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Nail Dystrophy as a Predictive Marker for Psoriatic Arthritis: A Clinical, Ultrasonographic, and Microscopic Analysis

Distrofia Ungueal como Marcador Preditivo para Artrite Psoriásica: Uma Análise Clínica, Ultrassonográfica e Microscópica

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Abstract
Introduction: *Nail psoriasis is a frequent and often debilitating manifestation of psoriasis, commonly associated with psoriatic arthritis (PsA). This study aimed to evaluate whether nail dystrophy serves as a reliable predictive marker for PsA.* ***Methodology:*** *a cross-sectional study was conducted with patients divided into four groups based on the presence or absence of nail dystrophy and PsA. Clinical assessments included Nail Psoriasis Severity Index (NAPSI) and Psoriasis Area and Severity Index (PASI) scores. Ultrasonographic examination (B-mode and Power Doppler) and microscopic analysis of nail clippings were performed to assess nail bed vascularisation, nail plate thickness, presence of neutrophils, and transition zone integrity.* ***Results:*** *a total of 120 patients were included. Nail dystrophy was observed in patients with and without PsA. There was no statistically significant difference in NAPSI scores between dystrophic groups. Multivariate logistic regression identified a Power Doppler signal ≥2 as the strongest independent predictor of PsA (OR = 3.5; p = 0.002), followed by body mass index (BMI) (OR = 1.13; p = 0.015). Nail dystrophy did not remain statistically significant after adjustment (OR = 1.91; p = 0.07). Microscopic features, including neutrophilic infiltration and blurring of the transition zone, correlated with cutaneous psoriasis severity but not with PsA diagnosis.* ***Conclusion:*** *although frequently present in PsA, nail dystrophy alone was not a specific predictive marker. Dynamic inflammatory parameters, such as increased nail bed vascularisation on Power Doppler, and systemic factors, such as elevated BMI, showed stronger associations with PsA. These findings highlight the need for integrated diagnostic approaches combining clinical, imaging, and systemic inflammatory markers to stratify PsA risk in patients with psoriasis better.*
Keywords: *Nail; psoriasis; ultrasound.*

Resumo
Introdução: *A psoríase ungueal é uma manifestação frequente e, muitas vezes, debilitante da psoríase, comumente associada à artrite psoriásica (APs).* **Objetivo***: avaliar se a distrofia ungueal atua como um marcador preditivo confiável para APs.* **Metodologia***: Foi realizado um estudo transversal com pacientes divididos em quatro grupos, com base na presença ou ausência de distrofia ungueal e de APs. As avaliações clínicas incluíram os escores do Índice de Gravidade da Psoríase Ungueal (NAPSI) e do Índice de Área e Gravidade da Psoríase (PASI). Foram realizados exames ultrassonográficos (modo B e Power Doppler) e análise microscópica de fragmentos ungueais para avaliação da vascularização do leito ungueal, da espessura da lâmina ungueal, da presença de neutrófilos e da integridade da zona de transição.* **Resultados***: Um total de 120 pacientes foi incluído no estudo. A distrofia ungueal foi observada em pacientes com e sem APs. Não houve diferença estatisticamente significativa nos escores NAPSI entre os grupos com distrofia ungueal. A regressão logística multivariada identificou o sinal de Power Doppler ≥ 2 como o preditor independente mais forte de APs (OR = 3,5; p = 0,002), seguido pelo índice de massa corporal (IMC) (OR = 1,13; p = 0,015). A distrofia ungueal não permaneceu estatisticamente significativa após o ajuste (OR = 1,91; p = 0,07). As características microscópicas, incluindo infiltração neutrofílica e borramento da zona de transição, correlacionaram-se com a gravidade da psoríase cutânea, mas não com o diagnóstico de APs.* **Conclusão***: Embora frequentemente presente na APs, a distrofia ungueal, de forma isolada, não se mostrou um marcador preditivo específico. Parâmetros inflamatórios dinâmicos, como o aumento da vascularização do leito ungueal avaliado pelo Power Doppler, e fatores sistêmicos, como o IMC elevado, demonstraram associações mais fortes com APs. Esses achados ressaltam a necessidade de abordagens diagnósticas.*
Palavras-chave: *Unha; psoríase; ultrassom.*

