



Research Article

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Inflammatory and Hematological Biomarkers as Indicators of Psoriatic Arthritis in Patients Attending Dermatology Clinic

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Abstract

Background: Psoriatic arthritis (PsA), a heterogeneous inflammatory arthropathy where inflammatory biomarkers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) show normal levels in over half of cases in spite of having clinically active disease. Hemoglobin, a readily available parameter, remains a largely underexplored surrogate of systemic inflammatory burden in PsA. **Methods:** Consecutive sampling was used to enroll 132 PsA patients (CASPAR criteria) attending the Dermatology Outpatient Clinics of Al Yarmouk Hospital via a cross-sectional study. Disease activity was stratified based on the Disease Activity Index for PsA (DAPSA) into low (≤ 13), moderate (14–27), and high (> 27). Demographic and lab tests, hematological and inflammatory (full blood count, CRP, and ESR), were measured for all. Anemia was defined by WHO sex-specific thresholds. **Results:** mean age of enrolled cases (46.3±14.3 years; 61.4% female). High disease activity was reported in 58.4% of patients. All hematological and inflammatory indicators show significant differences across DAPSA strata. Hemoglobin fell negatively with rising disease activity (12.80±1.95g/dL in moderate vs. 11.30±1.6g/dL in high activity). Anemia (56.1%) was independently associated with higher DAPSA scores (51.9±39.2 vs. 35.4±40.2; $p < 0.001$), increased acute-phase reactants, and female gender. Psoriasis duration correlated negatively with hemoglobin ($r = -0.28$, $p = 0.001$) but not with CRP or ESR. **Conclusions:** Hemoglobin is a cost-free, available, and clinically meaningful biomarker that matches DAPSA-defined disease activity in PsA more reliably than CRP or ESR in real-world practice.

Keywords: CASPAR criteria; C-reactive protein; DAPSA scores; Erythrocyte sedimentation rate; Psoriatic arthritis.

العلامات الحيوية الالتهابية والدموية كمؤشرات على التهاب المفاصل الصدفي لدى المرضى مراجعي عيادة الأمراض الجلدية

الخلاصة

الخلفية: التهاب المفاصل الصدفي (PsA)، وهو اعتلال مفصلي التهابي غير متجانس حيث تظهر المؤشرات الالتهابية مثل البروتين التفاعلي C ومعدل ترسيب كريات الدم الحمراء (ESR) مستويات طبيعية في أكثر من نصف الحالات رغم وجود مرض نشط سريريا. الهيموغلوبين، وهو معلم متاح بسهولة، لا يزال بديلا غير مستكشف إلى حد كبير للعبء الالتهابي الجهازى في PsA. **الطرائق:** تم أخذ عينات متتالية لتسجيل 132 مريضا من PsA (معايير CASPAR) الذين يراجعون عيادات الأمراض الجلدية الخارجية في مستشفى اليرموك عبر دراسة مقطعية. تم تصنيف نشاط المرض بناء على مؤشر نشاط المرض لاضطراب الكهوف المنكوبة (DAPSA) إلى منخفض (≥ 13)، متوسط (14–27)، ومرتفع (> 27). تم قياس الفحوصات الديموغرافية والمخبرية، والفحوصات الدموية والالتهابية (عد دم كامل، CRP، و ESR)، لجميع الفحوصات. تم تعريف فقر الدم بواسطة عتبات محددة للجنس حسب منظمة الصحة العالمية. **النتائج:** متوسط عمر الحالات المسجلة (46.3±14.3 سنة؛ 61.4% إناث). تم الإبلاغ عن نشاط مرض مرتفع في 58.4% من المرضى. جميع مؤشرات الدم والالتهاب تظهر اختلافات كبيرة بين طبقات DAPSA. انخفض الهيموغلوبين سلبيا مع زيادة نشاط المرض (12.80±1.95 في المتوسط مقابل 11.30±1.6 جرام/ديسيلتر في النشاط العالي). كان فقر الدم (56.1%) مرتبطا بشكل مستقل بدرجات DAPSA الأعلى (51.9±39.2 مقابل 35.4±40.2؛ $p < 0.001$)، وزيادة في تفاعلات المرحلة الحادة، وزيادة الجنس الأنثوي. كانت مدة الصدفية مرتبطة سلبا بالهيموغلوبين ($r = -0.28$ ، $p = 0.001$) ولكن ليس مع CRP أو ESR. **الاستنتاجات:** الهيموغلوبين مؤشر حيوي مجاني ومتاح وذو معنى سريري يتوافق مع النشاط المرضي المعرف بـ DAPSA في PsA بشكل أكثر موثوقية من CRP أو ESR في الممارسة الواقعية.

