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OBSERVATIONAL RESEARCH

Factors influencing the transition time from psoriasis to psoriatic arthritis: a real-world multicenter analysis

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Abstract

To identify clinical and demographic predictors associated with the timing of transition from psoriasis (PsO) to psoriatic arthritis (PsA), and to compare the characteristics of patients with concurrent PsO-PsA onset versus those with prolonged transition. A multi-center, observational study was conducted using data from the Turkish League Against Rheumatism (TLAR) network including PsA patients fulfilling CASPAR criteria. Patients were categorized into two groups: Group 1 (concurrent PsO and PsA onset within ±1 year) and Group 2 (prolonged transition to PsA, >1 year after PsO). Demographic, clinical, and laboratory characteristics, disease activity, and patient-reported outcomes were compared between groups. Logistic regression was employed to determine independent predictors of prolonged transition. Among 799 patients (mean age 46.8±12.3 years), 237 (29.7%) had concurrent onset and 562 (70.3%) had a prolonged transition, with a mean PsO-to-PsA interval of 12.9±9.6 years. Depression ($p=0.005$) and fatigue levels ($p=0.011$) were significantly higher in patients with prolonged transition to PsA. Multivariate analysis revealed that scalp psoriasis (OR=7.162), nail psoriasis (OR=3.270), family history of PsO (OR=1.813), and enthesitis ever (OR=2.187) were associated with prolonged transition. Conversely, family history of PsA (OR=0.421) and older age at PsO onset (OR=0.957) predicted shorter transition. Prolonged transition from PsO to PsA is influenced by distinct clinical and demographic factors. Scalp/nail psoriasis, family history of PsO, and enthesitis ever may signal higher risk for prolonged PsA onset. Recognizing these markers can support timely referral and intervention, minimizing diagnostic delay and improving long-term patient outcomes.

Keywords Arthritis · Psoriasis · Psoriatic arthritis · Spondyloarthritis

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory disease characterized by a dysregulated immune response, leading to persistent inflammation that primarily affects the skin, entheses, and joints [1]. It is estimated that PsA affects approximately 20–30% of patients with psoriasis (PsO), with an annual incidence rate of 2.7 cases per 100 PsO patients, indicating a substantial disease burden in this population [2, 3]. Beyond musculoskeletal involvement, the presence of comorbidities such as cardiovascular and

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Extended author information available on the last page of the article

psychiatric disorders may adversely affect patient functioning and overall quality of life [4].

The relationship between PsO and PsA is complex and multifactorial. While the precise mechanisms underlying the pathogenesis of both conditions remain incompletely understood, it is believed that PsO and PsA share several common inflammatory pathways, suggesting a potential link between skin and joint disease [5, 6]. While PsO typically precedes PsA, the latter may also develop concurrently or, in some cases, precede the onset of skin manifestations [7, 8].

Understanding the factors that drive the transition from PsO to PsA is crucial for early diagnosis, effective management, and improved patient outcomes. Multiple factors, including the severity and extent of PsO, genetic predisposition, CD4 + T cell subsets exchange, comorbidities and environmental triggers, have been implicated in the development of PsA [9–12]. Despite ongoing research, key mechanisms underlying the transition from PsO to PsA remain incompletely understood. This uncertainty surrounding diagnoses often leads to delay, impacting about one-third of patients who experience such setbacks. Such delays may result in irreversible joint damage and substantial declines in health-related quality of life [13, 14].

The transition period from PsO to PsA is highly variable [7, 11], and the specific factors that influence this transition are not well understood. Limited research has pointed to possible risk factors, including the presence of HLA-Cw*0602, an early onset of psoriasis, more severe forms of psoriasis, and increased body mass index (BMI), which may correlate with longer transition durations [15–18]. However, the exact factors contributing to both early and late transitions are still not clearly defined. Early diagnosis and timely intervention are critical for effective patient management and enhancing long-term health outcomes. Therefore, this multicenter observational study aims to explore the clinical factors that affect the timing of the PsO to PsA transition and to compare the characteristics of patients experiencing prolonged transition times with those who develop PsA concurrently with PsO.

Materials and methods

Study design and data source

Participants in this multicenter, cross-sectional study were recruited from the Turkish League Against Rheumatism (TLAR)-Network, an extensive nationwide web-based registry designed to collect clinical and demographic information from patients with PsA. Detailed definitions and methodology of the registry were described in previously published studies [13, 19–21]. Ethical approval for the study was granted by the Ethics Committee of Sakarya University Medical School (25.01.2018/42), and written informed consent was obtained from all participants prior to their inclusion in the study.

Patients with PsA were classified into two groups based on the interval between the onset of PsO and PsA. Group 1 comprised patients whose onset of PsO and PsA occurred within one year of each other, indicating concurrent onset. Group 2 included patients who developed PsA beyond one year after the onset of PsO, representing a prolonged transition to PsA. This classification was adopted from the definitions used in a previous study by Karmacharya et al. [17].

Inclusion and exclusion criteria

All PsA patients included in the study were aged 18 years or older and met the CASPAR (ClASsification criteria for Psoriatic ARthritis) [22]. Patients with comorbidities potentially affecting outcome assessments, such as mental health disorders, cancer, or other rheumatic diseases, were excluded. Patients were also excluded if the onset of initial arthritis, enthesitis, or spondylitis findings preceded the onset of psoriasis by more than one year. Furthermore, pregnant or breastfeeding individuals were excluded from the study. To further minimize potential confounding, patients with other chronic inflammatory diseases (e.g., inflammatory bowel disease), serious systemic conditions (e.g., renal or hepatic failure), a history of substance abuse (e.g., alcohol), or those receiving immunosuppressive treatments for conditions unrelated to PsO and PsA were excluded.