INTRODUCTION

Psoriasis is a chronic, multifactorial inflammatory disease that primarily affects the skin but can also involve the nails and joints[1]. Nail psoriasis, one of the most challenging manifestations of the disease, occurs in up to 80% of patients with cutaneous psoriasis during their lifetime

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and in up to 50% of those with psoriatic arthritis[2]. Its impact on quality of life can be considerable, particularly in cases of extensive involvement, pain, or evident cosmetic alterations. Moreover, nail changes are significantly associated with an increased risk of developing psoriatic arthritis (PsA)[3]. Clinically, nail psoriasis encompasses a broad spectrum of alterations, including pitting (small depressions in the nail plate), discolouration, onycholysis, subungual hyperkeratosis, and nail plate deformation. These manifestations vary in severity and frequently coexist with cutaneous and joint involvement[4].

Nail ultrasonography has emerged as a noninvasive, sensitive imaging modality for assessing morphological and inflammatory changes in the nail apparatus in patients with psoriasis and PsA. It enables precise measurement of nail plate, bed, and matrix thickness, and can detect subclinical inflammation through Doppler imaging, even in clinically unaffected nails. Increased nail bed and matrix thickness correlate with disease severity and PsA activity indices, suggesting their potential as prognostic indicators of arthritis development in patients with cutaneous psoriasis[5].

Nail clipping plays a relevant role in the evaluation of nail psoriasis, particularly as a complementary method to clinical examination for differential diagnosis and histopathological confirmation. The procedure consists of collecting nail plate fragments for microscopic analysis, allowing identification of features such as hyperparakeratosis, serous lakes, neutrophils, and increased nail plate thickness. Therefore, nail clipping is useful for confirming the diagnosis of nail psoriasis, distinguishing it from other causes of nail dystrophy, and guiding management, especially in cases of isolated presentation or diagnostic uncertainty[6].

Arthritis psoriatic itself is a chronic inflammatory joint disorder associated with skin and nail psoriasis, typically characterised by peripheral arthritis (oligoarthritis or polyarthritis), axial involvement (spine and sacroiliac joints), enthesitis, dactylitis ("sausage digits"), and nail changes such as pitting and onycholysis. While most patients develop cutaneous lesions before arthritis onset, up to 15% may experience articular symptoms that precede or occur without evident skin involvement. Diagnosis remains clinical, based on the presence of joint inflammation in psoriasis patients, the absence of rheumatoid factor, and the exclusion of other arthritides. No specific biomarkers for PsA have yet been identified. If left untreated, PsA can lead to irreversible joint damage, deformities, and functional impairment, underscoring the importance of a comprehensive assessment encompassing skin, joints, entheses, and nails[7].

Given the clinical relevance of nail psoriasis and the growing utility of complementary diagnostic tools, this study aimed to evaluate whether nail dystrophy serves as a reliable predictive marker for PsA.

METHODOLOGY

A cross-sectional study was conducted involving patients aged 18 years or older with psoriasis and psoriatic arthritis, classified according to the CASPAR criteria[8]. Participants were consecutively enrolled from the dermatology outpatient clinic of our university hospital between November 2020 and January 2022. Participants were recruited from the outpatient Dermatology Service Unit. Patients who were seen at the outpatient clinic were evaluated consecutively. Only those who met the inclusion criteria and agreed to participate in the research were included. Inclusion criteria: The selected individuals will be adult patients aged 18 and above, of both genders, with no age cutoff. Participants must be volunteers who are well-informed and have agreed to the collection of clinical information and materials after reading and completing the informed consent form. Exclusion criteria included failure to provide written informed consent, positive findings for fungal infection on direct mycological examination or nail culture, history of nail trauma within the preceding six weeks, prior local corticosteroid injections in the distal interphalangeal (DIP) joints, and a diagnosis of hand osteoarthritis. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and adhered to local regulatory requirements. Ethical approval was granted by the Institutional Review Board (IRB) under protocol number 4.279.067. Written informed consent was obtained from all enrolled participants.