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INTRODUCTION

Psoriatic arthritis (PsA) is chronic inflammatory arthritis with immune system involvement with well-recognized clinical variability. The condition has peripheral arthritis, enthesitis, and dactylitis in addition to axial involvement, skin, and nail involvement. The prevalence of the condition in the world population with psoriasis is 17.58%. The pooled prevalence in the general population was 112 per 100,000 [1,2]. The prevalence of the condition in the world population has been noted to be higher in

Europe and America compared to Asia and South America [3]. Beyond joints, PsA also has an inflammatory burden linked to cardiovascular diseases, metabolic syndromes, and structural damage. Accurate disease activity evaluation is central to the treat-to-target paradigm of affected cases [4]. The Disease Activity index for PsA (DAPSA) has been noted to be the preferred and validated disease composite measure [5]. Nonetheless, the validity of conventional biomarkers in accurately measuring the inflammatory load in PsA is fundamentally questionable. It is worth noting that C-reactive protein

(CRP) and erythrocyte sedimentation rate (ESR) are elevated in less than ½ of PsA patients, even in the existence of obvious clinical disease activity [6]. Moreover, there is inconsistency in the reporting of inflammatory indicators across published literature, particularly in established disease or in the context of biologic therapy [7]. The need for an effective surrogate marker of inflammatory load remains an important unmet need [8]. Consequently, hematological parameters like WBC and platelets are often investigated as markers owing to their clinical feasibility [9]. Hemoglobin levels represent an unexplored aspect of managing PsA; although anemia of chronic diseases is commonly encountered in PsA, its correlation with DAPSA-defined disease activity levels and its potential as a biomarker have not yet been investigated in Arab-region PsA populations [10]. PsA is an enigmatic condition with poor management in Africa and the Middle Eastern regions, especially considering the lack of information in the Iraqi population [11]. The study aimed at investigating the hematological and inflammatory biomarkers, including full blood count parameters, levels of hemoglobin, CRP, ESR, and platelet count, across various validated DAPSA-defined disease activity levels in order to identify independent predictors of disease burden in a real-world cohort of Iraqi PsA patients.

METHODS

Study design and setting

A cross-sectional study employed consecutive sampling and took place at the Dermatology Clinics of Al Yarmouk Hospital in Baghdad, Iraq, from January to October 2025. Study ethical approval was granted from Mustansiriyah University's institutional review board (IRB No. 122; 24th December 2024). All patients gave informed consent before embarking on the study. PsA patients with confirmed diagnosis based on CASPAR criteria were included in the final analysis [12].

Participant eligibility criteria

Adult patients attending the dermatology clinic were invited into the study after explaining the study's aim and methods. The study inclusion criteria were 1) a diagnosis of psoriatic arthritis based on the CASPAR criteria [12], 2) patients aged 18 years and above, 3) patients with an illness duration of at least six months, and 4) patients with all clinical and lab information available.

Exclusion criteria

Patients were excluded in the case of 1) patients with blood-related diseases, 2) patients with an active bacterial, viral, or fungal infection, 3) patients with other autoimmune and connective diseases aside from psoriatic arthritis, 4) patients with cancer, 5) patients with chronic kidney diseases with an eGFR less than

60 mL/min/1.73 m², 6) patients with liver diseases, and 7) patients who are pregnant or breastfeeding.

