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Clinical-Metabolic and Inflammatory Correlates of Psoriatic Arthritis in Patients with Severe Psoriasis

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Abstract

Background: Psoriatic arthritis (PsA) is a chronic inflammatory musculoskeletal disease that develops in a substantial proportion of patients with psoriasis, particularly those with severe disease. Increasing evidence suggests that clinical severity, metabolic dysfunction, and systemic inflammation may contribute to the transition from cutaneous psoriasis to articular involvement; however, their relative contributions remain incompletely defined.

Aim: To evaluate the clinical, metabolic, and inflammatory correlates of PsA in patients with severe psoriasis, with particular emphasis on disease duration, adiposity measures, and systemic inflammatory status.

Material and methods: This analytical cross-sectional study included 100 adult patients with severe psoriasis, of whom 60 had clinically confirmed PsA and 40 had no joint involvement. Data were collected on demographic characteristics, disease duration, anthropometric measures (body mass index [BMI] and waist circumference), and inflammatory markers (erythrocyte sedimentation rate [ESR]). Group comparisons were performed using independent *t*-tests, and multivariate logistic regression analysis was conducted to identify independent predictors of PsA. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs).

Results: Patients with PsA had a significantly longer disease duration compared to those without PsA (12.0 vs 6.4 years; $p < 0.0001$). BMI (28.9 vs 26.6 kg/m^2; $p = 0.00047$) and waist circumference (91.7 vs 85.4 cm; $p = 1.20 \times 10^{-7}$) were significantly higher in the PsA group. ESR levels were also markedly elevated in patients with PsA (19.6 vs 11.0 mm/hr; $p = 8.61 \times 10^{-14}$). In multivariate analysis, disease duration (OR 5.49, 95% CI 1.30–23.15; $p = 0.020$), waist circumference (OR 1.37, 95% CI 1.05–1.79; $p = 0.022$), and ESR (OR 1.60, 95% CI 1.13–2.27; $p = 0.008$) remained independent predictors of PsA, whereas BMI and age were not statistically significant after adjustment.

Conclusion: Psoriatic arthritis in patients with severe psoriasis is independently associated with prolonged disease duration, central obesity, and increased systemic inflammation. Waist circumference appears to be a more clinically relevant metabolic predictor than BMI, underscoring the role of visceral adiposity in disease pathogenesis. These findings support the implementation of integrated clinical and metabolic assessment strategies to facilitate early identification of patients at increased risk of PsA.

Keywords: Psoriasis; central obesity; waist circumference; erythrocyte sedimentation rate; metabolic syndrome.

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Introduction

Psoriasis is a chronic, immune-mediated inflammatory disorder increasingly recognised as a systemic disease with multisystem involvement extending beyond the skin [1]. Among its extracutaneous manifestations, psoriatic arthritis (PsA) is of particular clinical importance, affecting up to one-third of patients and contributing to progressive joint damage, functional disability, and reduced quality of life [2,3]. The risk of PsA is notably higher in individuals with severe psoriasis, suggesting that the magnitude and persistence of cutaneous inflammation—often quantified using indices such as the Psoriasis Area and Severity Index (PASI)—may reflect an underlying systemic inflammatory burden predisposing to musculoskeletal involvement [4].

The pathogenesis of PsA is complex and multifactorial, involving a dynamic interaction between genetic predisposition, environmental triggers, and immune dysregulation [5]. A positive family history has been consistently associated with increased susceptibility, underscoring the contribution of heritable factors to both psoriasis and PsA development [6]. In parallel, disease duration has emerged as a clinically relevant parameter, with longer-standing psoriasis associated with a higher cumulative risk of transition to PsA, possibly reflecting prolonged exposure to systemic inflammation and immune activation [7].

