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Report

A comprehensive investigation of novel and traditional inflammatory and metabolic markers as predictive indicators in psoriasis

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Abstract

Background Psoriasis is a chronic inflammatory and papulosquamous dermatological disorder. While previous studies have discussed certain inflammatory markers for diagnosing and monitoring psoriasis, there is an absence of comprehensive research encompassing both novel and traditional inflammatory markers, as well as metabolic markers, in relation to psoriasis.

Methods A total of 209 individuals participated, including 54 psoriasis patients and 155 controls. Psoriasis Area Severity Index (PASI) was calculated for the patient group. Potential predictive markers for psoriasis were identified: Uric acid/HDL ratio (UHR), D-dimer/albumin ratio (DAR), fibrinogen/albumin ratio (FAR), erythrocyte sedimentation rate, CRP, WBC, HOMA-IR, and vitamin D levels. Differences between groups and correlations with PASI and each other were analyzed using the Mann–Whitney U test and Spearman correlation coefficient.

Results The results indicate that the patient group exhibited statistically significantly higher levels of UHR, FAR, CRP, WBC, and HOMA-IR. Upon analyzing the correlations between PASI and the identified markers, statistically significant positive correlation with WBC and negative correlation with vitamin D were observed. The correlations of PASI with other markers did not reach statistical significance. It should be underlined that our study was conducted in a predominantly mild-to-moderate patient population.

Conclusions The absence of specificity of these markers for psoriasis limits their practical application. However, the development of new objective measures by using them in combination with specific data such as PASI will provide significant benefits in terms of disease diagnosis, follow-up, and treatment.

Introduction

Psoriasis, a chronic inflammatory papulosquamous skin disease, manifests in individuals across all age groups and affects both genders equally. It presents with diverse clinical phenotypes, including chronic plaque, guttate, pustular, and erythrodermic types. The predominant clinical presentation of the disease is chronic plaque type, which is characterized by symmetrical, red, scaly papules, and plaques.[1]

The severity of psoriasis is often quantified using the widely utilized Psoriasis Area Severity Index (PASI) score. PASI systematically assesses key symptoms, such as erythema, scaling, plaque thickness, and infiltration, considering their specific anatomical locations. This standardized and consistent method reliably reflects the clinical severity of psoriasis in adult individuals.[2]

The precise mechanisms underlying the development of psoriatic lesions are still not fully understood. However, experimental evidence strongly suggests that psoriasis is an autoimmune disorder primarily mediated by T cells.[1] The pathogenesis involves a crucial role played by the chronic interplay between immune cells, proinflammatory cytokines, and the balance of

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Th1/Th2 and Th17/T regulatory cytokines. Additionally, the IL-23/Th17 axis is implicated in this process.[1,3]

The uric acid/HDL ratio (UHR), D-dimer/albumin ratio (DAR), and fibrinogen/albumin ratio (FAR) have been associated with increased inflammation, and it has been proposed that they can also be used as a marker of oxidative stress in chronic inflammatory diseases.[4–9] These ratios have also been recently utilized in assessing intensive care and mortality rates in diverse chronic diseases and COVID-19 infection.[10,11] The evaluation of these ratios in relation to psoriasis and its various aspects, including disease severity, progression, and treatment response, has the potential to enhance our comprehension of the disease's underlying mechanisms and its association with inflammation and oxidative stress.

There are also traditional inflammatory markers proposed to be linked to psoriasis. These markers, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), white blood cell (WBC) count, haptoglobin, fibrinogen, TNF-α, IFN-α, IFN-γ, and various interleukins, have been proposed as potential indicators of the inflammatory processes associated with psoriasis.[12–15] The persistent release of all inflammatory cytokines not only contributes to the chronic inflammation observed in psoriasis but also leads to long-term organ damage.[16]

Psoriasis, classified as a multisystemic chronic inflammatory disorder, exhibits associations with various comorbidities. Among these comorbidities, psoriatic arthritis stands out prominently. Furthermore, metabolic syndrome, Crohn's disease, cancer, depression, diabetes, and atherosclerotic disease represent additional examples of comorbidities frequently observed.[1]

Although the exact mechanism is not known, numerous clinical studies have increasingly shown a connection between psoriasis and metabolic syndrome, including conditions like obesity, hypertension, diabetes mellitus, hyperlipidemia, and lowered HDL.[16,17] Herein, insulin resistance plays a pivotal role as a fundamental mechanism underlying the development of metabolic syndrome.[18] The investigation of the role of Homeostatic Model Assessment Insulin Resistance (HOMA-IR), a significant predictor of insulin resistance, in the context of psoriasis can be considered an important and worthwhile subject for further research.

