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Original Research Article

Histopathological correlation of diagnostic parameters in psoriasis: A prospective observational study

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Abstract

Background: Psoriasis is a chronic immune-mediated skin disorder characterized by abnormal keratinocyte proliferation, inflammation, and dermal vascular changes. Histological features are well documented, but the correlations among them provide important clues to disease mechanisms and therapeutic opportunities.
Aim: To analyze and correlate key histopathological parameters in psoriasis to better understand their role in disease pathogenesis.
Materials and Methods: This prospective observational study included 50 histologically confirmed cases of psoriasis. Eight histological parameters were evaluated: hyperkeratosis, parakeratosis, suprapapillary thinning, inflammatory infiltrate, capillary proliferation, Munro's microabscesses, pustules of Kogoj, and widened rete ridges. Features were graded on a visual analogue scale, and Spearman's correlation coefficient was used for analysis.
Results: Hyperkeratosis correlated strongly with parakeratosis (r = 0.62) and pustules of Kogoj (r = 0.65). Parakeratosis also correlated with Munro's abscesses, suprapapillary thinning, and widened rete ridges. Munro's microabscesses and Kogoj's pustules were closely associated (r = 0.77). Inflammatory infiltrate correlated strongly with capillary proliferation (r = 0.72), which also associated with suprapapillary thinning.
Conclusion: Significant correlations among histopathological parameters confirm that immune activation and angiogenesis are central to psoriasis pathogenesis. Findings support the involvement of T-cell mediated inflammation and suggest that histological patterns can inform targeted therapies and disease monitoring strategies.

Keywords: Psoriasis, Histopathology, Parakeratosis, Capillary proliferation, Immune-mediated inflammation

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1. Introduction

Psoriasis is a chronic, immune-mediated inflammatory dermatosis that affects approximately 0.5% to 1.5% of the global population.[1] Clinically, it presents with erythematous, scaly plaques, most commonly on extensor surfaces. Beyond its cutaneous manifestations, psoriasis is increasingly recognized as a systemic disease with significant comorbidities including psoriatic arthritis, metabolic syndrome, cardiovascular disease, and depression.[2,3] The chronic inflammatory state in psoriasis is thought to contribute to these systemic associations.

Histopathologically, psoriatic lesions demonstrate a spectrum of epidermal and dermal changes, including hyperkeratosis, parakeratosis, suprapapillary thinning, elongation of rete ridges, neutrophilic collections (Munro's microabscesses and Kogoj's pustules), inflammatory infiltrate, and increased dermal capillaries.[4,5] While these features are individually well described, fewer studies have attempted to assess their interrelationships systematically. Understanding such correlations may provide insight into the sequence of pathogenic events and may also have diagnostic and prognostic value.

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At the molecular level, psoriasis is driven by a dysregulated immune response characterized by activation of dendritic cells, Th1 and Th17 lymphocytes, and the release of pro-inflammatory cytokines such as TNF-α, IL-17, and IL-23.[6,7] These cytokines stimulate keratinocyte hyperproliferation, angiogenesis, and leukocyte recruitment, changes which are mirrored in histological features.

The present study was undertaken to evaluate correlations among major histopathological parameters of psoriasis. By identifying significant associations, we aim to reinforce the immunopathogenic model of the disease and highlight the morphologic insights that may aid clinicians in diagnosis, monitoring, and therapeutic decision-making.

2. Materials and Methods

This prospective study was conducted in the Department of Pathology from June 2018 to July 2019. A total of 50 consecutive skin biopsy specimens with histologically confirmed psoriasis were included.

2.1. Histological assessment

Biopsies were processed and stained with hematoxylin and eosin (H&E). The following parameters were evaluated: epidermal hyperplasia, suprapapillary thinning, inflammatory infiltrate, capillary proliferation, parakeratosis, Munro's microabscesses, pustules of Kogoj, and widened rete ridges. Grading was performed using a semi-quantitative scale (1 to 3 for mild, moderate, and marked changes). Munro's abscesses and Kogoj's pustules were noted as present or absent.

2.2. Statistical analysis

Spearman's correlation coefficient was used to assess the strength of association between histological features. A correlation value above 0.6 was considered strong.

