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ORIGINAL ARTICLE

The association of serum S100A8-A9 levels with disease activity, radiological findings and laboratory parameters in patients with psoriatic arthritis

Burcu Avşar[1] **Tuba Erdem Sultanoğlu**[1] **Şengül Cangür**[2]
Merve Alpay[3] **Safinaz Ataoğlu**[1]

[1] Department of Physical Medicine and Rehabilitation, Faculty of Medicine, Düzce University. Düzce / Türkiye
[2] Department of Biostatistics and Medical Informatics, Faculty of Medicine, Düzce University. Düzce / Türkiye
[3] Department of Medical Biochemistry, Faculty of Medicine, Düzce University. Düzce / Türkiye

Abstract

S100A8 and S100A9 play a crucial role in immune system functions. S100 proteins are expressed in different cell types and have important roles in regulating various cellular processes and in the haemostasis of the immune system. The aim of this study was to investigate the association between serum S100A8/S100A9 levels and disease-related variables in patients with psoriatic arthritis (PsA), healthy controls, and individuals with psoriasis. 62 PsA patients, 52 healthy controls and 54 psoriasis patients were included. Sociodemographic and clinical characteristic of the groups were recorded. Serum levels of S100A8 and S100A9 were accessed in all groups by ELISA. In the psoriasis group, the S100A8 level was statistically lower and the S100A9 level was statistically higher than in the PsA and control groups. In the PsA group, there was a statistically significant positive correlation between S100A9 level and number of tender and swollen joints and DAPSA score. In the PsA group, there was a statistically significant negative correlation between S100A8 level and number of tender joints, VAS, DAPSA and BASDAI scores. In the PsA group, there was no significant correlation between S100A8-S100A9, CRP and ESR, MSHS and PASRI radiological scores. In psoriasis patients, we found positive correlation between BMI and S100A9 levels. S100A9 level, the number of swollen and tender joints in the PsA group correlated with DAPSA. S100A9 can be used as a new molecule in the follow-up of PsA disease activity, and it may be associated with psoriasis. The lower stability of S100A8 compared to S100A9 and its susceptibility to oxidation suggest that it may not be suitable for diagnosis of PsA. The results were showed that there are limitations regarding the use of these biomarkers in routine diagnosis and follow-up.

Keywords: S100 proteins, disease activity, diagnosis, psoriasis

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Corresponding Author:
Tuba Erdem Sultanoğlu
Email: drtubaerdem@gmail.com

Introduction

Psoriatic arthritis (PsA) is a rheumatic disease, and smoking, trauma, bacterial and viral infections, and obesity are defined environmental and systemic causative factors in the etiopathogenesis of PsA [1-3]. In 61.3-82.3% of patients with PsA, psoriatic skin lesions precede the manifestation of arthritis, while in 10.5-23.8%, skin lesions and arthritis occur simultaneously. In 7.1-14.8%, psoriasis develops after the diagnosis of PsA. The heterogeneous clinic of PsA, the lack of specific laboratory tests and the appearance of psoriatic skin lesions after joint findings in some patients make the diagnosis of PsA difficult [4]. Although numerous biomarkers have been investigated for early diagnosis, disease activity assessment, and treatment monitoring in recent years, no biomarker has yet been identified that is specific and reliable for the diagnosis and follow-up of PsA [5,6]. Recent studies have emphasized the pivotal role of S100 proteins, particularly S100A8, S100A9, and S100A12, in the immunopathogenesis and inflammatory mechanisms of psoriatic arthritis PsA [5,7]. These calcium-binding proteins are expressed by various immune cells and regulate key cellular functions including inflammation, modulation of immune responses, and tissue remodelling [7,8]. Clinical studies have revealed a strong correlation between elevated serum levels of S100 proteins and disease activity in PsA patients, underscoring their potential as biomarkers for diagnosis and treatment monitoring [5,6]. Moreover, combining established diagnostic criteria such as CASPAR with these biomarkers may improve the accuracy of PsA diagnosis and disease management [9,10].

In this study, we aimed to investigate the correlation of S100A8/S100A9 levels with disease activity, radiologic findings and laboratory parameters and to evaluate the potential of this for monitoring inflammatory disease activity.