Both psoriasis and metabolic syndrome exhibit an association with prothrombotic states, which can increase the risk of cardiovascular complications.[19,20] Hence, it is once again important to emphasize the remarkable significance of UHR, DAR, and FAR in elucidating the potential interplay between psoriasis, metabolic syndrome, and cardiovascular complications.

Studies have demonstrated that vitamin D and its analogs play a regulatory role in cellular differentiation, proliferation, and apoptosis. In addition, they can modulate both humoral and cellular immune systems. Consistent findings have revealed significant connections between psoriasis and a deficiency in vitamin D.[21,22] Extensive evidence also supports the safety and efficacy of vitamin D compounds for topical and oral treatment of psoriasis.[21,23] Therefore, the relationship between psoriasis and vitamin D has the potential to be evaluated together in many aspects such as disease severity, progression, and treatment options.

The objective of this study was to explore the relationship between UHR, DAR, and FAR with psoriasis and its severity. Traditional inflammation markers (CRP, ESR, and WBC) as well as metabolic markers (vitamin D, HOMA-IR) were also included in the study. The study aimed to assess the intercorrelations among these markers, offering a comprehensive analysis of their associations.

Materials and methods

Study design and patients

A total of 209 individuals, consisting of 54 patients diagnosed with psoriasis and 155 participants in the control group, were enrolled in the study conducted at the dermatology outpatient clinic of Kirsehir Training and Research Hospital. This study was conducted in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Kirsehir Ahi Evran University (Date: 09.05.2023/No: 2023-09/67). All individuals in the patient and control groups participated in the study voluntarily, and an informed consent form was obtained from all of them. The patient and control groups consisted of individuals without any known inflammatory or metabolic diseases. The control group is composed of individuals who are healthy, without any current complaints, and attend the outpatient clinic for regular monitoring.

In both the patient and control groups, the levels of vitamin D (25-hydroxyvitamin D, ng/ml), fasting blood glucose (mg/dl), fasting insulin (uIU/ml), uric acid (mg/dl), HDL cholesterol (mg/dl), fibrinogen (mg/dl), d-dimer (mg/l), albumin (g/l), hemogram parameters, CRP (mg/l), and ESR (mm/h) were assessed. To evaluate insulin resistance, HOMA-IR was employed. The ratios of uric acid/HDL, D-dimer/albumin, and fibrinogen/albumin were also computed.

In the calculation of PASI, both the severity and extent of the disease are considered simultaneously. The affected areas, including the head, upper limbs, trunk, and lower limbs, were evaluated separately. A standard PASI calculator was used to calculate the PASI score. The final score ranges from 0 to 72, and it was classified as mild (score <5), moderate (score 5–10), or severe psoriasis (score >10).[24]

The determined inflammatory and metabolic markers were compared in the patient and control groups. In addition, the correlations of these markers with each other and with the PASI score were examined.

Individuals under the age of 18, using drugs that may affect the determined markers, and those with inflammatory or metabolic comorbidity were not included in the study.

Statistical analysis

Statistical analysis was performed using SPSS (IBM SPSS Statistics 27) software. Frequency tables and descriptive statistics were utilized to interpret the data, with continuous variables presented as "mean ± standard deviation" and categorical variables as "percentages (%)".

The Kolmogorov–Smirnov test was employed to assess the distribution normality of the parameters. The Mann–Whitney U test was employed to compare the measurement values of more than two independent groups that exhibited non-normal distribution. Within this test, the median values of the parameters were subjected to comparative analysis.
The Spearman correlation coefficient was utilized to assess the relationship between two quantitative datasets that exhibited non-normal distribution. Statistical significance was set as $P < 0.05$.