Disease activity assessment

Disease activity was stratified according to the Disease Activity Index for Psoriatic Arthritis (DAPSA) and categorized as low (≤ 13), moderate (14–27), or high (> 27), per Schoels *et al.* (2016) [13]. Skin disease severity was determined using the Psoriasis Area and Severity Index (PASI) [14]: mild (< 10), moderate (10–20), and severe (> 20). Axial involvement, dactylitis, and HLA-B27 status were recorded for all patients.

Laboratory assessment

Venous blood was taken in the morning following an overnight fast (07:00–10:00 hours). Measurement of hemoglobin (Hb), white blood cells (WBCs), and platelets (PLTs) was done in an automated analyzer (NORMA Icon 3 hematology analyzer). Immunoturbidimetry of serum C-reactive protein (CRP) was carried out, and the erythrocyte sedimentation rate (ESR) was done using the Westergren method in the Central Laboratory of Al Yarmouk Teaching Hospital.

Statistical analysis

Continuous data are expressed as mean \pm SD. Categorical data was expressed as frequency (%). Between-group differences across DAPSA strata were calculated by one-way ANOVA with LSD post-hoc correction. Bivariate association was calculated by Pearson's correlation coefficient (r). Between-group comparisons of criteria by anemia status were performed via the Mann–Whitney U test for continuous variables and the chi-square test for categorical data. Statistical significance was set at $p < 0.05$. All analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY).

RESULTS

One hundred and thirty-two PsA patients were enrolled; they had a mean age of 46.3 ± 14.3 years (61.4% female). Most patients had high disease activity (DAPSA > 27 : 58.4%) (Table 1). The majority of cases had mild skin disease (PASI < 10 : 60.7%). Axial involvement was found in 28.8% and dactylitis in 17.4%. HLA-B27 was positive in 38.6% of tested patients (see Table 1 and Figure 1). In Table 2, all clinical and hematological parameters demonstrated significant differences across DAPSA strata (all have $p < 0.05$). Tender and swollen joint counts, patient and physician global assessments, CRP, ESR, and PLT worsened progressively with disease activity severity. Most striking between-group differences were observed in CRP (1.70 ± 1.14 vs. 36.21 ± 39.91 mg/L; low vs. high activity) and ESR (20.66 ± 17.55 vs. 60.77 ± 29.78 mm/hr).

Table 1: basic demographic and clinical criteria of the study cases (n= 132)

Characteristic	Value
Age, (years)	46.26±14.30 [18-84]
<i>HLA-B27</i>	
Not done	51(38.6)
Positive	51(38.6)
Negative	30(22.8)
<i>Gender</i>	
Male	51(38.6)
Female	81(61.4)
<i>Axia</i>	
Yes	38(28.8)
No	94(71.2)
<i>Dactylitis</i>	
Yes	23(17.4)
No	109(82.6)

Values are presented as frequency, percentage, and mean±SD. Disease activity was assessed using the Disease Activity index for PsA (DAPSA). Following validated criteria (Schoels *et al.*, 2016) [13], scores are grouped into low (≤13), moderate (14–27), and high (> 27) activity states.