In recent years, increasing attention has been directed toward the role of metabolic comorbidities in psoriasis and PsA. Patients with severe psoriasis frequently exhibit features of metabolic syndrome, including obesity, type 2 diabetes mellitus, and hypertension [8]. Anthropometric indices such as body mass index (BMI) and waist circumference provide important measures of generalised and central adiposity, respectively, both of which are closely linked to systemic inflammation [9]. Adipose tissue functions as an active immunometabolic organ, secreting pro-inflammatory cytokines and adipokines that may potentiate the IL-23/IL-17 axis and tumour necrosis factor (TNF)-mediated pathways implicated in PsA pathogenesis [10]. Consequently, obesity and related metabolic disturbances are not only comorbid conditions but may also act as drivers of disease severity and joint involvement.

Inflammatory markers further contribute to understanding the systemic nature of psoriasis and its progression to PsA. The erythrocyte sedimentation rate (ESR), a widely available indicator of systemic inflammation, has been associated with both disease activity and the presence of joint involvement in psoriatic disease [11]. Elevated ESR levels may reflect heightened inflammatory activity that bridges cutaneous and articular manifestations. When considered alongside clinical parameters such as PASI score and disease duration, as well as metabolic indicators including BMI, waist circumference, diabetes status, and hypertension, ESR may provide valuable insight into the inflammatory milieu associated with PsA development.

Despite these advances, the integration of clinical severity indices, metabolic parameters, and inflammatory markers into a unified framework for predicting PsA in patients with severe psoriasis remains insufficiently defined. In particular, the interplay between demographic variables (such as age and sex), disease-specific factors (including duration and family history), metabolic comorbidities, and systemic inflammation warrants further investigation in well-characterised patient cohorts.

Therefore, the present study is designed to evaluate the clinical, metabolic, and inflammatory correlates of psoriatic arthritis in patients with severe psoriasis, with specific emphasis on PASI-defined disease severity, duration of illness, familial predisposition, anthropometric measures (BMI and waist circumference), metabolic comorbidities (diabetes and hypertension), and systemic inflammatory status as reflected by ESR. By delineating these associations, the study aims to enhance risk stratification and facilitate earlier identification of patients at increased risk for PsA.

Materials and methods

Study design and sampling

This was a hospital-based analytical study designed to evaluate the clinical, metabolic, and inflammatory correlates of psoriatic arthritis (PsA) among patients with severe psoriasis. The study included a total of 100 adult patients diagnosed with severe psoriasis, recruited consecutively from a tertiary dermatology centre. All participants underwent systematic clinical and laboratory evaluation.

Patients were stratified into two groups based on the presence of psoriatic arthritis:

- Psoriasis with PsA (n = 60)
- Psoriasis without PsA (n = 40)

The diagnosis of PsA was established based on clinical rheumatological assessment, supported by standard classification criteria where applicable.

Inclusion criteria

- Adults aged ≥18 years
- Clinically confirmed severe psoriasis
- Availability of complete clinical, metabolic, and laboratory data

Exclusion criteria

- Coexisting autoimmune or inflammatory arthritis
- Acute or chronic inflammatory conditions unrelated to psoriasis
- Incomplete dataset variables

Statistical analysis

Data were analysed using Statistical Package for the Social Sciences (SPSS) 26. Continuous variables were expressed as mean ± standard deviation (SD). Comparative analyses between PsA and non-PsA groups were performed using Independent samples t-test for normally distributed continuous variables and Chi-square test for categorical variables. Multivariate logistic regression analysis was conducted to identify independent predictors of PsA, incorporating clinically relevant variables such as age, sex, disease duration, BMI, waist circumference, diabetes, hypertension, and ESR. A two-tailed p-value <0.05 was considered statistically significant with coefficient interval 95% CI.

Ethical considerations

The study protocol was approved by the institutional ethical review board of Hammurabi college of medicine/University of Babylon dated 20th October 2025. All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment, and confidentiality of patient data was strictly maintained.