Results

The study included a total of 209 participants, consisting of 54 patients with psoriasis and 155 healthy individuals serving as the control group. In the control group, there were 134 (86.5%) females and 21 (13.5%) males, while the patient group consisted of 32 (59.3%) females and 22 (40.7%) males. The patients' age ranged from 18 to 72, with a mean age of 40.5. The control group had an age range of 18–65, with a mean age of 23.8. All patients included in our study exhibited the chronic plaque type of psoriasis.

As a result of the Kolmogorov–Smirnov test, it was observed that the PASI scores of the patients did not follow a normal distribution ($P < 0.001$). Based on the boxplot analysis, it can be observed that the 44th and 46th observations are mild outliers, while the 5th and 19th observations are extreme outliers (Figure 1). These observations significantly impact the distribution of the data. The patients demonstrated a predominant distribution within the mild-to-moderate range, as indicated by the PASI scores ranging from 0 to 42, with a median value of 3.75 and an average PASI score of 5.98 (Table 1).

The Kolmogorov–Smirnov test also revealed that none of the parameters in Table 1 exhibited a normal distribution ($P < 0.001$). Therefore, the relationship between the patient and control groups was assessed through the utilization of the Mann–Whitney U test. According to the test results, it was observed that the mean age of individuals in the patient group (5, 40) was statistically significantly higher than the mean age of the control group (8, 23) ($Z = -8.363$; $P < 0.001$). CRP ($Z = -3.370$, $P < 0.001$), WBC ($Z = -2.197$, $P = 0.028$), FAR ($Z = -5.069$, $P < 0.001$), UHR ($Z = -7.269$, $P < 0.001$), and HOMA-IR ($Z = -2.400$, $P = 0.016$) mean values of the patient group were found to be significantly higher than the control group. There was no statistically significant difference in terms of ESR, DAR, and vitamin D mean values (Table 1).

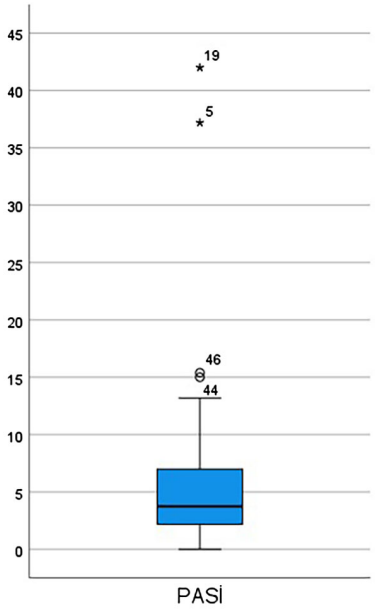

Figure 1 Box plot illustrating the distribution of PASI scores

Due to the non-normal distribution of the parameters in both the patient and control groups, the Spearman correlation coefficient was employed to conduct the correlation analyses. Accordingly, a weak and statistically significant negative correlation was found between the PASI scores and the vitamin D levels of the patients ($r = -0.301$, $P = 0.027$) (Table 2).

A statistically significant weak positive correlation was found between the PASI scores and WBC count of the patients ($r = 0.276$, $P = 0.043$) (Table 2).

Although there was a weak negative correlation between PASI and UHR, and weak positive correlations between PASI and FAR and DAR, these relationships were not statistically significant. Similarly, although there were weak positive relationships between PASI and ESR, CRP, and HOMA-IR, they were not statistically significant (Table 2).

Upon investigating the correlations between the markers in the patient group, it was observed that vitamin D exhibited a statistically significant and weak positive correlation with UHR ($r = 0.275$, $P = 0.044$) (Table 2). However, this correlation was not observed in the control group ($r = 0.042$, $P = 0.602$) (Table 3). All other details are shown in Tables 2 and 3.