Outcome measures

Our assessment protocol included multiple domains relevant to PsA evaluation. It included demographic details such as age, gender, and body mass index (BMI), as well as PsA associated clinical features and initial clinical presentation. The presence or history of extra-articular manifestations such as enthesitis, dactylitis, tenosynovitis, inflammatory bowel disease, and uveitis were also recorded. The onset of psoriasis was defined as the first occurrence of skin lesions identified by a healthcare provider, such as a dermatologist or rheumatologist. PsA onset was defined as the time of diagnosis by a rheumatologist, based on clinical, laboratory, and/or imaging findings consistent with classification criteria. Additionally, family history of psoriasis or PsA, as well as comorbidities, were documented. Laboratory investigations included measurements of inflammatory markers.

Disease activity was assessed using three validated instruments: the Disease Activity in Psoriatic Arthritis (DAPSA) score, the Minimal Disease Activity (MDA) criteria, and the Bath Ankylosing Spondylitis Disease Activity Index

(BASDAI) [23–25]. The patient's perception of their disease status and the physician's evaluation of disease activity were recorded using the patient's global assessment (PtGA) and the physician's global assessment (PhGA), respectively [26]. Fatigue severity and its impact on daily functioning were evaluated using the Functional Assessment of Chronic Illness Therapy–Fatigue (FACIT-Fatigue) scale [27]. Functional capacity was assessed by the Health Assessment Questionnaire (HAQ), while disease-specific quality of life was measured using the Psoriatic Arthritis Quality of Life Questionnaire (PsAQoL) [28, 29]. Skin involvement was quantified using the Psoriasis Area and Severity Index (PASI), which evaluates lesion severity across anatomical regions [30]. Furthermore, psychological symptoms were assessed with the Hospital Anxiety and Depression Scale (HADS), which includes two subscales measuring anxiety and depression [31].

Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics version 23 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as numbers and percentages, and continuous variables were expressed as means ± standard deviation (SD). The normality of distribution for continuous variables was assessed using the Kolmogorov-Smirnov test. Comparisons between patients with concurrent onset of psoriasis and psoriatic arthritis (Group 1) and those with a time interval longer than one year from psoriasis to psoriatic arthritis onset (Group 2) were performed using the Chi-square test or Fisher's exact test for categorical variables, and the independent samples t-test for continuous variables.

To identify independent predictors of a prolonged transition time (>1 year) from psoriasis to psoriatic arthritis, a multivariate binary logistic regression analysis was performed. Variables with a p-value < 0.25 in univariate analyses and those deemed clinically relevant were included in the multivariate logistic regression model. Multicollinearity was assessed using variance inflation factor (VIF) and tolerance values, and no significant multicollinearity was detected (all VIF values were < 2). The results of the logistic regression analysis are presented as odds ratios (ORs) with 95% confidence intervals (CIs). A two-tailed p-value < 0.05 was considered statistically significant.

Results

Demographic and clinical characteristics

A total of 904 PsA patients with complete data on PsA and PsO onset dates were identified. Of these, 105 patients who were diagnosed with PsO more than one year after arthritis/enthesitis/spondylitis onset were excluded. Consequently, 799 patients, with a mean age of 46.8 ± 12.3 years, were included in the final analysis. Among them, 237 (29.7%) patients had a concurrent onset of PsO and PsA, while 562 (70.3%) patients developed PsA more than one year after the onset of PsO. The comparison of clinical, demographic, and disease activity parameters between the two groups, based on PsO to PsA onset time, is summarized in Table 1.

Among patients in Group 2, the mean interval from PsO onset to PsA onset was 12.9 ± 9.6 years. These patients were significantly older at the time of PsA onset compared to Group 1 (39.7 ± 12.4 years vs. 34.8 ± 13.9 years, $p < 0.001$), despite having an earlier onset of PsO (26.8 ± 12.9 years vs. 34.7 ± 13.8 years, $p < 0.001$). Baseline characteristics such as gender distribution, BMI, family history of PsA, and PsO subtype did not differ significantly between the groups ($p > 0.05$). However, the family history of PsO was significantly more frequent in Group 2 than in Group 1 (37.0% vs. 29.1%, $p = 0.032$).

At initial presentation, the proportion of patients with enthesitis (16% vs. 8%, $p = 0.003$), low back pain (36.3% vs. 27.4%, $p = 0.015$), and peripheral arthritis (69.4% vs. 59.5%, $p = 0.007$) were more frequently observed in Group 2.

No significant differences were found regarding the uveitis (current/past) ($p = 0.291$), HLA-B27 positivity ($p = 0.381$), comorbidities ($p = 0.340$), smoking status ($p = 0.880$), or current use of biologic therapy ($p = 0.434$) between the groups. However, current use of conventional synthetic DMARDs (csDMARDs) was significantly higher in Group 2 than in Group 1 (54.4% vs. 45.6%, $p = 0.022$). There was no significant difference in the initial pattern of peripheral joint involvement between the groups ($p = 0.496$). However, in the current clinical status, polyarthritis was significantly more frequent in Group 2 compared to Group 1 (27.1% vs. 15.8%, $p = 0.003$).