Materials and Methods

Study design and participants

This study was designed as a case-controlled, and 62 patients with PsA, and 52 healthy controls were enrolled among patients who presented to the physical medicine and rehabilitation outpatient clinics. Patients were recruited from the outpatient clinic between November 2021 and October 2022. Inclusion was based on the classification criteria for PsA established by the CASPAR Study Group [9,10]. 54 patients with psoriasis patients referred from a dermatology clinic, and that they were not diagnosed with PsA. The flow chart of the study was shown in Figure 1. Düzce University Institutional Review Board approval was obtained for the study (Decision no: 2021/233; date: 15.11.2021). Written informed consent was obtained from all participants. The study complied with the ethical standards of the institutional and/or national research committee and the Declaration of Helsinki.

The exclusion criteria were (i) presence of acute or chronic infections, (ii) history of malignancy, (iii) secondary rheumatic diseases associated with PsA, renal failure and liver disease (iv), pregnant and post-partum women. Sociodemographic and clinical characteristics of patients were recorded. The electronic medical records of the PsA patients were analyzed. The radiographs of the patients were evaluated, and radiologic changes were recorded by PASRI and MSHS scoring. The presence of active sacroiliitis in the sacroiliac joints was determined by magnetic resonance imaging (MRI) using 3.0T MR scanners (Magnetom Lumina, Siemens Helthineers, Erlangen, Germany) after intravenous injection of contrast agent (gadobutrol).

Outcome measurement

Pain level in patients with PsA was assessed using a visual analog scale (VAS) [11]. Disease activity in PsA was classified according to the Psoriatic Arthritis Disease Activity Scale [12]. The Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) scores was used to divide the PsA group into active and inactive [13]. Quality of life of the patients were evaluated with Health Assessment Questionnaire (HAQ) and Psoriatic Arthritis Quality of Life Scale (PSAQOL) [14,15]. The Maastricht Ankylosing Spondylitis Enthesitis Scoring (MASES) was used to assess the severity of enthesitis. The total score ranges from 0-13. High scores indicate increased enthesitis load [16]. Radiographic findings in peripheral joints were evaluated with the Modified Sharp-van der Heijde Scoring (MSHS) and axial findings were evaluated with the PsA spondylitis radiological index (PASRI) [17,18]. PASI was calculated to determine the severity of skin findings in patients with psoriasis [18]. S100A8/A9 immunoassay Enzyme-linked immunosorbent assay (ELISA) kits (S100A8 for Sunredbio, Chinese; Catalog N: 201-12-1571; S100A9 Sunredbio, Chinese; Catalog N: 201-12-1572) were used to determine serum levels of the S100A8/S100A9 homodimer for all subjects included in the study, and the manufacturer's instructions were followed.

Statistical analysis

The minimum sample size was calculated with 80% power, 5% significance level and 0.26 effect size, including 49 in the PsA group, 49 in the psoriasis group and 49 in the control group [19,20]. Descriptive statistics were calculated. Shapiro-Wilk test and Levene test were used. One-way ANOVA and Kruskal-Wallis test were used in the comparison between groups. The relationship between variables was calculated with Spearman correlation. Logistic regression analysis was used to determine significant factors. The most appropriate cut-off value was determined with ROC analysis. Pearson chi-square and Fisher-Freeman-Halton (post-hoc Bonferroni test) tests were used to examine the relationship between categorical variables. SPSS 22 was used in statistical analyses, $p<0.05$ was considered significant.

Results

A total of 168 volunteers, including 62 patients with PsA, 54 patients with psoriasis and 52 healthy participants for the control group, were included in to the study. The sociodemographic and clinical characteristics of PsA, psoriasis and control groups are shown in Table 1.

Distribution of clinical features of PsA group are shown in Table 2.

We found a significant difference in S100A8/S100A9 levels among the groups included in the study ($p<0.05$). S100A8 levels were significantly lower in psoriasis patients than in the PsA and control groups. S100A9 was significantly higher in the psoriasis group than in the other groups. When we compared the PsA and control groups, we did not find any significant difference in serum S100A8 and S100A9 levels (Table 3).