Discussion

Psoriasis is an immune-mediated, chronic, recurrent, inflammatory skin disease that is linked to comorbidities associated with early mortality. The disease most often manifests itself in the form of symmetrical, erythematous, scaly papules and plaques called chronic plaque type, and this type comprises approximately 90% of the cases.[1] While our study did not specifically

Table 1 Patient-control group comparisons in terms of parameters

Parameters	Patient-control groups	Mean ± SD	Median [min–max]	Statistical analysis (Mann–Whitney U)
Age (years)	Control	23.80 ± 6.55	22 [18–65]	$Z = -8.363$
	Patient	40.54 ± 14.23	37 [18–72]	***P* < 0.001**
ESR (mm/h)	Control	5.81 ± 4.84	4 [1–30]	$Z = -1.280$
	Patient	7.83 ± 8.14	5 [1–39]	$P = 0.200$
CRP (mg/l)	Control	1.50 ± 3.77	1 [0–36]	$Z = -3.370$
	Patient	4.28 ± 6.88	1 [0–36]	***P* < 0.001**
WBC ($\times 10^3/\mu l$)	Control	7.32 ± 2.11	7.1 [3.23–14.88]	$Z = -2.197$
	Patient	8.05 ± 2.30	7.6 [3.08–13.88]	***P* = 0.028**
FAR	Control	6.13 ± 1.64	6.02 [0.20–19.57]	$Z = -5.069$
	Patient	7.46 ± 1.77	6.96 [4.37–12.68]	***P* < 0.001**
UHR	Control	0.0881 ± 0.1128	0.0655 [0.0219–0.9100]	$Z = -7.269$
	Patient	0.1200 ± 0.0363	0.1245 [0.0556–0.2083]	***P* < 0.001**
DAR	Control	0.0061 ± 0.0106	0.0043 [0.0037–0.1214]	$Z = -1.685$
	Patient	0.0050 ± 0.0022	0.0043 [0.0037–0.1460]	$P = 0.092$
HOMA-IR	Control	3.11 ± 2.26	2.6 [0.5–15.90]	$Z = -2.400$
	Patient	4.06 ± 2.95	3.0 [0.6–14.90]	***P* = 0.016**
Vitamin D (ng/ml)	Control	12.77 ± 7.27	11 [5–64]	$Z = -1.217$
	Patient	11.24 ± 5.16	10 [4–28]	$P = 0.224$
PASI	Patient	5.98 ± 7.61	3.75 [0–42]	

The bold values indicate statistical significance of $P < 0.05$.
CRP, C-reactive protein; DAR, d-dimer/albumin ratio; ESR, erythrocyte sedimentation rate; FAR, fibrinogen/albumin ratio; HOMA-IR, Homeostatic Model Assessment Insulin Resistance; PASI, Psoriasis Area Severity Index; UHR, uric acid/HDL ratio; WBC, white blood cell count.

Table 2 Correlations among parameters in patient group

	Vitamin D (ng/ml)	ESR (mm/h)	WBC ($\times 10^3/\mu l$)	CRP (mg/l)	HOMA-IR	UHR	FAR	DAR
PASI								
r	**−0.301**	0.221	**0.276**	0.206	0.152	−0.199	0.119	0.042
P	**0.027**	0.108	**0.043**	0.134	0.274	0.150	0.390	0.764
Vitamin D (ng/ml)								
r		−0.068	−0.060	−0.176	0.007	**0.275**	0.028	−0.030
P		0.626	0.667	0.203	0.958	**0.044**	0.842	0.830
ESR (mm/h)								
r			0.071	0.250	0.182	−0.152	**0.367**	**0.374**
P			0.609	0.069	0.189	0.273	**0.006**	**0.005**
WBC ($\times 10^3/\mu l$)								
r				**0.366**	0.259	−0.165	0.073	0.172
P				**0.007**	0.059	0.234	0.599	0.213
CRP (mg/l)								
r					0.381	−0.171	**0.384**	0.164
P					0.005	0.216	**0.004**	0.236
HOMA-IR								
r						0.065	0.061	−0.037
P						0.639	0.663	0.793
UHR								
r							−0.154	**−0.297**
P							0.266	**0.029**
FAR								
r								**0.437**
P								**0.001**

The bold values indicate statistical significance of $P < 0.05$.
CRP, C-reactive protein; DAR, d-dimer/albumin ratio; ESR, erythrocyte sedimentation rate; FAR, fibrinogen/albumin ratio; HOMA-IR, Homeostatic Model Assessment Insulin Resistance; PASI, Psoriasis Area Severity Index; *r*, Spearman correlation coefficient; UHR, uric acid/HDL ratio; WBC, white blood cell count.