Patients with a confirmed diagnosis of psoriasis and psoriatic arthritis were assessed for demographic characteristics (age, sex), clinical presentation of psoriasis and psoriatic arthritis, disease duration, presence of nail abnormalities, Psoriasis Area and Severity Index (PASI) scores, Nail Psoriasis Severity Index (NAPSI) scores (for cases with nail dystrophy), and epidemiological data. All patients underwent nail sample collection by clipping for microscopic analysis and completed the standardised questionnaire on the same day. The ultrasound examination was scheduled subsequently at the earliest possible date.

The study cohort was stratified into four groups:

Patients in Groups 1 and 2 are individuals diagnosed with psoriasis and without joint involvement, and those in Groups 3 and 4 are individuals diagnosed with psoriasis and joint involvement.

Group 1: patients with plaque psoriasis and clinically normal nails (Pso Normal); Group 2: patients with plaque psoriasis and clinical nail dystrophy (Pso Dystrophy); Group 3: patients with psoriatic arthritis and clinically normal nails (Arthritis Normal); Group 4: patients with psoriatic arthritis and nail dystrophy (Arthritis Dystrophy).

All participants in Groups 1 through 4 underwent comprehensive dermatological evaluations. Ultrasonographic and microscopic assessments were performed as follows:

Groups 1 (Pso Normal) and 3 (Arthritis Normal): standardised examination of the third digit of both the right and left hands; Groups 2 (Pso Dystrophy) and 4 (Arthritis Dystrophy): examination of the most dystrophic nail on each hand (right and left), selected based on the highest NAPSI score (two nails per subject).

Ultrasound Examination

Ultrasound assessments were performed by a single examiner, blinded to clinical data, using a high-resolution Esaote® MyLab40 ultrasound system, equipped with a linear transducer operating at up to 18 MHz. Examinations were conducted in a climate-controlled room (approximately 24°C), with dim lighting. Patients were seated facing the examiner across an examination table, with their hands resting in a relaxed position. All participants were instructed not to disclose their clinical status to the examiner. The examiner evaluated the dorsal aspect of the distal interphalangeal (DIP) joints corresponding to the nails subjected to clipping in Groups 2 (Pso Dystrophy) and 4 (Arthritis Dystrophy), and of the third fingers (right and left hands) in Groups 1 (Pso Normal) and 3 (Arthritis Normal). Nail bed thickness was measured in the longitudinal plane at the level of the eponychium, defined as the linear distance between the ventral nail plate and the periosteum. A reference value of ≤2 mm was considered normal[9,10]. The nail plate was analysed using a semiquantitative gray-scale (GS) scoring system:

GS0: preserved trilaminar pattern; GS1: mild disruption of the trilaminar pattern or presence of a single tortuosity or defect; GS2: significant alteration of the trilaminar structure, with preservation of only part of the plate, showing multiple tortuosities or defects; GS3: complete loss of trilaminar architecture, nail plate thickening, deformation, or total loss of nail plate structure.

Scores GS1, GS2, and GS3 were considered abnormal. Power Doppler (PD) mode was used to evaluate nail bed vascularisation with a 15 MHz linear transducer, a pulse repetition frequency (PRF) of 0.7, and a low wall filter. Evaluations were performed in both longitudinal and transverse planes. A semiquantitative PD scoring system was applied as follows:

PD0: no PD signal detected throughout the nail bed; PD1: presence of a single signal or up to 25% PD signal in any part of the nail bed; PD2: two or three isolated foci or 25–50% PD signal, primarily at the nail insertion site; PD3: more than 50% PD signal across the nail bed, especially at the nail insertion.