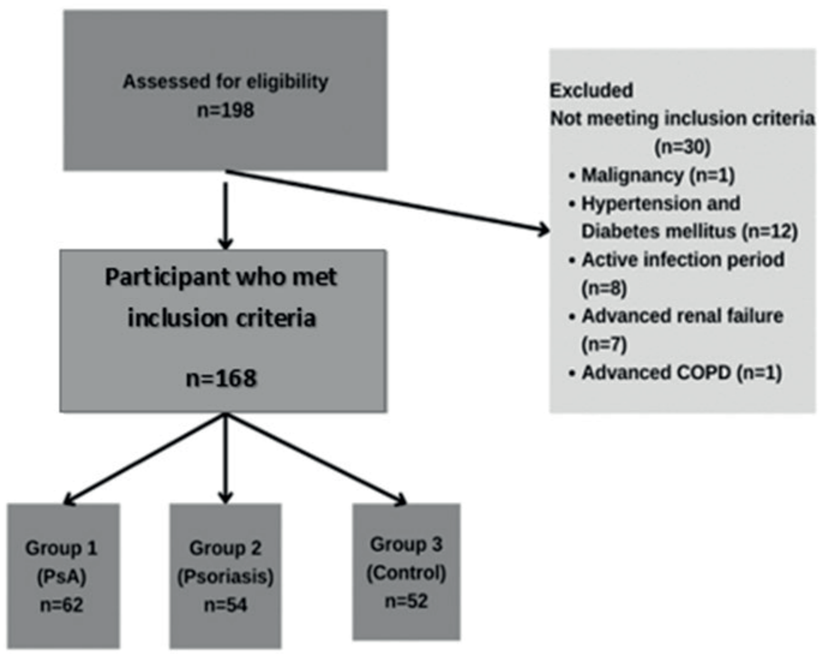

Figure 1. The participant recruitment scheme

Table 1. Sociodemographic and clinical characteristics of PsA, psoriasis and control groups.

		Grup						p
		Psoriatic Arthritis		Psoriasis		Control		
		N	%	N	%	N	%	
Gender	Female	40	64.5	28	51.9	25	48.1	0.202£
	Male	22	35.5	26	48.1	27	51.9	
Age*		43.2±12.1 43 (18)		40.7±12.9 39.5 (18)		40.5±12.4 40 (18)		0.411&
BMI * (kg/m^2)		28.1±6.2 26.6 (8.2)		27.1±5.6 25.4 (8.2)		26.3±4.4 25.6 (4.2)		0.365¥
Educational Status	Literate	2	3.2	5	9.3	1	1.9	**0.007τ**
	Primary School	9	14.5	1	1.9	6	11.6	
	Secondary School	21	33.9	15	27.8	12	23.2	
	High School	21	33.9	26	48.1	15	28.8	
	University	9	14.5	7	13.0	18	34.5	
Marital Status	Single	10	16.1	13	24.1	13	25.0	0.417£
	Married	52	83.9	41	75.9	39	75.0	
Professional Status	Employed	39	62.9	32	59.3	29	55.8	0.690£
	Unemployed	23	37.1	22	40.7	23	44.2	
Smoke	Yes	28	45.2	19	35.2	18	34.5	0.446£
	No	34	54.8	35	64.8	34	65.5	
CRP (mg/dL)*		0.9±1.2 0.4 (1.3)		0.3±0.5 0.2 (0.30)		0.3±0.5 0.2 (0.2)		**0.001¥**
ESR (mm/h)*		20.8±17.6 16 (24)		13.1±11.3 10.5 (13)		15.3±9.9 12 (13)		0.060¥

***BMI,** Body Mass Index; **CRP,** C-reactive protein; **ESR**, Erythrocyte sedimentation rate;*
** Mean±Standard Deviation, Median (Interquartile Range); & One Way ANOVA; **£** Pearson's chi-square test; τ Fisher-Freeman-Halton test; **¥** Kruskal-Wallis*

Table 2. Distribution of clinical features of PsA group.