Disease activity, quality of life and psychological status

There were no significant differences between Group 1 and Group 2 in terms of VAS pain, PtGA, PhGA, BASDAI, DAPSA, CRP levels, PASI total scores, PsAQoL, HAQ scores and achievement of MDA ($p > 0.05$). However, HADS-Depression scores were significantly higher in

Table 1 Comparison of clinical, demographic, and disease activity parameters in psoriatic arthritis patients based on PsO to PsA onset time

	Concurrent PsO and PsA (Group 1, $n=237$)	PsO to PsA > 1 year (Group 2, $n=562$)	p
Age, year	45.7 ± 12.9	47.2 ± 12.1	0.095
Male	97(40.9)	196(34.9)	0.105
BMI, kg/m^2	28.5 ± 5.5	28.8 ± 4.7	0.402
PsA onset Age	34.8 ± 13.9	39.7 ± 12.4	**< 0.001**
PsO onset Age	34.7 ± 13.8	26.8 ± 12.9	**< 0.001**
Family history of PsA	43(18.1)	81(14.4)	0.183
Family history of PsO	69(29.1)	208(37)	**0.032**
Initial presentation with;			
Dactylitis	20(8.4)	39(6.9)	0.548
Enthesitis	19(8)	90(16)	**0.003**
Low back pain	65(27.4)	204(36.3)	**0.015**
Peripheral arthritis	141(59.5)	390(69.4)	**0.007**
Enthesitis (ever)	91(38.4)	325(57.8)	**< 0.001**
Tenosynovitis (ever)	81(34.2)	260(46.3)	**0.007**
Current use of csDMARD	108(45.6)	306(54.4)	**0.022**
Current peripheral joint involvement			**0.003**
Mono/oligoarhtritis	83(37.6)	194(35.3)	
Polyarthritis	35(15.8)	149(27.1)	
DIP	9(4.1)	22(4)	
Artritis mutilans	1(0.5)	2(0.4)	
Initial peripheral joint involvement			0.496
Monoarthritis	53(23.3)	113(20.4)	
Oligoarthritis	93(41.0)	211(38.0)	
Polyarthritis	53(23.3)	137(24.7))	
DIP	4(1.8)	12(2.2)	
VAS-pain (0–10)	4.6 ± 2.7	4.9 ± 2.5	0.160
PtGA (0–10)	4.5 ± 2.5	4.7 ± 2.5	0.297
PhGA (0–10)	3.9 ± 2.2	4.1 ± 2.2	0.306
BASDAI (0–10)	4.23 ± 2.1	4.4 ± 2.0	0.333
DAPSA	16.5 ± 12.8	17.6 ± 12.8	0.286
MDA achievement	41(19)	78(14.5)	0.124
PASI total	3.0 ± 3.8	3.6 ± 5.5	0.081
CRP mg/L	7.22 ± 11.3	9.0 ± 12.6	0.068
PsAQoL	6.2 ± 6.3	7.1 ± 6,2	0.053
HADS-anxiety score	6.7 ± 4.5	6,8 ± 4.1	0.643
HADS-depression score	6.2 ± 4.1	7.2 ± 4.1	**0.001**
HADS-anxiety ≥10	59(24.9)	132(23.6)	0.689
HADS-depression ≥ 7	106(44.7)	311(55.5)	**0.005**
FACIT	17.7 ± 10.2	19.7 ± 10.3	**0.011**
HAQ	0.42 ± 0.50	0.43 ± 0.46	0.695

Values are expressed as mean ± SD or n(%). *BASDAI* Bath Ankylosing Spondylitis Disease Activity Index; *BMI* body mass index; *CRP* C-Reactive Protein; *csDMARD* Conventional synthetic disease modifying antirheumatic drug; *FACIT* Functional Assessment of Chronic Illness Therapy; *HADs* Hospital Anxiety and Depression Scale; *HAQ* Health Assessment Questionnaire; *MDA* Minimal Disease Activity; *PASI* Psoriasis Area Severity Index; *PhGA* Physician Global Assessment; *PtGA* Patient Global Assessment; *PsA* Psoriatic Arthritis; *PsO* Psoriasis; *PsO2PsA* PsA-to PsA; *PsAQoL* Psoriatic Arthritis Quality of Life; *VAS* Visual Analogue Scale

Group 2 compared to Group 1 (7.2 vs. 6.2, $p=0.001$), with a greater proportion of patients in Group 2 meeting the threshold for depression (55.5% vs. 44.7%, $p=0.005$) (Fig. 1). HADS-Anxiety scores were similar between the two groups ($p>0.05$). Regarding fatigue, Group 2 reported significantly higher FACIT-Fatigue scores compared to Group 1 (19.7 vs. 17.7, $p=0.011$).

Predictors of prolonged transition from PsO to PsA

In the multivariate logistic regression analysis, several factors were significantly associated with a prolonged transition from PsO to PsA (Table 2; Fig. 2). Scalp psoriasis (OR = 7.162, $p=0.014$), and nail psoriasis (OR = 3.270, $p=0.041$) were significantly associated with a prolonged transition to PsA. Additionally, a family history of psoriasis (OR = 1.813, $p=0.006$) and enthesitis ever (OR = 2.187, $p<0.001$) were positively associated with prolonged transition to PsA. In contrast, family history of PsA was negatively associated with prolonged transition (OR = 0.421, $p=0.002$). Furthermore, older age at PsO onset had a significantly lower likelihood of prolonged transition (OR = 0.957, $p=0.001$).

Discussion

This multicenter study provides comprehensive insight into the clinical characteristics and predictive factors associated with the transition from PsO to PsA. Most patients with PsA in our cohort had preceding PsO, supporting the sequential development of these conditions. Additionally, we identified key demographic and clinical factors that influence the timing of this disease progression, providing valuable insights for clinical practice.