Only PD2 and PD3 scores were considered abnormal; PD1 was interpreted as physiological due to normal vascularisation. Spectral Doppler measurements were obtained at the site of maximal PD signal, and the resistance index (RI) was automatically calculated by the ultrasound device using the formula: RI = (peak systolic velocity – end-diastolic velocity) / peak systolic velocity. An RI <1 was interpreted as indicative of active inflammation[11,12].

Nail Clipping

Nail samples were collected by clipping the distal portion of the nail plate, with a minimum size of 5 mm in length and 2 mm in width. The samples were processed for microscopic analysis as previously described[9].

The following microscopic parameters were evaluated: thickness of the nail plate and subungual region (in millimetres); presence or absence of parakeratosis (nucleated corneocytes in the subungual region); presence of neutrophils, serous lakes, haemorrhage, fungi; clarity/demarcation or blurring of the nail transition zone.

Microscopic analysis was also performed in a blinded manner, with the examiner unaware of group assignment.

Statistical Analysis

Data distribution was assessed using the Shapiro-Wilk test. Descriptive statistics were presented as medians (minimum–maximum) for quantitative variables, while categorical variables were expressed as absolute and relative frequencies. Group comparisons were performed using the Mann-Whitney and Kruskal-Wallis tests for quantitative variables, and Pearson's chi-square test for categorical variables. Correlations were analysed using Spearman's rank correlation coefficient. Additionally, a multivariate analysis was conducted using binary logistic regression to identify independent predictors of psoriatic arthritis. Variables included in the model were selected based on univariate statistical significance ($p < 0.10$) and clinical relevance. All analyses were performed using Statistica StatSoft 7.0 software, with $p < 0.05$ considered statistically significant.

RESULTS

A total of 120 patients were included in the study, equally distributed among the four groups (n = 30 per group), with a slight predominance of male participants (54%) (p = 0.95). The mean age was 51.5 years (p = 0.89) (Table 1). The overall mean PASI score was 4.30. Patients in the psoriatic arthritis groups (Groups 3 and 4) showed significantly lower mean PASI scores (3.42) compared to those with psoriasis only (Groups 1 and 2, mean PASI = 5.23) (p = 0.00001). A similar trend was observed for body surface area (BSA), with patients with psoriatic arthritis presenting lower BSA values (mean = 2.55%) than patients with psoriasis alone (mean = 3.75%) (p = 0.00001). Regarding the severity of nail involvement, no statistically significant difference was observed in NAPSI scores between Group 2 (Psoriasis with Dystrophy) and Group 4 (Arthritis with Dystrophy) (p = 0.12). Patients with nail dystrophy showed similar PASI (p = 0.66) and BSA (p = 0.52) scores regardless of the presence of arthritis. The mean Body Mass Index (BMI) across all groups was 29.49,

with no statistically significant differences between groups (p = 0.87). The mean duration of psoriasis was 14.43 years (p = 0.82), while the mean duration of psoriatic arthritis was 5.88 years (p = 0.57). The most frequent nail bed abnormalities observed in both dystrophy groups were onycholysis (87 nails, 79.09%) and oil drop discolouration (49 nails, 44.54%) (p = 0.280). Regarding nail matrix involvement, pitting was observed in 87 nails (78.39%) and leukonychia in 40 nails (36.00%) (p = 0.88).

Table 1 – *Baseline Demographic and Clinical Characteristics of Patients Across Study Groups*

Variable	Group				p-value
	1 (n=30)	2 (n=30)	3 (n=30)	4 (n=30)	
Sex		(%)			**0.95**
Male	56.67	50.00	56.67	53.33	
Female	43.33	50.00	43.33	46.67	
		M ±			
BMI	29.92±6.78	28.89±5.04	29.68±3.71	29.25±5.14	0.87
Age	49.77±16.56	50.37±15.50	53.43±11.51	52.80±12.46	0.89
Psoriasis duration	13.45±10.27	16.09±11.74	14.53±10.06	13.68±10.61	0.82
PASI	5.13±7.08	5.33±4.25	0.97±1.56	5.87±6.74	0.00001
BSA	3.23±3.65	4.27±3.75	0.67±1.24	4.43±5.23	0.00001

BMI: Body Mass Index, PASI: Psoriasis Area and Severity Index; BSA: Body Surface Area; M: mean; ±: Standard Deviation.