Results

A total of 100 patients with severe psoriasis were included in the final analysis. Of these, 60 patients (60%) were diagnosed with psoriatic arthritis (PsA), while 40 patients (40%) had no clinical or rheumatological evidence of joint involvement. The baseline clinical and demographic characteristics of the study population are presented in Table 1. The mean age of patients with PsA was 36.4 ± 5.3 years, compared with 34.2 ± 4.7 years in the non-PsA group. Although this difference reached nominal statistical significance (p = 0.0358), its effect size was modest and did not persist in multivariate modelling, suggesting limited clinical relevance. In contrast, disease duration demonstrated a pronounced and statistically robust association with PsA. Patients with PsA had a significantly longer duration of psoriasis (12.0 ± 3.1 years) compared to those without PsA (6.4 ± 2.1 years), with a highly significant difference (p < 0.0001). This finding indicates a strong temporal relationship between chronicity of cutaneous disease and the development of articular involvement.

Table 1. Clinical and metabolic characteristics of patients with and without PsA.

Variable	PsA (n=60), Mean ± SD	Non-PsA (n=40), Mean ± SD	p-value
Age (years)	36.4 ± 5.3	34.2 ± 4.7	0.0358
Duration (years)	12.0 ± 3.1	6.4 ± 2.1	<0.0001
BMI (kg/m²)	28.9 ± 2.6	26.6 ± 2.7	0.00047
Waist circumference (cm)	91.7 ± 5.6	85.4 ± 4.7	1.20×10^{-7}
ESR (mm/hr)	19.6 ± 4.8	11.0 ± 4.7	8.61×10^{-14}

Values are presented as mean ± standard deviation (SD). Comparisons between groups were performed using independent t-tests. P-values represent two-tailed significance levels.

Metabolic characteristics

As shown in Table 1, metabolic parameters differed significantly between the two groups. Body mass index

(BMI) was significantly higher in patients with PsA (28.9 ± 2.6 kg/m²) compared to those without PsA (26.6 ± 2.7 kg/m²; p = 0.00047), suggesting an association between increased body mass and PsA. More notably, waist circumference—an indicator of central adiposity—was markedly elevated in the PsA group (91.7 ± 5.6 cm) relative to the non-PsA group (85.4 ± 4.7 cm), with a highly significant difference ($p = 1.20 \times 10^{-7}$). The magnitude and strength of this association highlight the potential role of visceral fat distribution in the pathogenesis of PsA.

Inflammatory marker

A substantial difference was observed in systemic inflammatory burden, as reflected by ESR levels (Table 1). Patients with PsA exhibited significantly higher ESR values (19.6 ± 4.8 mm/hr) compared to patients without PsA (11.0 ± 4.7 mm/hr), with a highly significant difference ($p = 8.61 \times 10^{-14}$). This finding supports the presence of heightened systemic inflammation in patients with concomitant joint disease.

Multivariate analysis

To identify independent predictors of PsA, a multivariate logistic regression model was constructed incorporating clinically relevant variables (Table 2). Disease duration emerged as a strong independent predictor of PsA (OR 5.49, 95% CI 1.30–23.15; p = 0.020), reinforcing the importance of prolonged disease exposure. Waist circumference also remained independently associated with PsA (OR 1.37, 95% CI 1.05–1.79; p = 0.022), whereas BMI lost statistical significance after adjustment (OR 0.81, 95% CI 0.39–1.67; p = 0.567). This suggests that central obesity, rather than overall adiposity, is more closely linked to the risk of PsA. Similarly, ESR retained independent significance (OR 1.60, 95% CI 1.13–2.27; p = 0.008), indicating that systemic inflammation plays a key role in the development of psoriatic joint disease. Age was not independently associated with PsA (OR 1.19, 95% CI 0.85–1.66; p = 0.321), consistent with its limited effect observed in univariate analysis.

Table 2. Multivariate logistic regression analysis of predictors of PsA

Variable	OR	95% CI	p-value
Duration	5.49	1.30–23.15	0.020
Waist circumference	1.37	1.05–1.79	0.022
ESR	1.60	1.13–2.27	0.008
BMI	0.81	0.39–1.67	0.567
Age	1.19	0.85–1.66	0.321

Odds ratios (ORs) were derived from multivariate logistic regression analysis. Confidence intervals (CI) represent 95% intervals. Variables were entered simultaneously into the model.