Previous population-based and cohort studies have reported that the interval between PsO onset and the development of PsA ranges from 9 to 13 years, with the proportion of PsA patients having prior PsO ranging between 60% and 88.7% [7, 17]. In line with these findings, our real-world multicenter study showed that 70% of PsA patients had a prior history of PsO, with a mean PsO-to-PsA interval of approximately 13 years. These findings support the need for continued long-term monitoring of PsO patients to facilitate earlier PsA diagnosis.

While considerable research has highlighted numerous risk factors associated with the development of PsA in individuals with PsO, there exists a limited number of studies that specifically examine the clinical and demographic factors that affect the duration of time between the initial onset of PsO and the subsequent development of PsA [9, 10]. One of the first population-based investigations ($N=164$) on

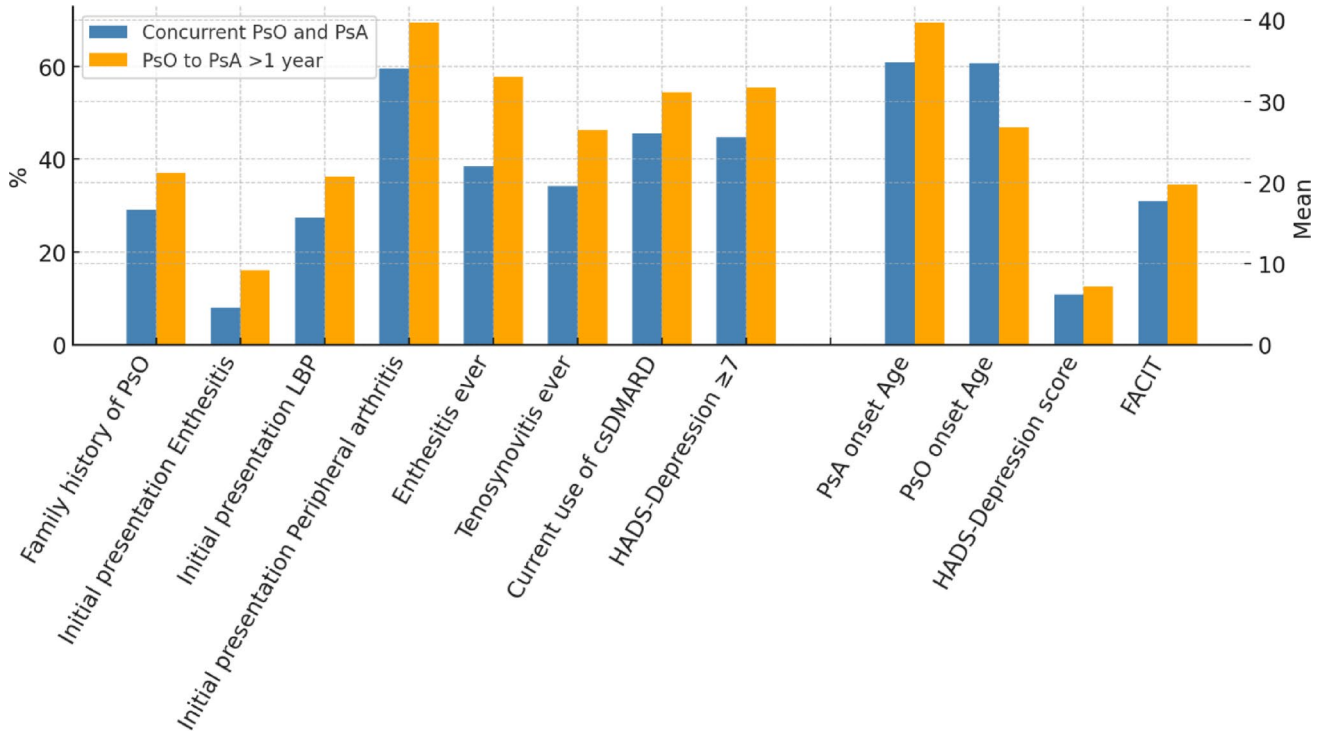

Fig. 1 Comparison of selected clinical and demographic parameters between patients with concurrent PsO and PsA versus those with PsA onset > 1 year after psoriasis

Table 2 Multivariate logistic regression analysis of factors associated with a prolonged transition (> 1 year) from psoriasis to psoriatic arthritis

	OR (95% CI)	*p*
Psoriasis subtypes		
Plaque psoriasis	2.182 (0.830–5.737)	0.114
Erythrodermic psoriasis	1.229 (0.319–4.742)	0.765
Nail psoriasis	3.270 (1.049–10.190)	**0.041**
Pustular psoriasis	1.504 (0.354–6.395)	0.580
Palmoplantar psoriasis	1.347 (0.398–4.556)	0.632
Scalp psoriasis	7.162 (1.488–34.474)	**0.014**
Guttate psoriasis	1.679 (0.422–6.679)	0.462
Family history of PsO	1.813 (1.189–2.765)	**0.006**
Family history of PsA	0.421 (0.241–0.737)	**0.002**
Enthesitis ever	2.187 (1.566–3.056)	**<0.001**
PsO onset age	0.957 (0.945–0.969)	**0.001**
PASI-lower extremity	1.070 (0.989–1.157)	0.092

All OR for psoriasis subtypes are calculated in comparison to generalized psoriasis