Table 2: consolidated clinical and lab criteria by disease activity group

Variable	Group ≤13 (n=15)	Group 14-27 (n=40)	Group >27 (n=77)	p-value
Tender joint count (TJC 68)	2.0±1.51 ^b	3.35±1.7 ^b	9.24±6.81 ^a	<0.05
Swollen joint count (SJC 66)	0.2±0.41 ^b	1.07±1.32 ^b	4.0±3.9 ^a	<0.05
Patient Assessment	3.86±1.72 ^c	6.25±1.59 ^b	8.02±1.66 ^a	<0.05
Physician Assessment	2.53±1.24 ^c	4.85±1.76 ^b	7.0±1.87 ^a	<0.05
C-reactive protein (mg/L)	1.7±1.14 ^b	3.85±2.5 ^b	36.21±39.91 ^a	<0.05
ESR (mm/hr)	20.66±17.55 ^b	26.4±19.13 ^b	60.77±29.78 ^a	<0.05
Hemoglobin (g/dL)	12.2±2.07 ^{ab}	12.8±1.95 ^a	11.3±1.6 ^b	<0.05
White Blood Cell Count (x10 ⁹ /L)	7.06±3.79 ^b	7.7±2.56 ^{ab}	8.7±3.1 ^a	<0.05
Platelet Count (x10 ⁹ /L)	252±67.49 ^b	278.75±95.96 ^b	350.54±128.44 ^a	<0.05

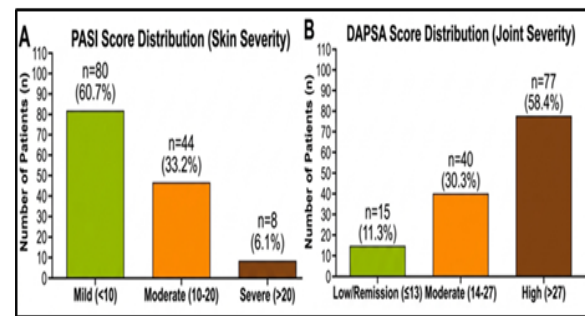
Values are presented as mean±SD. Grouping was done in accordance with Disease Activity index for PsA (DAPSA). Means within a row sharing a common superscript letter (a,b,c) are not significantly different ($p > 0.05$) according to the LSD test. Different letters indicate statistically significant differences. Non-significant variables (Ps duration and PsA duration) have been omitted for clarity and focus on significant findings. ESR: Erythrocyte Sedimentation Rate; LSD: Least Significant Difference.

Patients' age correlated positively with both psoriasis duration ($r = 0.58$, $p < 0.001$) and PsA duration ($r = 0.32$, $p < 0.001$). Psoriasis duration was inversely associated with hemoglobin ($r = -0.28$, $p = 0.001$), indicating that extended disease chronicity is associated with greater suppression of erythropoiesis.

Table 3: Correlations with disease duration

Parameter	Duration of psoriasis (r)	p-value	Duration of psoriatic arthritis (r)	p-value
Age	0.582	<0.001	0.318	<0.001
Tender joint count	0.165	0.059	0.106	0.225
Swollen joint count	0.114	0.192	-0.012	0.894
Pain assessment	0.108	0.220	0.102	0.244
C-reactive protein	0.085	0.333	0.026	0.766
Erythrocyte sedimentation rate	0.121	0.166	0.103	0.239
Hemoglobin	-0.284	0.001	-0.145	0.096
White Blood Cell Count	-0.007	0.941	0.031	0.725
Platelets	0.031	0.725	0.014	0.871

To further characterize the relationship between hemoglobin and disease activity, patients were stratified by WHO-defined anemia criteria (Table 4). Anemic cases (n= 74; 56%) demonstrated significantly higher DAPSA scores vs. non-anemic patients (51.9 ± 39.2 vs. 35.4 ± 40.2 ; $p < 0.001$). A significant sex imbalance was noticed, with a higher proportion of females in the anemic group (74.3% vs. 44.8%; $p = 0.001$). Additionally, anemic cases exhibited significantly elevated ESR, CRP, and platelet count; $p < 0.001$; $p = 0.025$, and $p = 0.025$, respectively). The WBC showed no significant difference between groups ($p = 0.133$).

**Figure 1:** Comparison of Disease Severity in PsA (n=132) Assessing Skin (PASI) and Joint (DAPSA) Activity. **A)** Psoriasis Area and Severity Index (PASI), **B)** Disease Activity index for Psoriatic Arthritis (DAPSA).