Table 3 Correlations among parameters in control group

	CRP (mg/l)	WBC ($\times 10^3/\mu l$)	FAR	HOMA-IR	UHR	DAR	Vitamin D (ng/ml)
ESR (mm/h)							
r	**0.158**	−0.100	**0.457**	0.148	−0.127	0.108	0.000
P	**0.048**	0.210	**<0.001**	0.064	0.112	0.177	0.997
CRP (mg/l)							
r		0.222	**0.185**	0.009	0.132	0.002	−0.102
P		0.005	**0.021**	0.907	0.100	0.984	0.205
WBC ($\times 10^3/\mu l$)							
r			**−0.169**	0.008	**0.174**	−0.143	−0.117
P			**0.035**	0.916	**0.029**	0.074	0.145
FAR							
r				0.099	−0.044	0.137	−0.017
P				0.220	0.582	0.087	0.836
HOMA-IR							
r					0.139	0.098	−0.139
P					0.083	0.224	0.083
UHR							
r						0.002	0.042
P						0.984	0.602
DAR							
r							−0.087
P							0.278

The bold values indicate statistical significance of $P < 0.05$.
CRP, C-reactive protein; DAR, d-dimer/albumin ratio; ESR, erythrocyte sedimentation rate; FAR, fibrinogen/albumin ratio; HOMA-IR, Homeostatic Model Assessment Insulin Resistance; *r*, Spearman correlation coefficient; UHR, uric acid/HDL ratio; WBC, white blood cell count.

focus on the selection of patients with chronic plaque type psoriasis, the diagnosis of all participants was in this form.

Psoriasis is associated with multiple comorbidities, such as psoriatic arthritis, Crohn's disease, cancer, depression, obesity, metabolic syndrome, hypertension, hyperlipidemia, diabetes, and atherosclerotic disease.[1,16] Metabolic syndrome stands out as one of the prevailing comorbidities among them. The literature highlights a greater occurrence of metabolic syndrome in individuals with moderate-to-severe chronic plaque psoriasis.[25] Additionally, psoriatic patients have a greater propensity for obesity and diabetes mellitus compared to the general population.[26–28] In this context, insulin resistance may play a pivotal role in the development of comorbidities associated with psoriasis.[29,30]

In our study, we examined the relationship and correlation between HOMA-IR, a known predictor of insulin resistance, and psoriasis, PASI, and other relevant markers. Our objective was to assess the potential of HOMA-IR as a reliable marker for psoriasis.[31,32] Consistent with previous studies, our study revealed a significant increase in HOMA-IR among psoriatic patients ($P = 0.016$). In the study of Bari et al., a statistically significant positive correlation was mentioned between HOMA-IR and PASI.[30] Their study also revealed a notable increase in HOMA-IR among patients with severe psoriasis. In our study, there was a weak positive correlation between HOMA-IR and PASI, but it was not statistically significant ($r = 0.152$, $P = 0.274$). The non-normal distribution of the PASI score in our study, along with most patients presenting with mild-to-moderate cases of psoriasis, may have influenced these findings. Hence, further research is necessary to reevaluate the findings by conducting studies that focus on patient cohorts primarily consisting of individuals with moderate-to-severe psoriasis.

In the study, there was also a positive correlation between HOMA-IR and the inflammatory markers CRP ($r = 0.381$, $P = 0.005$) and WBC ($r = 0.259$, $P = 0.059$) in psoriatic patients. This significant correlation was not observed in the control group (CRP: $r = 0.009$, $P = 0.907$; WBC: $r = 0.008$, $P = 0.916$). In conjunction with HOMA-IR, adipose tissue appears to play a significant role in this relationship. Obesity induces a state of chronic low-grade inflammation, characterized by increased secretion of inflammatory cytokines from adipose tissue.[33,34] The presence of an activated cytokine system may contribute to the elevation of WBC and CRP. The relationship between psoriasis, asymptomatic insulin resistance, and HOMA-IR elevation in our study may indicate a common mechanism involving the adipose tissue. However, it should be noted that the mean age of psoriatic patients was higher than that of the control group, and this may affect the outcome of the study. Finally, when evaluated together with other publications, it can be concluded that although HOMA-IR is not commonly used as a primary marker for psoriasis, it can be utilized as a predictor

in conjunction with other markers, particularly when considering comorbidities.