CI Confidence Interval; *OR* Odds Ratio; *PsA* Psoriatic Arthritis; *PsO* Psoriasis

this topic was conducted by Karmacharya et al. in Olmsted County, Rochester, MN [17]. Their study categorized PsA patients into concurrent and delayed-onset groups (based on medical record reviews) and found that younger age at PsO onset and greater PsO severity were associated with a longer transition period. Building on this, Cheemalavagu et al. employed a zero-inflated negative binomial model within a biorepository cohort (N=384) drawn from a tertiary care center and similarly reported that older age at psoriasis onset predicted a shorter time to PsA development [15]. Consistent with these two single-center studies, our multi-center cohort confirmed that younger age at PsO onset was a predictor of prolonged transition to PsA. Unlike these previous studies, which included relatively limited sample sizes and more specific patient groups from specialized clinics, our study was conducted across multiple centers and included a broader patient population. This wider and more varied sample may offer a more realistic picture of how the transition from PsO to PsA occurs in everyday clinical settings. Such diversity enhances our understanding of different patient profiles and supports the relevance of our findings in routine practice.

Moreover, in the study by Karmacharya et al., an association was reported between certain PsO phenotypes particularly plaque-type PsO and a prolonged transition to PsA [17]. In our study, nail and scalp involvement emerged as more consistent phenotypic predictors of prolonged transition to PsA. These findings suggest that the specific distribution of psoriatic lesions may serve as indicators of prolonged PsA development. Consistently, the 2023 EULAR task force emphasized that nail involvement, although recognized as an established risk factor, should not be regarded as imminent predictor of PsA onset but rather as long-term marker to guide preventive strategies [10]. Our results support this

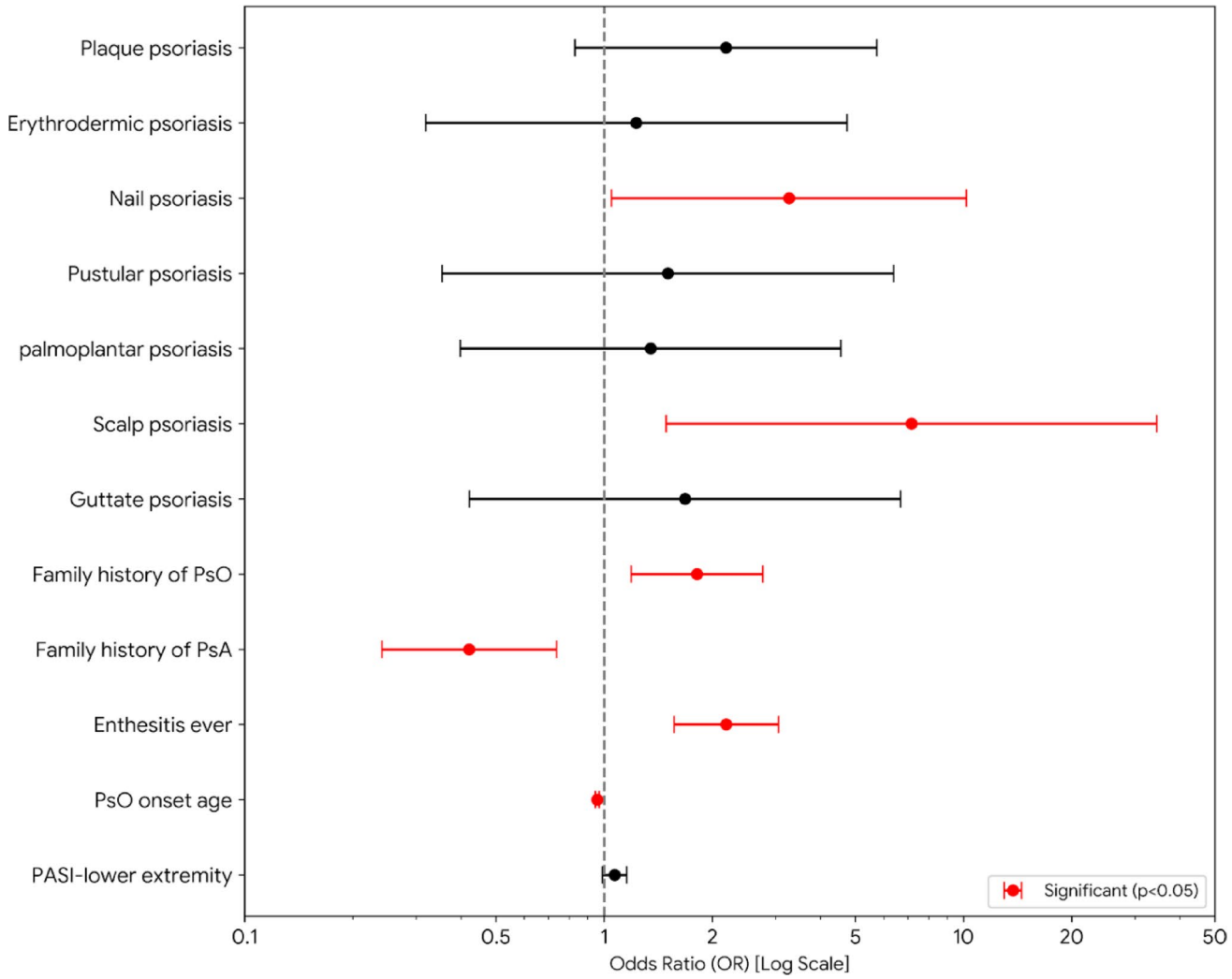

Fig. 2 Multivariate logistic regression results show factors associated with prolonged (>1 year) transition from psoriasis to psoriatic arthritis. Odds ratios and 95% confidence intervals are shown

perspective, highlighting the relevance of these phenotypes in capturing the heterogeneity of PsA progression. Additionally, such patterns may reflect a distinct immunopathologic profile that predisposes patients to a more gradual or insidious transition from skin to joint involvement.