		N	%
HLA-B27	Pozitive	21	33.9
	Negative	41	66.1
Active sacroiliitis	No	40	64.5
	Unilateral	10	16.1
	Bilateral	12	19.4
Psoriasis Severity	Mild	49	79
	Moderate	10	16.1
	Severe	3	4.8
PsA Developmental Period	Before psoriasis	12	19.4
	At the same time	4	6.5
	After psoriasis	46	74.2
Nail dystrophy	Exist	39	62.9
	Not exist	23	37.1
DAPSA	Remission	17	27.4
	Low Disease Activity	17	27.4
	Medium Disease Activity	20	32.3
	High Disease Activity	8	12.9
HAQ	Mild difficulties and moderate disability	43	69.4
	Moderate to severe disability	17	27.4
	Severe difficulties and very severe disability	2	3.2
BASDAI	Inactive	35	56.5
	Active	27	43.5
Family History	Yes	23	37.1
	No	39	62.9
PsA diagnosis time (year) *		5.3±6.2 3 (7)	
VAS#		4 (5)	
DAPSA*		15.5±14.5 13.5 (20.1)	
PSAQOL*		8±6.5 6 (12)	
HAQ*		0.6±0.6 0.4 (0.9)	
PASRI*		9.1±6.8 7.5 (9.5)	
BASDAI*		3.5±2.4 3.5 (3.4)	
MSHS*		31.5±23.5 27 (3.4)	
MASES*		2.5±3 1 (4)	

***BASDAI,** Bath Ankylosing Spondylitis Disease Activity Index; **DAPSA,** Disease Activity Index for Psoriatic Arthritis; **HAQ,** Health Assessment Questionnaire; **MASES**, Maastricht Ankylosing Spondylitis Enthesitis Score; **MSHS,** Modified Sharp-van der Heijde Scoring; **PASRI,** PsA Spondylitis Radiological Index; **PSAQOL,** Psoriatic Arthritis Quality of Life Scale; **VAS,** Vsual Analog Scale* Mean±Standard Deviation, Median (Interquartile Range); # Median (Interquartile Range)*

Table 4. Comparison of predictive values of serum S100A8, serum S100A9, CRP and ESR for DAPSA and BASDAI scores.

	B	*SE*	*Wald*	*Df*	*P*	*OR*	*OR for %95 Confidence Interval*
S100A8							
S100A8	0	0	0.590	1	0.442	1.000	0.999-1.000
Constant	0.983	0.307	10.275	1	**0.001**	2.673	
Model Significance and Compatibility	χ^2=0.574 sd=1 *p*=0.449 Nagelkerke R^2=0.013 χ^2_{HL}=8.217 sd=8 *p*=0.413						
S100A9							
S100A9	0.048	0.024	4.080	1	**0.043**	1.049	1.001-1.098
Constant	-0.395	0.750	0.277	1	0.599	0.674	
Model Significance and Compatibility	χ^2=4.438 sd=1 ***p*=0.035** Nagelkerke R^2=0.147 χ^2_{HL}=7.261 sd=8 *p*=0.509						
CRP							
CRP	1.521	0.640	5.649	1	**0.017**	4.578	1.306-16.047
Constant	-0.006	0.393	0	1	0.988	0.994	
Model Significance and Compatibility	χ^2=11.336 sd=1 ***p*=0.010** Nagelkerke R^2=0.239 χ^2_{HL}=2.761 sd=8 *p*=0.948						
ESR							
ESR	0.065	0.027	5.781	1	**0.016**	1.068	1.012-1.126
Constant	-0.204	0.474	0.186	1	0.666	0.815	
Model Significance and Compatibility	χ^2=8.867 sd=1 ***p*=0.003** Nagelkerke R^2=0.190 χ^2_{HL}=2.127 sd=8 *p*=0.977						

Df, *Degrees of Freedom;* ***B,*** *Regression coefficient;* ***SE,*** *Standart Error;* ***OR,*** *Odds Ratio;* ***HL,*** *Hosmer Lemeshow test*

Table 3. S100A8 and S100A9 levels in PsA, psoriasis and control groups.