Hemoglobin showed an inverse pattern, with the lowest levels recorded in the high-activity group (11.30 ± 1.60 g/dL).

No significant correlations were identified between disease duration and acute inflammatory markers (CRP and ESR) or joint counts, suggesting that inflammatory burden is driven by current disease activity rather than cumulative disease exposure (Table 3).

DISCUSSION

The current study has shown that hematological and inflammatory markers are significantly different based on PsA disease activity. The most notable difference was the levels of CRP and ESR. Moreover, an inverse correlation between hemoglobin levels and disease burden was confirmed. Anemia was independently correlated with high DAPSA, high levels of acute-phase reactants, and a female predominance. This implies a direct correlation of anemia with the levels of systemic inflammation among PsA cases. There was an inconsistency of conventional biomarkers in PsA, which is well-

supported in clinical practice. In the Hackett *et al.* cohort, 2/3 of PsA patients have normal CRP values, and despite their CRP status, they showed a total

discrepancy with the cDAPSA and MDA classifications, with Cohen's kappa values of -0.21 to -0.26 [15].

Table 4: Stratification of the study cases based on anemia status and inflammatory and hematological biomarker distribution

Variable	Anemic (n=74)	Non-Anemic (n=58)	p-value
DAPSA score	51.9±39.2	35.4±40.2	<0.001
Haemoglobin (g/dL)	10.63±1.16	13.44±1.39	<0.001
C-reactive protein (mg/L)	26.3±31.9	17.6±37.4	0.025
ESR (mm/hr)	54.3±30.2	35±29.4	<0.001
Platelet count ($\times 10^9/L$)	339.9±125	289.1±107.7	0.025
White Blood Cell Count ($\times 10^9/L$)	8.61±3.31	7.71±2.69	0.133
Sex (F/M), n	55/19	26/32	0.001

A recent review by Queiro *et al.* discussed that CRP frequently fails to detect low-grade or residual disease activity, mostly in cases with established disease or those receiving biologic therapy. On the other hand, ESR appears to encompass broader and more chronic disease burdens, such as structural damage and functional limitations [7]. Wirth *et al.*'s systematic review and meta-analysis of 124 PsA biomarker studies discussed that there are no specific recommended diagnostic biomarkers for PsA and advised further work to measure the performance that discriminates PsA from other inflammatory conditions [16]. The inconsistency in reporting may be attributed to the intrinsic biological heterogeneity of PsA. Cytokine-driven inflammation in PsA is frequently compartmentalized to synovial, enthesial, and cutaneous microenvironments [17]. Additionally, there is systemic spillover of acute-phase reactants, which underlies the rise in CRP and ESR that variably occurs in an incomplete manner even in the presence of active synovial inflammation [18]. Moreover, interleukin-17 and interleukin-23, dominant cytokines implicated in the pathogenesis of PsA, are relatively ineffective stimulators of the hepatic acute-phase response compared with the TNF- α and IL-6-driven inflammation found in rheumatoid arthritis, where IL-6 dominates. The patient with PsA exhibits active synovitis with near-normal CRP levels [19]. The analysis identified anemia in 56.1% of our cohort — a prevalence consistent with reported rates in inflammatory arthritis, where sustained cytokine activation suppresses erythropoiesis. IL-6 is the dominant driver of this process, stimulating hepatic hepcidin production via STAT3 signaling, which sequesters iron in reticuloendothelial cells and renders it unavailable for red cell synthesis [20]. The resulting anemia of inflammation is typically mild-to-moderate, normochromic, and normocytic, characterized by low circulating iron alongside normal or elevated ferritin and hepcidin [21]. In this study, anemic patients exhibited significantly higher DAPSA scores ($p < 0.001$) alongside elevated ESR, CRP, and platelet counts, a pattern consistent with anemia reflecting cumulative inflammatory burden rather than nutritional deficiency. This parallel was reported in both rheumatoid and PsA, where proinflammatory cytokines—principally TNF- α and IL-6—temper iron metabolism and suppress erythropoiesis in the bone marrow. Similarly, anti-cytokine treatment reverses hemoglobin suppression, combined with improving disease activity markers [22]. The current analysis