Family history also emerged as a significant variable in our analysis. A family history of PsA was associated with concurrent onset of PsO and PsA, while a family history of PsO was associated with prolonged transition to PsA. This builds on findings from the PsART international cohort (abstract, 2019), which reported only the latter association [32]. This contrast may reflect differing genetic contributions to skin versus joint disease, or possibly greater clinician awareness and surveillance in patients with known PsA familial clustering.

Furthermore, our study identified that PsA patients with enthesitis ever had a higher likelihood of experiencing a prolonged transition from PsO to PsA. This is clinically relevant, as enthesitis is not only a hallmark feature of PsA but also an early pathological event in its development, often triggered by mechanical stress and sustained by pro-inflammatory cytokines such as TNF and IL-17 [33, 34]. Moreover, previous study has reported that enthesitis is more frequently observed in older patients and those with a longer history of psoriasis, suggesting a possible link between chronic cutaneous inflammation and entheseal involvement [35]. The association between enthesitis and prolonged PsA onset may reflect a low-grade, subclinical inflammatory process at entheseal sites that evolves slowly before manifesting as overt arthritis. This highlights the need for heightened clinical vigilance and proactive monitoring of musculoskeletal inflammation, even in the absence of obvious joint involvement, particularly in psoriatic patients with a history of enthesitis [36].

The most recent inception cohort study investigated differences in disease-related features and outcomes between

early and late PsA transition using a median PsO-to-PsA interval of nine years as the cutoff [16]. They reported that early transition patients tended to exhibit higher disease activity over five years, while late transition patients were more likely to be obese at presentation [16]. In contrast, our study found that although disease activity, function, and quality of life were similar between groups, patients with prolonged transition experienced significantly higher levels of fatigue and depressive symptoms. Several factors may contribute to this observation. Psoriasis, even in the absence of joint involvement, can impose a significant psychological burden due to its visibility, chronic nature, and frequent associations with stigmatization, low self-esteem, and social withdrawal [37]. From a biological perspective, prolonged exposure to systemic inflammation mediated by cytokines such as TNF-α and IL-6 may directly contribute to fatigue and depressive symptoms [37, 38]. These pro-inflammatory mediators are not only central to the pathogenesis of psoriatic disease but have also been implicated in neuroinflammation and disruptions of neuroendocrine pathways involved in mood and energy regulation [39]. Fatigue, even in patients with minimal disease activity, is a prevalent and important complaint in PsA and appears to have a multifactorial etiology. Its association with disease activity and chronicity suggests an inflammatory component that is amenable to treatment, although this alone does not fully explain its persistence [40]. Collectively, these findings underscore a potentially underrecognized psychological burden in patients with prolonged disease progression, highlighting the importance of a holistic clinical approach that integrates both physical and mental health assessments in individuals with longstanding PsO.

This study has several strengths that distinguish it from previous literature. The multicenter design and relatively large sample size enhance the representativeness and robustness of the findings within the Turkish population. Furthermore, the detailed clinical characterization of PsA related manifestations alongside assessments of disease activity, physical function, fatigue, and quality of life provides a holistic view of the patient experience and offers valuable insights into predictors of prolonged PsA transition. Nevertheless, certain limitations should be acknowledged. The observational, cross-sectional design of the study restricts causal inference and raises the possibility that unmeasured confounding factors may have influenced the observed associations. Additionally, the retrospective and self-reported nature of certain clinical variables, such as the timing of PsO and PsA onset, may introduce recall bias and compromise the accuracy of temporal assessments. Given the geographic scope of the study, the findings may also have limited generalizability to populations outside Turkey. Future prospective, longitudinal studies in more diverse populations are warranted to validate and expand upon these results [41].

In conclusion, this multicenter observational study provides robust real-world evidence on the clinical and demographic factors that influence the timing of transition from PsO to PsA. Younger age at PsO onset, specific phenotypes such as nail and scalp involvement, enthesitis ever, and a family history of PsO were identified as independent predictors of prolonged transition to PsA. These findings highlight the heterogeneous nature of psoriatic disease progression and underscore the clinical importance of individualized risk profiling to enable earlier detection and timely intervention for PsA. Early recognition of these predictive markers in routine dermatologic and rheumatologic care may facilitate timely referral, specialist assessment, proactive intervention and targeted monitoring.

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Author contributions All authors contributed to the data collection. Significant contributions to the conceptualization and design of the study were made: G.K., E.K., SO., and I.T. Data analysis was performed by EK and GK. The initial manuscript was drafted: G.K., E.K., SO. All authors critically revised the manuscript for important intellectual content. The final version for publication received approval from all authors. Full responsibility for the integrity and accuracy of all aspects of the work is assumed by all authors. All authors were involved in the acquisition of data and its interpretation.

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Data availability The data supporting the findings of this study are not publicly accessible but can be obtained from the corresponding author upon reasonable request.

Declarations

Conflict of interest The authors declare that they have no competing interest.

Ethical approval The protocols of this study were approved by local ethics committee (25.01.2018/42), and written informed consent was obtained from each participant.