	Group	*Mean*	*Standard Deviation*	*Median*	*Interquartile Range*	*p*
S100A8 (ng/ml)	Psoriatic Arthritis	339.3	825.1	43.2	55.8	**0.003**¥
	Psoriasis	126.2	356.5	35.4	12.1	
	Control	64.1	40.3	54	47.7	
S100A9 (ng/ml)	Psoriatic Arthritis	32.7	16	34.7	31.8	**<0.001**¥
	Psoriasis	46.3	8.6	51.4	9.8	
	Control	24.2	15.4	24.9	23.5	

¥ *Kruskal-Wallis*

S100A9, CRP, ESH levels were found to be effective factors on DAPSA. The increase in S100A9, CRP and ESR levels increased the odds ratio of being active according to DAPSA by 1.049, 4.578 and 1.068 times, respectively. A significant logistic regression model in which we could examine the effect of S100A8, S100A9, CRP, ESR levels on BASDAI could not be obtained (Table 4).

In the PsA group, there was a negative correlation between S100A8 and the number of tender joints and VAS (*p*<0.05) and a positive correlation between S100A9 and the number of tender and swollen joints (*p*<0.05). In the PsA group, there was a negative correlation between S100A8 and DAPSA and BASDAI and a positive correlation between S100A9 and DAPSA (*p*<0.05) (Table 5).

No significant suitable cut-off value was found for the S100A8 value separating the DAPSA (active/inactive) groups (AUC: 0.62 *p*=0.133). The most suitable cut-off value for the S100A9 value separating the DAPSA (active/inactive) group was 34.77 (AUC: 0.72 *p*=0.007). No significant suitable cut-off value was found for the S100A9 value separating the BASDAI (active/inactive) group (AUC: 0.61 *p*=0.195). The most suitable cut-off value for the S100A8 value separating the BASDAI (active/inactive) group was 36.07 (AUC: 0.71).

Table 5. Correlation between S100A8 and S100A9 levels and disease activity and radiographic scores in the PsA group.

		S100A8 (ng/ml)	S100A9 (ng/ml)
BMI (kg/m^2)	r	0.070	-0.058
	p	0.590	0.716
PsA duration (year)	r	0.106	-0.075
	p	0.411	0.636
VAS	r	-0.376	0.200
	p	**0.003**	0.204
Number of tender joints	r	-0.289	0.318
	p	**0.023**	**0.040**
Number of swollen joints	r	-0.184	0.345
	p	0.152	**0.025**
MASES	r	-0.059	0.122
	p	0.650	0.440
PASI	r	-0.059	-0.220
	p	0.649	0.161
MSHS	r	0.057	0.231
	p	0.659	0.142
PASRI	r	-0.013	0.169
	p	0.924	0.296
DAPSA	r	-0.266	0.307
	p	**0.037**	**0.048**
BASDAI	r	-0.325	0.150
	p	**0.010**	0.344
CRP (mg/dL)	r	0.044	0.021
	p	0.737	0.897
ESR (mm/h)	r	0.160	0.120
	p	0.213	0.449
PSAQOL	r	-0.203	0.089
	p	0.113	0.573
HAQ	r	-0.233	0.093
	p	0.068	0.557

***r**: Spearman correlation coefficient*

Discussion

The aim of the study was to investigate the relationship between serum S100A8/S100A9 levels and laboratory parameters, radiological findings and disease activity in PsA patients; and also to investigate the potential of these biomarkers as guiding biomarkers in the diagnosis of PsA and determination of disease activity. The results of the study showed that S100A8 levels were lower and S100A9 levels were higher in the psoriasis group compared to the PsA and control groups; and that there was no significant difference in S100A8/S100A9 levels between the PsA and control groups. While S100A8 levels were negatively correlated with VAS, tender joint count, DAPSA, and BASDAI in the PsA group, S100A9 levels were positively correlated with tender and swollen joint count and DAPSA. There was a positive correlation between BMI and S100A9 levels in psoriasis patients [21,22]. The search for biomarkers associated with early diagnosis and activity of the disease has attracted interest in the literature. However, to our knowledge, a PsA-specific biomarker that can be used in practice has not yet been identified [23-26].

The functional roles of S100A8, S100A9 homodimers, and the S100A8/A9 heterodimer remain controversial in the literature. Some studies have shown that S100A9 has a higher affinity for TLR4 and RAGE receptors compared to S100A8 or the heterodimer, and suggest that S100A9 is the primary active component [27-29]. In contrast, other studies indicate that S100A8 may also be a major contributor to inflammatory signalling but is more susceptible to oxidative degradation [27,30].