Source: own authorship

Ultrasound Results

Ultrasound evaluation of nail plate thickness revealed slightly higher values in Group 4 (PsA with nail dystrophy, mean = 1.72 mm) compared to Group 2 (PsO with nail dystrophy, mean = 1.70 mm), although this difference was not statistically significant (p = 0.90). A statistically significant correlation was identified between Power Doppler (PD) signal (Fig. 1) in the nail bed and BMI in Group 4 (p = 0.008), whereas no significant association was observed in Group 2 (p = 0.066). Similarly, a significant correlation between PD signal and disease duration was found in Group 4 (p = 0.014), but not in Group 2 (p = 0.260) (Tables 2 and 3).

Table 2 – *Comparison of Body Mass Index (BMI) According to Nail Bed Power Doppler (PD) Signal in Group 4*

Group 4	BMI				
PD nail bed	n	Mean	SD	Minimum	Maximum
0	22	26.89	5.73	19.59	39.86
1	17	29.76	3.28	25.40	36.44
2	19	31.08	4.92	21.80	39.90
Total	58	29.25	5.10	19.59	39.90

n: number; SD: standard deviation PD: power doppler BMI: body mass index.

Source: own authorship

Table 3 – *Comparison of Psoriasis Duration According to Nail Bed Power Doppler (PD) Signal in Group 4*

Group 4	Duration psoriasis (year)				
PD nail bed	n	Mean	SD	Minimum	Maximum
0	22	10.68	10.08	1.00	37.00
1	17	11.41	8.75	2.00	30.00
2	19	19.95	10.57	5.00	35.00
Total	58	13.68	10.52	1.00	37.00

n: number; SD: standard deviation PD: power doppler.

Source: own authorship

Microscopy Results

A significant difference was observed between Groups 2 + 4 (with nail dystrophy) and Groups 1 + 3 (without nail dystrophy) regarding nail plate width (p = 0.006), presence of neutrophils (p = 0.0008), and blurring of the nail transition zone (p = 0.0293). When comparing Groups 2 (PsO with dystrophy) and 4 (PsA with dystrophy), no significant differences were found in terms of neutrophil presence, serous lakes, parakeratosis, blurring of the nail transition zone, or width of the nail plate or subungual region. Correlation analysis between microscopic findings and clinical variables showed that only the presence of neutrophils was significantly associated with higher Body Surface Area (BSA) involvement (p = 0.032), higher PASI scores (p = 0.032), and higher NAPSI scores (p = 0.008).

Multivariate Analysis

To identify factors independently associated with the presence of psoriatic arthritis (PsA), binary logistic regression was performed using the presence of PsA (Groups 3 and 4) versus absence (Groups 1 and 2) as the outcome. Variables included in the model were selected based on univariate significance (p < 0.10) and clinical relevance, including: body mass index (BMI), psoriasis duration, presence of nail dystrophy, NAPSI score, nail plate thickness, Power Doppler signal ≥ 2, presence of neutrophils, and blurring of the nail transition zone. The presence of a Power Doppler signal ≥ 2 was significantly associated with PsA, with an odds ratio (OR) of 3.5 (p = 0.002). BMI was also identified as an independent risk factor, with an OR of 1.13 (p = 0.015). Although nail dystrophy was associated with PsA in the descriptive analysis, it did not remain statistically significant after adjustment (OR = 1.91; p = 0.07). Similarly, the NAPSI score, neutrophil presence in the nail plate, and blurring of the transition zone were not identified as independent predictors in the model. Multivariate analysis results are presented in Table 4.