The independent effects of key predictors are visually summarised in the forest plot (Figure 1), demonstrating the magnitude and precision of associations.

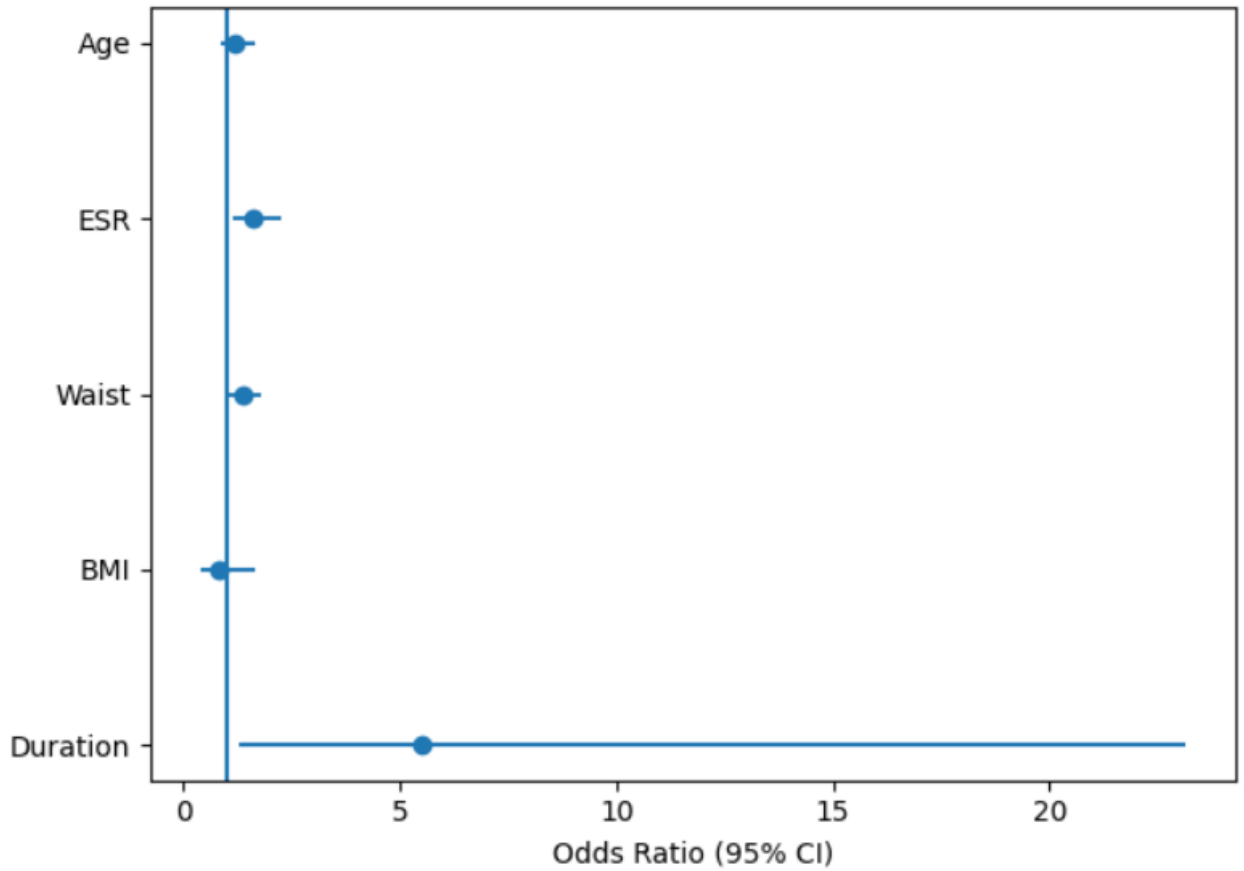

Figure 1. Forest plot of independent predictors of psoriatic arthritis.
Forest plot illustrating adjusted odds ratios (ORs) and 95% confidence intervals for predictors of psoriatic arthritis. The vertical reference line represents OR = 1.