In our study, serum S100A8 levels were found to be lower in the psoriasis group. This finding suggests that S100A8 may play a limited role in PsA pathogenesis, although further studies are needed to support this interpretation [34]. In contrast, S100A9 levels were significantly higher in the psoriasis group than in the other groups. Among PsA patients, 54.8% were in remission or had low disease activity, and 61.3% were receiving systemic therapy. Considering that biological DMARDs reduce disease activity and inhibit radiographic progression [31], the relatively low S100A9 levels observed in our PsA group may be attributed to treatment effect and disease control.

Moreover, a positive correlation was observed between serum S100A9 levels and the number of tender and swollen joints in PsA patients. This aligns with previous studies reporting similar correlations in various inflammatory diseases [26,32,33]. On the other hand, serum S100A8 levels showed a negative correlation with tender joint count and patient global assessment (VAS), as well as overall disease activity. This finding is consistent with previous reports that demonstrated variable S100A8 levels across different body fluids and inflammatory conditions [27,34]. These findings suggest that S100A9 may be more strongly associated with joint involvement and disease activity in PsA than S100A8.

In the literature, results of studies investigating the relationship between serum S100A8/A9 heterodimer levels and disease activity scores are contradictory [35,36]. Similar to the study by Mellor et al. [26], in which they evaluated disease activity in PsA with serum S100A9 homodimer levels, we also found a positive correlation between serum S100A9 levels and DAPSA scores in the PsA group. However, according to regression analysis results, serum S100A9 was not superior to CRP and ESR in PsA follow-up. In a study including ankylosing spondylitis patients, no correlation was found between serum S100A8/A9 levels and disease activity or quality of life scores [36]. We also did not find a significant correlation between S100A9 levels and quality of life scores. The lack of a significant correlation between S100A9 levels and quality of life scores suggests that while this protein is associated with inflammation and disease activity, it may not directly reflect patients' quality of life. Quality of life is influenced by a complex interplay of factors including pain, functional impairment, psychosocial aspects, and individual variability. Therefore, S100A9 alone might have limited capacity to fully capture quality of life measures. This finding highlights the importance of a multidisciplinary and patient-centered approach in the assessment of PsA patients alongside biomarker evaluation.

Elwan et al. reported in their studies that there is a correlation between serum S100A8/A9 level and MASES score, presence of enthesitis and subclinical enthesitis in PsA [37]. Unlike our study, no correlation was found between MASES score and S100A9 level. However, the low enthesitis burden and low disease activity of PsA patients participating in our study may explain this result. In a study including AS and non-radiological SpA patients, it was reported that S100A8/A9 levels had no correlation with radiological scores [38]. In our study, we did no find significant correlation between radiological scores and serum S100A8/A9 levels in PsA patients. In PsA, where bone formation and destruction processes proceed together, the evaluation of different findings together in scoring may have concealed the relationship between S100A8/A9 level and radiological findings.

Limitations

The most important limitations of the study were the small number of patients, and the low disease activity of PsA patients who participated in the study. Higher disease activity and lower rates of systemic treatment might provide more insight into the role of this molecule in pathogenesis. Additionally, the psoriasis group exhibited low PASI scores, indicating mild disease severity. Due to these factors, it is challenging to make broad generalizations about the findings.

Conclusion

We think that the specified markers will yield more useful results if the participants are followed longitudinally for a certain period of time and measured during follow-ups. The positive correlation of S100A9 with swollen and tender joints and DAPSA scores in PsA suggests that S100A9 can be used as a new molecule in the follow-up of PsA disease activity, and the high level of S100A9 in psoriasis patients in PsA and control groups suggests that it may be involved in the pathogenesis of psoriasis. The lower stabilization and oxidation susceptibility of S100A8 compared to S100A9 made it unsuitable for use in diagnosis and treatment monitoring. The results of our study, which aimed to identify a new potential biomarker in PsA patients, suggest that there are limitations regarding the use of these biomarkers in routine diagnosis and follow-up.

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Conflict of interest

The authors declare that they have no conflict to interest.

Data availability statement

The data sets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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