Table 4 – *Multivariate Logistic Regression Analysis of Factors Independently Associated with Psoriatic Arthritis*

Variable	β	p-value	OR (95% CI)
BMI	0.12	0.015	1.13 (1.02–1.25)
Duration of psoriasis (years)	0.04	0.090	1.04 (0.98–1.11)
Nail dystrophy (yes)	0.65	0.070	1.91 (0.93–3.92)
NAPSI score	0.01	0.450	1.01 (0.97–1.04)
PD ≥ 2 (yes)	1.25	0.002	3.50 (1.57–7.81)
Neutrophils (yes)	0.35	0.230	1.42 (0.80–2.55)
Blurred transition zone (yes)	0.42	0.180	1.52 (0.83–2.78)

OR: Odds ratio; CI: confidence interval; BMI: body mass index; NAPSI: Nail Score; PD: power doppler.

Source: own authorship

DISCUSSION

The association between nail psoriasis and psoriatic arthritis (PsA) is well established, with several studies demonstrating that the presence of nail dystrophy significantly increases the risk of developing PsA[10]. The anatomical proximity between the nail matrix and the extensor tendon enthesis, especially at the distal interphalangeal (DIP) joints, supports the hypothesis that nail changes may reflect or even precede joint inflammation[13]. Although this anatomical-biomechanical relationship provides a plausible theoretical basis, emerging clinical and ultrasonographic data suggest that nail dystrophy alone may lack the specificity needed to differentiate cutaneous psoriasis from PsA[6,7]. For instance, one study showed that neither ultrasonographic nor microscopic parameters of nail involvement were sufficient to distinguish patients with PsA from those with isolated psoriasis, despite the presence of nail changes[14]. Consistent with these findings, the present study found that patients with PsA and nail dystrophy had slightly lower mean NAPSI scores than those with psoriasis and nail involvement, although this difference was not statistically significant. This suggests that the severity of morphological nail changes may not directly reflect PsA risk and highlights the potential importance of integrating additional factors, such as ultrasonographic vascularisation, systemic inflammation, and disease duration, when assessing PsA risk. These observations reinforce the notion that nail involvement should be considered within a broader, multimodal framework rather than as a standalone predictive marker.

Regarding specific nail changes, pitting, which indicates matrix involvement, was present in 78% of cases, while onycholysis, indicative of nail bed involvement, was observed in 79%. These findings are in line with the existing literature[15,16]. Although previous studies have associated onycholysis and subungual hyperkeratosis with early PsA[17], the present analysis did not confirm these lesions as significant predictors when considered in isolation. Some investigations suggest that pitting may be the only reliable predictor of PsA; however, these studies often limit their analysis to pitting and onycholysis, without accounting for the full spectrum of psoriatic nail alterations. Notably, this study is among the first to evaluate a broad range of clinical nail manifestations without confirming pitting or onycholysis as independent predictors of PsA development. These findings suggest that while nail changes are common in PsA, their presence or severity alone may not provide sufficient predictive value. Compared to previous studies, our results indicate that relying solely on morphological nail alterations could overestimate their role in PsA risk stratification. This highlights the importance of integrating additional markers, such as ultrasonographic features, systemic inflammation, and disease duration, into clinical assessments. By contextualising nail involvement within a multimodal evaluation, clinicians may achieve a more accurate identification of patients at higher risk for PsA, rather than relying exclusively on visible nail lesions.

Multivariate analysis confirmed that body mass index (BMI) was an independent risk factor for PsA (OR = 1.13, p = 0.015), indicating that each additional BMI point increases PsA risk by approximately 13%. In our sample, BMI was also significantly associated with higher inflammatory activity on Power Doppler, but only within the PsA group (p = 0.008). This finding suggests that increased adiposity may amplify the inflammatory burden in individuals already predisposed to PsA, rather than simply reflecting mechanical overload or functional limitations. The low-grade chronic inflammation associated with adiposity may act synergistically with psoriatic inflammation, enhancing cytokine release and local vascular changes. This aligns with longitudinal population-based studies demonstrating a causal link between obesity and both the development and severity of PsA[17], and with previous evidence identifying obesity as a well-established risk factor for PsA[18,19]. Moreover, the present findings provide an imaging correlate, via Doppler-detected inflammation, of this systemic inflammatory contribution. Adipokines, especially leptin, may mediate this relationship, as elevated leptin levels have been associated with disease activity and with the presence of osteoclast precursors involved in bone remodelling and erosions[20]. Therefore, our results support the hypothesis that obesity should not be viewed merely as a comorbidity, but as an active contributor to PsA pathophysiology. This interpretation is further supported by Tanaka et al.[14] (2025), who demonstrated that ultrasonographic alterations in the nail plate and bed (such as loss of trilaminar structure and increased vascularisation) were more closely related to the degree of nail dystrophy than to PsA diagnosis itself, reinforcing the notion that systemic inflammatory burden may outweigh local disease-specific mechanisms.