Taken together, these results demonstrate that disease duration is the strongest clinical correlate of PsA, central obesity (waist circumference) is a more relevant metabolic predictor than BMI, systemic inflammation (ESR) independently contributes to PsA risk and age does not exert an independent effect.

Disscution
Discussion
The present study provides an integrated evaluation of clinical, metabolic, and inflammatory correlates of psoriatic arthritis (PsA) in a well-characterised cohort of patients with severe psoriasis. The findings demonstrate that prolonged disease duration, central obesity, and systemic inflammation are independently associated with PsA, offering important insights into the multifactorial nature of disease transition from cutaneous to articular involvement.
One of the most consistent observations in this study is the strong association between disease duration and PsA. Patients with PsA had a markedly longer history of psoriasis, and this variable remained an independent predictor in multivariate analysis. This finding supports the concept of a cumulative inflammatory burden, whereby chronic exposure to immune-mediated pathways—particularly the IL-23/IL-17 axis—progressively increases the likelihood of musculoskeletal involvement [12,13]. Previous longitudinal and population-based studies have similarly reported that the risk of PsA rises with increasing duration of psoriasis, reinforcing the importance of long-term surveillance in patients with established disease [14]. From a clinical perspective, this underscores the need for heightened vigilance and early rheumatological assessment in patients with longstanding psoriasis, even in the absence of overt joint symptoms.
The role of metabolic dysfunction, particularly obesity, has been increasingly recognised in psoriatic disease. In the present study, both BMI and waist circumference were elevated in patients with PsA in univariate analysis; however, only waist circumference retained significance after adjustment. This distinction is noteworthy, as it suggests that central (visceral) adiposity is a more relevant determinant than overall body mass. Visceral fat is metabolically active and contributes to systemic inflammation through the secretion of adipokines

and pro-inflammatory cytokines, including TNF-α and IL-6, which are implicated in PsA pathogenesis [15,16]. These findings are consistent with previous reports demonstrating stronger associations between abdominal obesity and PsA compared with BMI alone [17]. The attenuation of BMI significance in multivariate modelling likely reflects collinearity with waist circumference, highlighting the importance of selecting appropriate anthropometric measures in clinical research.

Systemic inflammation, as reflected by ESR, emerged as a robust independent correlate of PsA in this cohort. Elevated ESR levels in PsA patients are indicative of heightened inflammatory activity and align with prior studies linking acute-phase reactants to disease activity and structural progression [18]. While ESR is a non-specific marker, its persistence as an independent predictor suggests that it captures a clinically meaningful aspect of systemic immune activation that bridges cutaneous and articular disease domains. This supports the broader concept of psoriasis as a systemic inflammatory disorder, rather than a condition confined to the skin [19].

Interestingly, age did not demonstrate an independent association with PsA, despite a modest difference in univariate analysis. This finding aligns with previous literature suggesting that while PsA can occur across a wide age range, its development is more closely related to disease-specific and inflammatory factors than to chronological age alone [20].

Similarly, although metabolic comorbidities such as diabetes and hypertension were examined, they did not emerge as significant predictors in this cohort. This may reflect limited sample size, population characteristics, or the dominant influence of adiposity and inflammatory pathways.

Taken together, the findings of this study support a multidimensional model of PsA pathogenesis in which chronic inflammation, metabolic dysregulation, and disease chronicity interact to drive joint involvement. The identification of disease duration, central obesity, and ESR as key correlates provides clinically actionable insights. In particular, routine assessment of waist circumference and inflammatory markers, alongside careful evaluation of disease duration, may enhance early detection strategies for PsA in patients with severe psoriasis.

Conclusion

This study demonstrates that disease duration, central obesity, and systemic inflammation are key independent correlates of psoriatic arthritis in patients with severe psoriasis. Among these, disease duration appears to exert the strongest clinical influence, while waist circumference provides a more precise metabolic indicator than BMI. Elevated ESR further reflects the central role of systemic inflammation in PsA pathogenesis. These findings highlight the importance of integrated clinical and metabolic assessment in patients with severe psoriasis and support the implementation of targeted screening strategies aimed at early identification of individuals at increased risk for PsA. Early recognition and intervention may ultimately improve long-term outcomes and reduce the burden of irreversible joint damage.