Vitamin D is a hormone that is synthesized in the skin in response to ultraviolet light and exerts various immunomodulatory and anti-inflammatory effects.[35,36] Due to its immunomodulatory effects, topical vitamin D is utilized in the therapeutic management of psoriasis. Recent studies have reported lower serum vitamin D levels in psoriatic patients compared to healthy individuals.[21,22,36] In a study conducted by Mattozzi et al. involving 141 individuals affected by moderate to severe psoriasis, it was observed that psoriatic patients had significantly lower serum vitamin D levels, and a negative correlation was found between vitamin D levels and the severity of psoriasis.[37] In our study, although vitamin D levels were observed to be low in psoriatic patients, this finding did not reach statistical significance ($P = 0.224$) (Table 1). However, the results of our study demonstrate a statistically significant and weak negative correlation between PASI scores and vitamin D levels ($r = -0.301$, $P = 0.027$). Thus, it can be predicted that the severity of the disease may increase as the vitamin D level decreases in psoriatic patients.

We did not observe a significant correlation between vitamin D levels and other inflammatory markers. However, a statistically significant and weak positive correlation was identified between vitamin D levels and UHR in psoriatic patients ($r = 0.275$, $P = 0.044$) (Table 2). There was no such correlation in the control group ($r = 0.042$, $P = 0.602$). According to the study by Chen et al., it was concluded that increased levels of vitamin D could serve as a predictor of hyperuricemia.[38] However, the existence of conflicting findings in the literature regarding this association suggests that the relationship between vitamin D and uric acid is intricate and warrants further investigation. As a result, if vitamin D and UHR are to be considered as a marker in psoriasis or a predictor in the follow-up of severity, it would be beneficial to assess them together.

Uric acid, derived from purine catabolism, contributes to atherosclerotic diseases and insulin resistance by inhibiting nitric oxide release. There are several potential mechanisms that contribute to this idea, including inflammation, generation of reactive oxygen species, activation of oxidative stress, and other related processes.[39] In general, studies have demonstrated an elevation in uric acid levels among patients with psoriasis.[40,41] Recently, UHR has been reported in studies as an increased marker in inflammatory conditions.[4–7] In our study, there was a statistically significant increase in UHR levels among the patient group compared to the control group ($P < 0.001$) (Table 1). However, there was no statistically significant correlation between UHR and PASI ($r = -0.199$, $P = 0.150$) (Table 2). The predominance of mild-to-moderate cases may have influenced these findings. The negative correlation between UHR and PASI should also be reassessed in severe cases of psoriasis, considering individual and environmental factors. Therefore, further studies with larger patient cohorts are needed to obtain more conclusive results.

Fibrinogen is an essential component of the coagulation cascade and is also an acute phase reactant that reflects the systemic inflammatory state. Albumin is a negative acute phase reactant that tends to decrease in acute situations such as inflammation, trauma, surgery, and burns. The importance of FAR as a predictive and prognostic marker has been increasingly recognized, with published studies demonstrating its association with inflammation and its prognostic value in cardiovascular patients, as well as in patients with stroke and chronic diseases.[9,42] In our study, we investigated the association between FAR and psoriasis and observed a statistically significant elevation in FAR levels among psoriatic patients ($P < 0.001$) (Table 1). However, there was no significant correlation between FAR and PASI ($r = 0.119$, $P = 0.390$) (Table 2). Further investigation of this correlation in individuals with severe psoriasis may lead to divergent findings.

A statistically significant positive correlation between FAR and ESR ($r = 0.367$, $P = 0.006$) and CRP ($r = 0.384$, $P = 0.004$) in psoriatic patients is also noteworthy in the study. Similarly, a significant correlation was also observed in the control group (FAR-ESR: $r = 0.457$, $P < 0.001$; FAR-CRP: $r = 0.185$, $P = 0.021$). It could have been expected that the correlation would be stronger in the patient group. However, the predominance of mild-to-moderate psoriasis cases in our study might have limited the manifestation of such a correlation.