3. Results

The study population comprised 50 patients, aged between 9 and 76 years, with a mean age of 41.6 years. All skin biopsy samples exhibited classical histopathological features of psoriasis.

Hyperkeratosis was observed universally and showed a strong positive correlation with parakeratosis (r = 0.62), suggesting concurrent keratinocyte hyperproliferation and impaired terminal differentiation. A notable correlation was also found between hyperkeratosis and the presence of Kogoj's pustules (r = 0.65), indicating the association between epidermal thickening and intraepidermal neutrophilic collections.

Parakeratosis was significantly associated with several parameters: Munro's microabscesses, suprapapillary thinning, and widened rete ridges. These findings reflect the multifaceted nature of disturbed keratinization in psoriatic lesions. Munro's microabscesses and pustules of Kogoj, both indicative of neutrophilic infiltration, were frequently coexistent and exhibited a strong correlation (r = 0.77), supporting their linked pathogenic mechanism.

Inflammatory infiltrate in the dermis showed a robust correlation with capillary proliferation (r = 0.72), highlighting the close interplay between immune activation and vascular response. Capillary proliferation also correlated significantly with suprapapillary thinning, further underscoring the angiogenic component of psoriatic pathology.

Overall, the data demonstrate a network of statistically significant correlations among classical histopathological features, reinforcing the complex but coordinated histological architecture underlying the pathogenesis of psoriasis.

Table 1: Frequency of histopathological features (n = 50)

Feature	Frequency (n)	Percentage (%)
Hyperkeratosis	50	100%
Parakeratosis	48	96%
Suprapapillary Thinning	43	86%
Inflammatory Infiltrate	47	94%
Capillary Proliferation	45	90%
Munro's Microabscesses	40	80%
Pustule of Kogoj	38	76%
Widened Rete Ridges	42	84%

Table 2: Spearman correlation between various histopathological parameters in psoriasis

Parameters	Hyperkeratosis	Parakeratosis	Munro's Microabscess	Pustule of Kogoj	Suprapapillary Thinning	Inflammatory Infiltrate	Capillary Proliferation	Widening of Rete Ridges
Hyperkeratosis	1	0.62	0.39	0.65	0.33	0.34	0.48	0.53
Parakeratosis	0.62	1	0.70	0.77	0.72	0.51	0.58	0.89
Munro's Microabscess	0.39	0.70	1	0.62	0.47	0.37	0.36	0.41
Pustule of Kogoj	0.65	0.77	0.62	1	0.31	0.33	0.43	0.56
Suprapapillary Thinning	0.33	0.72	0.47	0.31	1	0.46	0.54	0.71

Inflammatory Infiltrate	0.34	0.51	0.37	0.33	0.46	1	0.72	0.56
Capillary Proliferation	0.48	0.58	0.36	0.43	0.54	0.72	1	0.59
Widening of Rete Ridges	0.53	0.89	0.41	0.56	0.71	0.56	0.59	1

Note: Bold values represent significant correlations (r > 0.6).

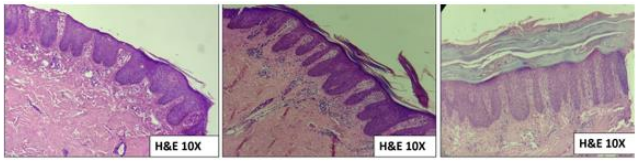

Figure 1: Grades of hyperkeratosis, mild, moderate, marked.

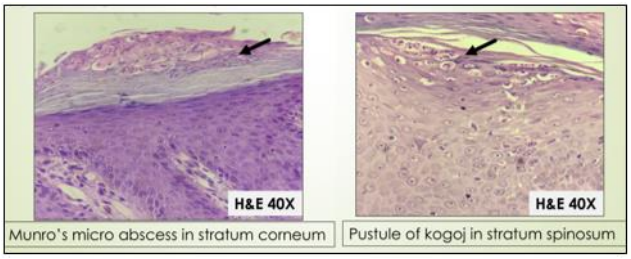

Figure 2: Munro's micro abscess and pustule of Kogoj.