Reference

1. Parisi R, Symmons DPM, Griffiths CEM, Ashcroft DM; Identification and Management of Psoriasis and Associated ComorbidiTy (IMPACT) project team. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol. 2013;133(2):377–385.
2. Mease PJ, Gladman DD, Papp KA, Khraishi MM, Thaçi D, Behrens F, et al. Prevalence of rheumatologist-diagnosed psoriatic arthritis in patients with psoriasis in European/North American dermatology clinics. Ann Rheum Dis. 2013;72(5):736–740.
3. Ogdie A, Weiss P. The epidemiology of psoriatic arthritis. Rheum Dis Clin North Am. 2015;41(4):545–568.
4. Langan SM, Seminara NM, Shin DB, Troxel AB, Kimmel SE, Mehta NN, et al. Prevalence of metabolic syndrome in patients with psoriasis: a population-based study in the United Kingdom. JAMA Dermatol. 2012;148(5):552–558.
5. Veale DJ, Fearon U. The pathogenesis of psoriatic arthritis. Lancet. 2018;391(10136):2273–2284.
6. Chandran V, Raychaudhuri SP. Geoepidemiology and environmental factors of psoriasis and psoriatic arthritis. J Autoimmun. 2010;34(3):J314–J321.

7. Wilson FC, Icen M, Crowson CS, McEvoy MT, Gabriel SE, Kremers HM. Time trends in epidemiology and characteristics of psoriatic arthritis over 3 decades: a population-based study. Arthritis Rheum. 2009;61(2):233–239.
8. Gisondi P, Tessari G, Conti A, Piaserico S, Schianchi S, Peserico A, et al. Prevalence of metabolic syndrome in patients with psoriasis: a hospital-based case–control study. Br J Dermatol. 2007;157(1):68–73.
9. Després JP. Body fat distribution and risk of cardiovascular disease: an update. Circulation. 2012;126(10):1301–1313.
10. Boehncke WH, Schön MP. Psoriasis. Lancet. 2015;386(9997):983–994.
11. Coates LC, Helliwell PS. Psoriatic arthritis: state of the art review. Ann Rheum Dis. 2016;75(2):193–199.
12. Scher JU, Ogdie A, Merola JF, Ritchlin C. Preventing psoriatic arthritis: focusing on patients with psoriasis at increased risk of transition. Nat Rev Rheumatol. 2019;15(3):153–166.
13. McGonagle D, Watad A, Savic S. Mechanistic immunological basis of psoriatic arthritis: the role of IL-23/IL-17 axis. J Intern Med. 2019;286(6):573–589.
14. Eder L, Chandran V, Gladman DD. The development of psoriatic arthritis in patients with psoriasis: a prospective cohort study. Arthritis Rheum. 2011;63(4):1212–1219.
15. Otero M, Lago R, Lago F, Dieguez C, Gómez-Reino JJ, Gualillo O. Leptin, from fat to inflammation: old questions and new insights. FEBS Lett. 2005;579(2):295–301.
16. Tilg H, Moschen AR. Adipocytokines: mediators linking adipose tissue, inflammation and immunity. Nat Rev Immunol. 2006;6(10):772–783.
17. Love TJ, Zhu Y, Zhang Y, Wall-Burns L, Ogdie A, Gelfand JM, et al. Obesity and the risk of psoriatic arthritis: a population-based study. Ann Rheum Dis. 2012;71(8):1273–1277.
18. Coates LC, FitzGerald O, Merola JF, Smolen JS, van Mens LJJ, Bertheussen H, et al. Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) treatment recommendations. Ann Rheum Dis. 2016;75(3):499–510.
19. Boehncke WH. Systemic inflammation and cardiovascular comorbidity in psoriasis patients: causes and consequences. Front Immunol. 2018;9:579.
20. Gladman DD. Clinical features and diagnostic considerations in psoriatic arthritis. Rheum Dis Clin North Am. 2015;41(4):569–579.