The D-dimer level serves as a criterion for identifying thrombosis, and an increase in its concentration in the bloodstream indicates the ongoing fibrinolysis activity. In various studies, DAR has been reported as a prognostic indicator in patients with critical conditions such as malignancy, subarachnoid hemorrhage, and infection.[8,11,43] Dinic et al. reported high serum D-dimer levels in patients with psoriasis and suggested that psoriasis may be an independent risk factor for early atherosclerosis.[44] In our study, although not statistically significant, psoriatic patients exhibited a lower DAR compared to the control group ($P = 0.092$) (Table 1). Also, there was no statistically significant correlation between DAR and PASI ($r = 0.042$, $P = 0.764$). As previously stated, these findings are primarily applicable to individuals with mild-to-moderate psoriasis. Hence, the present findings could vary with an escalation in the disease severity.

The significant correlation of DAR with ESR ($r = 0.374$, $P = 0.005$), UHR ($r = -0.297$, $P = 0.029$), and FAR ($r = 0.437$, $P = 0.001$) observed in this study also provides valuable insights for future investigations into the association between DAR or D-dimer and psoriasis.

Upon investigating the correlations between PASI and the novel inflammatory markers UHR ($r = -0.199$, $P = 0.150$), FAR ($r = 0.119$, $P = 0.390$), and DAR ($r = 0.042$, $P = 0.764$), it will be observed that UHR exhibits the highest potential for sensitivity in psoriasis, whereas DAR displays the lowest potential for sensitivity. Certainly, to establish definitive conclusions regarding these relationships, it is imperative to initially demonstrate

significant correlations with PASI, which underscores the necessity for comprehensive studies to be conducted in large cohorts.

Since psoriasis is an inflammatory disease, elevation of WBC, ESR, and CRP is generally expected. Wang et al. reported in their study that although there was no significant correlation between WBC and PASI, WBC levels were significantly higher in psoriatic patients.[45] In our study, we observed a significant elevation in WBC levels among psoriatic patients too ($P = 0.028$). Additionally, a statistically significant positive weak correlation was found between WBC levels and PASI ($r = 0.276$, $P = 0.043$).

According to the literature, elevated levels of CRP are commonly found in psoriatic patients and are positively correlated with the severity of the disease.[46] While ESR levels can also be elevated, they are more prominently associated with psoriatic arthritis.[47,48] In our study, we observed a significant increase in CRP levels among psoriatic patients ($P < 0.001$). However, there was no significant difference in ESR levels compared to the control group. In the examination of the correlations between CRP and ESR with PASI, it was observed that both markers exhibited a positive correlation, but these correlations did not reach statistical significance (Table 2).

Conclusion

The evaluation of psoriasis should encompass a comprehensive assessment of multiple factors, including prognosis, potential complications, treatment options, and treatment response. While treatment outcomes, such as PASI 75 and PASI 90, are used as a measure of the degree of improvement attained, it is crucial to incorporate predictive markers that provide more objective and precise outcomes in this assessment.

In our study, we identified UHR, FAR, DAR, ESR, CRP, WBC, HOMA-IR, and vitamin D levels as potential objective predictive markers for psoriasis. The results of our study indicate that the patient group exhibited statistically significantly higher levels of UHR, FAR, CRP, WBC, and HOMA-IR. Upon analyzing the correlations of PASI with identified markers, it was observed that its positive correlation with WBC, and its negative correlation with vitamin D was statistically significant. However, the correlations of PASI with other markers did not reach statistical significance. It is worth noting that PASI scores in our study exhibited a non-normal distribution. As a result, the findings of the study should be interpreted with consideration for their applicability primarily to patients with mild-to-moderate psoriasis.

The absence of the specificity of these markers for psoriasis limits their practical application. However, the development of new objective measures by using them in combination with specific data such as PASI will provide significant benefits in terms of disease diagnosis, follow-up, and treatment. To accomplish this, it is essential to conduct comprehensive studies that involve large cohorts.

Limitations

Due to our inclusion criteria aiming to recruit psoriatic patients who did not have any additional diseases, were not on medication, and did not show another active inflammation, the number of eligible participants in our study did not meet the desired level. As we aimed to include the healthiest individuals possible in the control group, the mean age was lower than that of the patient group. Additionally, the majority of patients exhibited mild-to-moderate psoriasis in our study.

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Data availability statement

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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