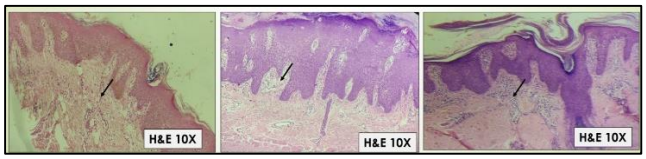

Figure 3: Inflammatory infiltrate: mild, moderate, and marked.

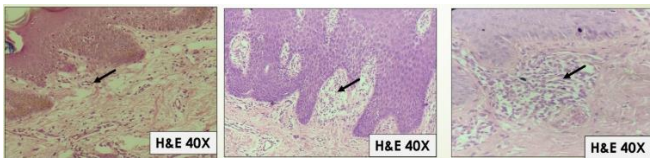

Figure 4: Capillary proliferation: Mild, moderate and marked.

4. Discussion

The findings of this study emphasize the intricate interrelationships among the histopathological features of psoriasis and support the concept of a multifactorial pathogenesis. The observed correlation between hyperkeratosis and parakeratosis aligns with the known pathophysiology wherein rapid keratinocyte proliferation leads to incomplete differentiation and nuclear retention in the stratum corneum.[6] These epidermal changes also correspond with increased neutrophilic activity, as reflected by the significant association with pustules of Kogoj.

The co-occurrence of Munro's microabscesses and Kogoj's pustules, with a strong correlation coefficient, highlights neutrophil migration through the epidermis as a central feature of psoriatic pathology. This phenomenon is likely mediated by IL-8, CXCL1, and other neutrophil-attracting chemokines secreted by activated T cells and keratinocytes.[8] Neutrophils themselves are not merely bystanders; they release proteases and reactive oxygen species that further injure the epidermis, perpetuating a cycle of inflammation and tissue remodeling. Our results, therefore, align with growing evidence that innate immune mechanisms play an essential role alongside adaptive responses in psoriasis.

Another important observation is the strong correlation between inflammatory infiltrates and capillary proliferation. This finding supports the view that psoriasis is not only an inflammatory but also an angiogenic dermatosis. Activated T cells and dendritic cells release vascular endothelial growth factor (VEGF), angiopoietins, and other proangiogenic mediators, which promote dermal vascular remodeling.[5,12] These new vessels are often dilated and tortuous, facilitating trafficking of immune cells into lesional skin. The close link between capillary proliferation and suprapapillary thinning suggests that vascular expansion may contribute mechanically and metabolically to the epidermal alterations seen in psoriasis. From a therapeutic standpoint, the evidence for angiogenesis as a driver of psoriatic pathology opens the door to anti-angiogenic strategies, either as standalone therapies or as adjuncts to biologics.[13]

4.1. Comparison with prior studies

Our findings are broadly consistent with earlier Indian and international studies that have evaluated histopathological parameters in psoriasis. Moorchung et al.[6] highlighted the frequent coexistence of parakeratosis, neutrophilic microabscesses, and suprapapillary thinning, which they proposed as a diagnostic triad. Similarly, Bai et al.[7] emphasized the diagnostic value of Munro's microabscesses and parakeratosis, which also showed significant correlations in our cohort. By systematically analyzing multiple parameters within the same patient group, our study extends these observations and demonstrates the interconnectedness of epidermal, dermal, and vascular changes.

More recent reviews have shifted focus toward immunological underpinnings, particularly the IL-23/Th17 axis, which is now considered central to psoriasis pathogenesis.[9,11,12] The histological features we observed—keratinocyte hyperplasia, parakeratosis, and neutrophilic infiltration—can be interpreted as morphological outcomes of IL-17 and IL-22 activity. These cytokines stimulate keratinocyte proliferation and impair differentiation, while simultaneously inducing angiogenic factors such as VEGF, thereby linking immune dysregulation with vascular changes. Our correlations between inflammatory infiltrates and capillary proliferation reinforce these molecular insights.

Armstrong and Read[12] have underscored that vascular alterations contribute not only to lesion persistence but also to systemic inflammatory burden. Campanati et al.[13] further elaborated on the role of angiogenesis in maintaining the chronicity of plaques and proposed that targeting angiogenesis could represent an adjunctive therapeutic strategy. Our study supports these observations by showing a strong histological association between inflammatory infiltrates and dermal vascular proliferation, providing direct morphological evidence of this immune–vascular interplay.