confirmed a significant sex imbalance in the anemic group (female predominance, $p = 0.001$). This may be partly attributable to the lower sex-specific WHO hemoglobin threshold [23]. Another explanation is the biological amplification. Sustained IL-6-mediated hepcidin elevation gradually worsens erythropoietic suppression in females with long-term disease [24]. Hemoglobin was negatively correlated with psoriasis duration ($p = 0.001$), which supports progressive erythropoietic suppression as the disease becomes more chronic. In excellent agreement with our results, anemia was similarly linked with higher disease activity scores in spondyloarthritis, reinforcing its role as a marker of inflammatory disease state across spondyloarthropathy cases [25]. Notably, neither CRP nor ESR correlated significantly with psoriatic arthritis disease duration (all have $p > 0.05$), which implies that systemic inflammatory burden in this study reflects current disease activity rather than collective exposure. This divergence between chronicity and acute-phase markers compared to significant hemoglobin association with disease duration supports hemoglobin as a cumulative marker of the inflammatory load rather than a transient snapshot of acute inflammatory activity [26]. The current cohort found anemia in 56% of the patient group, directly related to DAPSA disease severity, thus confirming the use of hemoglobin as a surrogate marker for inflammation. Hemoglobin, readily accessible in routine complete blood counts without additional cost, has an underappreciated potential for monitoring inflammatory arthritis. Critically, it is used not just as a passive marker of disease burden. The low hemoglobin level at baseline is predictive of greater radiographic progression. Moreover, hemoglobin levels tend to increase with effective management of inflammation across all DMARD categories, as discussed by the Weinblatt BRASS Registry [27]. IL-6 reduction also increases hemoglobin levels concomitantly with improving the clinical picture. This is supported by three phase III trials of sarilumab [28].

Study Limitations

A longitudinal multicentric study may capture the role of inflammatory and hematological parameters in more depth. From a clinical perspective, we did not address axial or enthesitis involvement, nor did we address therapeutic stratification of received drugs by enrolled cases (i.e., biologic vs. conventional

DMARD), which may confound biomarker assessment. To date, no specific diagnostic biomarkers have been established for PsA [29]. Prior works discussed that CRP is frequently normal despite clinically active disease, so relying solely on CRP may underestimate the true inflammatory load in those patients.

Study Strength

There is a lack of regional data that addresses the disease in Iraqi patients, so this work addresses a geographic knowledge gap that may form the foundation of longer studies. The study adopted a real-world observational design with relevant clinical data encompassing a full hematological panel across patients' strata, enabling generalization of its results in practice. We have used a validated disease-specific instrument, DAPSA, rather than relying on generic scores. The main strength of this work lies in the identification of hemoglobin as a strong independent disease activity biomarker that outperformed CRP and ESR—a parameter that is both practical, accessible, and cost-free as a surrogate of the inflammation burden of PsA.

Conclusion

The current study indicated that traditional inflammatory biomarkers are inadequate for assessing the complete inflammatory burden of patients with PsA. Conversely, hemoglobin, a universally available free complete blood count parameter, was found to be a significant independent biomarker for disease activity in patients with PsA. Its levels inversely correlate with DAPSA severity in a stronger and more consistent association than inflammatory indicators. The presence of anemia in patients with PsA is no longer considered a coincidental occurrence but a significant measurable parameter for systemic inflammation. This parameter could be especially important for evaluating patients, particularly in healthcare settings with limited resources and restricted access to advanced disease biomarkers.

Conflict of interests

The authors declared no conflict of interest.

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Data sharing statement

Supplementary data can be shared with the corresponding author upon reasonable request.

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