237±39.30 U/ml and 8435±1397.54 U/ml respectively) (p<0.001). As similar, in our study levels of GPx were found to be decreased in PS patients when compared to the control group. In a study, it has been revealed that NO levels were significantly higher in patients with PS than the control group.[24] As similar, NO levels were increased in PS patients compared to the control group (p=0.022) in our study.

In conclusion, from the literature, there have been some studies investigating oxidative stress markers and ischemia modified albumin levels in patients with PS, and some of these studies had showed controversial results. As far as we know, there are few articles available to explain the oxidative and antioxidative mechanisms in the etiology of PS. To clarify these conflicting findings, more comprehensive and further studies are needed.

Ethics Committee Approval: This study was approved by the Necmettin Erbakan University Faculty of Medicine Non-Invasive Clinical Research Ethics Committee (Date: 30/07/2021, decision no: 2021/3356). All the participants signed their consents prior the study.

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