Taken together, these comparisons suggest that psoriasis should not be regarded merely as a keratinocyte proliferation disorder. Instead, it is best understood as a complex immunovascular disease in which epidermal, dermal, and vascular changes reinforce one another. By corroborating earlier studies and aligning with modern immunological concepts, our findings contribute to a growing body of evidence that histopathology can bridge clinical presentation with underlying molecular mechanisms, thereby guiding both diagnosis and therapy

4.2. Clinical implications

The practical implications of our study are significant. First, histopathological correlations provide diagnostic support in challenging cases, particularly when psoriasis must be differentiated from chronic dermatitis, pityriasis rubra pilaris, or drug-induced eruptions. For example, the presence of concurrent parakeratosis, neutrophilic abscesses, and vascular proliferation strongly favors psoriasis over eczematous dermatitis, where spongiosis predominates.

Second, histological correlations may have therapeutic relevance. Patients with marked vascular proliferation and dense inflammatory infiltrates might respond more favorably to biologics targeting IL-17 or IL-23, which reduce angiogenic signaling as well as immune activation.[14] Conversely, in resource-limited settings where biologics are not accessible, histological severity could guide the intensity of conventional systemic therapy. Thus, integrating histopathology into clinical decision-making supports the broader movement toward precision medicine in dermatology.

Third, recognition of psoriasis as a systemic inflammatory disease is critical. Histological features such as inflammatory infiltrates and angiogenesis not only explain cutaneous changes but may also mirror systemic inflammation that contributes to comorbidities like cardiovascular disease, obesity, and diabetes.[4,12] This strengthens the case for early, aggressive management of psoriasis to reduce long-term systemic morbidity.

4.3. Future perspectives

The field of dermatopathology is evolving rapidly, and several avenues exist to expand on our findings. Molecular pathology techniques, such as immunohistochemistry for cytokines (IL-17, IL-23, TNF-α) or angiogenic markers (VEGF, CD31, angiopoietin), can validate histological correlations and provide mechanistic insights. Advances in digital pathology and machine learning may allow quantitative assessment of epidermal thickness, microvascular density, and inflammatory infiltrates, enabling objective and reproducible analysis. Such approaches could facilitate prognostication and personalized treatment selection.

Furthermore, longitudinal studies incorporating serial biopsies could clarify how histological correlations evolve with treatment, shedding light on which parameters best predict therapeutic response. Integration of histological, molecular, and clinical data in multi-omic approaches could redefine psoriasis not only as a clinical diagnosis but also as a biologically stratified entity.[15,16]

5. Conclusion

The present study demonstrates that psoriasis is a multifactorial disease with a strong immunological and angiogenic basis. Correlation among histopathological features such as parakeratosis, neutrophilic infiltrates, and capillary proliferation reinforces the central role of T-cell-mediated inflammation and vascular proliferation. By highlighting the interplay of epidermal and dermal changes, our findings provide a morphologic rationale for current targeted therapies and open avenues for novel therapeutic approaches that address both immune and vascular pathways.

From a clinical perspective, histopathology remains indispensable. It not only aids diagnosis in early or atypical cases but also provides prognostic insights. For example, marked vascular proliferation and dense inflammatory infiltrates may predict stronger responses to biologics targeting IL-17 and IL-23, while keratinocyte-predominant changes underline the value of therapies focused on epidermal turnover. Histopathology therefore bridges morphology with precision medicine in dermatology.

The research implications are equally important. Combining histology with molecular and immunohistochemical profiling could identify biomarkers predictive of severity, systemic risk, or treatment response. This is crucial because psoriasis is strongly linked with comorbidities such as cardiovascular disease, obesity, and psychiatric disorders. Emerging tools like digital pathology and AI-based image analysis may enable quantitative, reproducible assessment of histological features in the future.

Future studies with larger cohorts and integration of molecular techniques may further refine our understanding of psoriasis pathogenesis. Such research could help bridge the gap between histological observations and precision medicine, ultimately improving patient outcomes.

6. Source of Funding

None.

7. Conflict of Interest

None.

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