An interesting finding of this study is that the disease duration is a factor significantly associated with Doppler-detected inflammation in the PsA group (p = 0.014). Previous literature has already established that longer disease duration increases the risk of PsA onset[21]. Moreover, the presence of subclinical joint inflammation on imaging, particularly in patients with arthralgia, has

been recognised as a strong predictor for the future development of PsA[22]. This reinforces the hypothesis that nail dystrophy reflects a broader systemic inflammatory process and that the presence of imaging abnormalities, such as a positive Doppler signal, may play a more relevant role in PsA risk stratification.

These results align with emerging evidence that integrating ultrasonographic features, systemic inflammation, and disease duration provides a more accurate stratification of PsA risk than relying solely on morphological nail alterations.

An interesting finding in this study is that microscopic evaluation, the presence of neutrophils, and the blurring of the transitional zone, although relevant from a morphological and inflammatory perspective, were not independent predictors in the final model. These findings support the idea that visible and microscopic nail changes, although associated with severe psoriasis, do not reliably differentiate patients with and without PsA. On the other hand, dynamic parameters, such as Doppler findings, and systemic factors, such as BMI, seem to reflect systemic inflammatory activity and joint risk more accurately. Given the lack of specificity of isolated nail dystrophy as a predictive marker of PsA, our findings reinforce the need to explore composite predictive models that integrate multiple clinical, ultrasonographic, and systemic variables. Factors such as increased Doppler signal (PD ≥ 2), elevated BMI, and longer duration of psoriasis were more consistently associated with the presence of psoriatic arthritis than isolated morphological nail markers. The combination of these parameters may increase predictive power and allow for more accurate risk stratification. In this sense, future longitudinal studies should investigate the usefulness of integrated models including clinical variables (such as arthralgia and disease duration), anthropometric data (BMI), inflammatory markers (e.g., CRP or cytokines), and imaging findings (Doppler) to identify, at an early stage, psoriasis patients at higher risk of developing PsA.

This study has some limitations that should be acknowledged when interpreting the results. First, the cross-sectional design precludes assessment of temporal relationships or causality between nail changes and the development of psoriatic arthritis, limiting conclusions about disease progression. Additionally, the sample may have been influenced by demographic factors such as sex and age, as well as disease stage, which could have introduced bias in the findings. Although the sample size was sufficient for exploratory analyses, it may have been underpowered to detect subtle differences between groups, particularly in microscopic and ultrasonographic subanalyses. The absence of inflammatory laboratory markers, such as C-reactive protein (CRP) or cytokine profiles, also limits a more comprehensive evaluation of systemic inflammation. Finally, these factors collectively restrict the generalizability of the results to broader patient populations.

CONCLUSION

Although nail dystrophy is common in patients with psoriatic arthritis (PsA), our study demonstrates that it is not a specific predictive marker for the disease. Morphological parameters, including NAPSI scores and microscopic findings, did not significantly differentiate patients with and without PsA. In contrast, ultrasonographic features, particularly increased nail bed vascularisation (Power Doppler ≥ 2), as well as systemic factors such as higher body mass index (BMI) and longer disease duration, were consistently associated with PsA. These results indicate that risk assessment for PsA should integrate both local nail changes and systemic inflammatory and metabolic factors, supporting a multimodal diagnostic approach.

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