References

1. Barnas JL, Ritchlin CT (2015) Etiology and pathogenesis of psoriatic arthritis. Rheum Dis Clin North Am 41:643–663. https://doi.org/10.1016/j.rdc.2015.07.006
2. Haroon M, Kirby B, FitzGerald O (2013) High prevalence of psoriatic arthritis in patients with severe psoriasis with suboptimal

performance of screening questionnaires. Ann Rheum Dis 72:736–740. https://doi.org/10.1136/annrheumdis-2012-201706
3. Eder L, Haddad A, Rosen CF, Lee KA, Chandran V, Cook R et al (2016) The incidence and risk factors for psoriatic arthritis in patients with psoriasis: a prospective cohort study. Arthritis Rheumatol 68:915–923. https://doi.org/10.1002/art.39494
4. Biedron G, Wilk M, Nowakowski J, Kuszmiersz P, Gula Z, Strach M et al (2024) Impact of comorbidities on patient-reported outcomes in psoriatic arthritis: a single centre cohort study. Rheumatol Int 44:1435–1443. https://doi.org/10.1007/s00296-024-05632-2
5. Pap T, Sunderkotter C (2017) [Psoriasis vs. psoriatic arthritis: similarities and differences in the pathophysiology]. Z Rheumatol 76:477–483. https://doi.org/10.1007/s00393-017-0342-0
6. Dilek G, Kalcik Unan M, Nas K (2025) Immune response and cytokine pathways in psoriatic arthritis: a systematic review. Arch Rheumatol 40:144–156. https://doi.org/10.46497/ArchRheumatol.2025.10934
7. Tillett W, Charlton R, Nightingale A, Snowball J, Green A, Smith C et al (2017) Interval between onset of psoriasis and psoriatic arthritis comparing the UK clinical practice research datalink with a hospital-based cohort. Rheumatology (Oxford) 56:2109–2113. https://doi.org/10.1093/rheumatology/kex323
8. Gladman DD, Shuckett R, Russell ML, Thorne JC, Schachter RK (1987) Psoriatic arthritis (PSA)--an analysis of 220 patients. Q J Med 62:127–141
9. Scher JU, Ogdie A, Merola JF, Ritchlin C (2019) Preventing psoriatic arthritis: focusing on patients with psoriasis at increased risk of transition. Nat Rev Rheumatol 15:153–166. https://doi.org/10.1038/s41584-019-0175-0
10. Zabotti A, De Marco G, Gossec L, Baraliakos X, Aletaha D, Iagnocco A et al (2023) EULAR points to consider for the definition of clinical and imaging features suspicious for progression from psoriasis to psoriatic arthritis. Ann Rheum Dis 82:1162–1170. https://doi.org/10.1136/ard-2023-224148
11. Wilson FC, Icen M, Crowson CS, McEvoy MT, Gabriel SE, Kremers HM (2009) Incidence and clinical predictors of psoriatic arthritis in patients with psoriasis: a population-based study. Arthritis Rheum 61:233–239. https://doi.org/10.1002/art.24172
12. Gao W, Lei Y, Guo X, Luo Y, Zhang Z, Xi Y et al (2023) Comparison and subsets analysis of peripheral CD4(+)T cells in patients with psoriasis and psoriatic arthritis. Mol Immunol 163:174–180. https://doi.org/10.1016/j.molimm.2023.10.002
13. Kilic G, Kilic E, Tekeoglu I, Sargin B, Cengiz G, Balta NC et al (2024) Diagnostic delay in psoriatic arthritis: insights from a nationwide multicenter study. Rheumatol Int 44:1051–1059. https://doi.org/10.1007/s00296-023-05479-z
14. Haroon M, Gallagher P, FitzGerald O (2015) Diagnostic delay of more than 6 months contributes to poor radiographic and functional outcome in psoriatic arthritis. Ann Rheum Dis 74:1045–1050. https://doi.org/10.1136/annrheumdis-2013-204858
15. Cheemalavagu S, Jin Y, Husni ME (2024) What clinical factors affect length of transition to psoriatic arthritis in patients with psoriasis?? ACR Open Rheumatol 6:553–560. https://doi.org/10.1002/acr2.11703
16. Kharouf F, Mehta P, Gao S, Pereira D, Cook R, Gladman DD et al (2025) Comparative analysis of disease outcomes: early vs. late transition from psoriasis to psoriatic arthritis. Semin Arthritis Rheum 71:152639. https://doi.org/10.1016/j.semarthrit.2025.152639
17. Karmacharya P, Wright K, Achenbach SJ, Crowson CS, Ogdie A, Bekele D et al (2022) Time to transition from psoriasis to psoriatic arthritis: a population-based study. Semin Arthritis Rheum 52:151949. https://doi.org/10.1016/j.semarthrit.2021.12.013
18. Queiro R, Gonzalez S, Lopez-Larrea C, Alperi M, Sarasqueta C, Riestra JL et al (2006) Hla-c locus alleles may modulate the clinical expression of psoriatic arthritis. Arthritis Res Ther 8:R185. https://doi.org/10.1186/ar2097
19. Kilic E, Kilic G, Tekeoglu I, Sargin B, Acer Kasman S, Alkan H et al (2024) Regional variations in psoriatic arthritis: insights from a nationwide multicenter analysis in Turkiye. Arch Rheumatol 39:339–349. https://doi.org/10.46497/ArchRheumatol.2024.10591
20. Sunar I, Ataman S, Nas K, Kilic E, Sargin B, Kasman SA et al (2020) Enthesitis and its relationship with disease activity, functional status, and quality of life in psoriatic arthritis: a multi-center study. Rheumatol Int 40:283–294. https://doi.org/10.1007/s00296-019-04480-9
21. Gezer HH, Duruoz MT, Nas K, Kilic E, Sargin B, Kasman SA et al (2022) Inconsistencies of the disease activity assessment tools for psoriatic arthritis: challenges to rheumatologists. Joint Bone Spine 89:105296. https://doi.org/10.1016/j.jbspin.2021.105296
22. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H (2006) Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum 54:2665–2673. https://doi.org/10.1002/art.21972
23. Schoels M, Aletaha D, Funovits J, Kavanaugh A, Baker D, Smolen JS (2010) Application of the DAREA/DAPSA score for assessment of disease activity in psoriatic arthritis. Ann Rheum Dis 69:1441–1447. https://doi.org/10.1136/ard.2009.122259
24. Coates LC, Fransen J, Helliwell PS (2010) Defining minimal disease activity in psoriatic arthritis: a proposed objective target for treatment. Ann Rheum Dis 69:48–53. https://doi.org/10.1136/ard.2008.102053
25. Garrett S, Jenkinson T, Kennedy LG, Whitelock H, Gaisford P, Calin A (1994) A new approach to defining disease status in ankylosing spondylitis: the bath ankylosing spondylitis disease activity index. J Rheumatol 21:2286–2291
26. Talli S, Etcheto A, Fautrel B, Balanescu A, Braun J, Canete JD et al (2016) Patient global assessment in psoriatic arthritis - what does it mean? An analysis of 223 patients from the psoriatic arthritis impact of disease (PsAID) study. Joint Bone Spine 83(3):335–340. https://doi.org/10.1016/j.jbspin.2015.06.018
27. Webster K, Cella D, Yost K (2003) The functional assessment of chronic illness therapy (FACIT) measurement system: properties, applications, and interpretation. Health Qual Life Outcomes 1:79. https://doi.org/10.1186/1477-7525-1-79
28. Kucukdeveci AA, Sahin H, Ataman S, Griffiths B, Tennant A (2004) Issues in cross-cultural validity: example from the adaptation, reliability, and validity testing of a Turkish version of the Stanford health assessment questionnaire. Arthritis Rheum 51:14–19. https://doi.org/10.1002/art.20091
29. McKenna SP, Doward LC, Whalley D, Tennant A, Emery P, Veale DJ (2004) Development of the PSAQoL: a quality of life instrument specific to psoriatic arthritis. Ann Rheum Dis 63:162–169. https://doi.org/10.1136/ard.2003.006296
30. Feldman SR, Fleischer AB Jr., Reboussin DM, Rapp SR, Exum ML, Clark AR et al (1996) The self-administered psoriasis area and severity index is valid and reliable. J Invest Dermatol 106:183–186. https://doi.org/10.1111/1523-1747.ep12329912
31. Zigmond AS, Snaith RP (1983) The hospital anxiety and depression scale. Acta Psychiatr Scand 67:361–370. https://doi.org/10.1111/j.1600-0447.1983.tb09716.x
32. (2019) 2019 ACR/ARP Annual Meeting Abstract Supplement. Arthritis Rheumatol 71 Suppl 10:1-5362. https://doi.org/10.1002/art.41108
33. Araujo EG, Schett G (2020) Enthesitis in psoriatic arthritis (Part 1): pathophysiology. Rheumatology (Oxford) 59:i10–i14. https://doi.org/10.1093/rheumatology/keaa039
34. Pignon C, Bibas N, Lopez-Medina C, Fautrel B, Gossec L (2025) Clinical enthesitis in patient with psoriatic arthritis, a systematic

literature review with metaanalysis. Joint Bone Spine 92:105807. https://doi.org/10.1016/j.jbspin.2024.105807
35. Grazio S, Situm M, Grubisic F, Kavanagh HS, Vajdic ID, Krstanovic K et al (2024) Association of enthesitis with severity of psoriasis in psoriatic arthritis: an observational study. Rheumatol Int 44:2891–2896. https://doi.org/10.1007/s00296-024-05730-1
36. Aydin SZ, Ash ZR, Tinazzi I, Castillo-Gallego C, Kwok C, Wilson C et al (2013) The link between enthesitis and arthritis in psoriatic arthritis: a switch to a vascular phenotype at insertions may play a role in arthritis development. Ann Rheum Dis 72:992–995. https://doi.org/10.1136/annrheumdis-2012-201617
37. Sahi FM, Masood A, Danawar NA, Mekaiel A, Malik BH (2020) Association between psoriasis and depression: a traditional review. Cureus 12:e9708. https://doi.org/10.7759/cureus.9708
38. Chmielewski G, Majewski MS, Kuna J, Mikiewicz M, Krajewska-Wlodarczyk M (2023) Fatigue in Inflammatory Joint Diseases. Int J Mol Sci 24. https://doi.org/10.3390/ijms241512040
39. Salcudean A, Bodo CR, Popovici RA, Cozma MM, Pacurar M, Craciun RE et al (2025) Neuroinflammation-A Crucial Factor in the Pathophysiology of Depression-A Comprehensive Review. Biomolecules 15. https://doi.org/10.3390/biom15040502
40. Tan JSQ, Fong W, Kwan YH, Leung YY (2020) Prevalence and variables associated with fatigue in psoriatic arthritis: a cross-sectional study. Rheumatol Int 40:1825–1834. https://doi.org/10.1007/s00296-020-04678-2
41. Haberman RH, MacFarlane KA, Catron S, Samuels J, Blank RB, Toprover M et al (2022) Efficacy of guselkumab, a selective IL-23 inhibitor, in preventing arthritis in a multicentre psoriasis At-Risk cohort (PAMPA): protocol of a randomised, double-blind, placebo controlled multicentre trial. BMJ Open 12:e063650. https://doi.org/10.1136/bmjopen-2022-063650

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