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CASE REPORT

Lupus or psoriasis? The blurring boundary: a case report

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ABSTRACT

Psoriasis (Ps) and systemic lupus erythematosus (SLE) are immune-mediated disorders traditionally considered distinct entities with differing immunopathogenic pathways. However, emerging evidence reveals shared genetic susceptibilities and overlapping immune mechanisms, leading to rare but clinically significant co-occurrence syndromes. This overlap poses diagnostic and therapeutic challenges, given the contrasting management approaches for each condition. To present a detailed case of SLE with overlapping psoriatic lesions and review current understanding of shared pathogenesis, clinical presentation, and management strategies. Clinical, laboratory, and sequential histopathologic findings of a 47-year-old woman with a long history of SLE who developed atypical cutaneous lesions suggestive of both lupus and psoriasis were analyzed. A thorough literature review was conducted, focusing on genetic predisposition, immunological pathways, and therapeutic options for patients exhibiting features of both diseases. Consent for publication has been obtained from the patient. The patient presented with annular, scaly plaques affecting the skin of her body, scalp, and nails. Laboratory tests confirmed active SLE. Sequential skin biopsies revealed histopathological features of both psoriasis and subacute cutaneous lupus erythematosus (SCLE). Escalation of hydroxychloroquine initially undertaken in the context of severe flare paradoxically worsened the skin eruption, highlighting therapeutic complexity. Shared immunopathogenic mechanisms include activation of the IL-17/Th17 axis, type I interferon signature, innate immune dysregulation, and shared genetic susceptibility loci (PTPN22, STAT4, and TNIP1). Management requires individualized strategies that balance immunosuppressants and biologics, with vigilant monitoring. Although rare, the coexistence of psoriasis and SLE represents a clinically important overlap syndrome with shared immunopathogenic pathways. Enhanced awareness is critical for accurate diagnosis and tailored management.

Keywords: Psoriasis, Systemic Lupus Erythematosus, Overlap Syndrome, Histopathology, Hydroxychloroquine.

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1. INTRODUCTION

The skin, as the largest organ of the human body, serves as a critical interface between environmental factors and the immune system, playing a central role in both innate and adaptive immunity (1,2). Disruption of this balance may result in chronic inflammatory and autoimmune dermatoses, including psoriasis (Ps) and systemic lupus erythematosus (SLE), which are traditionally regarded as distinct immune-mediated diseases (3).

Psoriasis is a common chronic inflammatory disorder that most frequently presents as well-demarcated erythematous plaques with silvery scales, driven primarily by the IL-23/IL-17/TNF-α axis (4). SLE is a multisystem autoimmune disease characterized by loss of

immune tolerance, autoantibody production, and diverse clinical manifestations, including polymorphic cutaneous lesions often associated with photosensitivity (5). Cutaneous involvement is among the most frequent SLE manifestations and can present with a wide spectrum of lesions, including acute malar rash, subacute cutaneous lupus erythematosus (SCLE), and chronic discoid lupus (6).

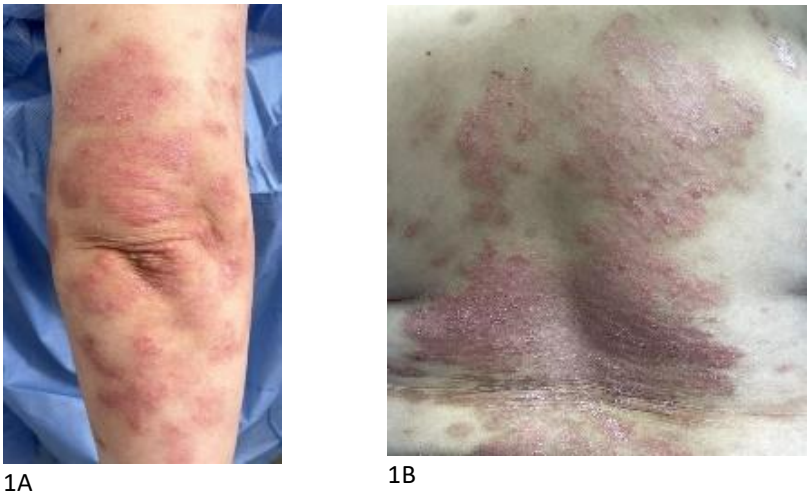
Although these conditions differ in classical pathogenic pathways, emerging evidence suggests possible overlap, with reports of coexistence in the same patient (7). Population-based studies estimate the prevalence of psoriasis among SLE patients to range from 0.69% to 4.9%, exceeding expected rates by chance alone, suggesting shared biologic mechanisms (2). This association poses diagnostic and therapeutic challenges, as treatments effective for one disease may exacerbate the other. Hydroxychloroquine (HCQ), a cornerstone of SLE management, has been reported to trigger or worsen psoriatic eruptions (8,9).

We report a case that illustrates features rarely documented together: the possible contribution of prior rituximab exposure through Th1/Th17 cytokine rebalancing to the emergence of psoriasiform lesions in a patient with established SLE; a clinicopathologically confirmed paradoxical worsening of psoriasis following HCQ dose escalation during a severe systemic flare; and the successful application of a multimodal diagnostic approach (combining dermoscopy, sequential skin biopsies, and direct immunofluorescence) to establish a dual diagnosis in real-world clinical practice.

2. CASE REPORT

A 47-year-old woman with a 17-year history of SLE (fulfilling ACR/EULAR 2019 classification criteria (10)), with photosensitivity, cutaneous involvement, non-deforming non-destructive arthritis, and pancytopenia, was followed in our department. Maintenance therapy included methotrexate 20 mg weekly, hydroxychloroquine 400 mg daily, and methylprednisolone 4 mg daily. She had previously received rituximab for refractory pancytopenia; this achieved hematological remission but provided only partial relief of articular symptoms. Notably, the introduction of rituximab, by depleting B cells and shifting the Th1/Th17 cytokine balance, may have contributed to the subsequent emergence of psoriasiform inflammation, a mechanism warranting prospective investigation.

During follow-up, she developed diffuse annular erythematous scaly plaques initially predominantly over photo-exposed areas and subsequently spreading to the entire body (figures 1(A) and 1(B)), with concurrent involvement of the scalp and nails. The clinical presentation suggested features of both psoriatic and lupus-related cutaneous involvement, significantly complicating the clinical diagnostic assessment. Concurrent alopecia, mucosal ulcers, and arthritis affected the wrists, metacarpophalangeal joints, and ankles, which were also assessed. The patient also presented with diffuse, recurrent headaches without other disorders, with a normal neurological examination and repeated normal MRI angiography.



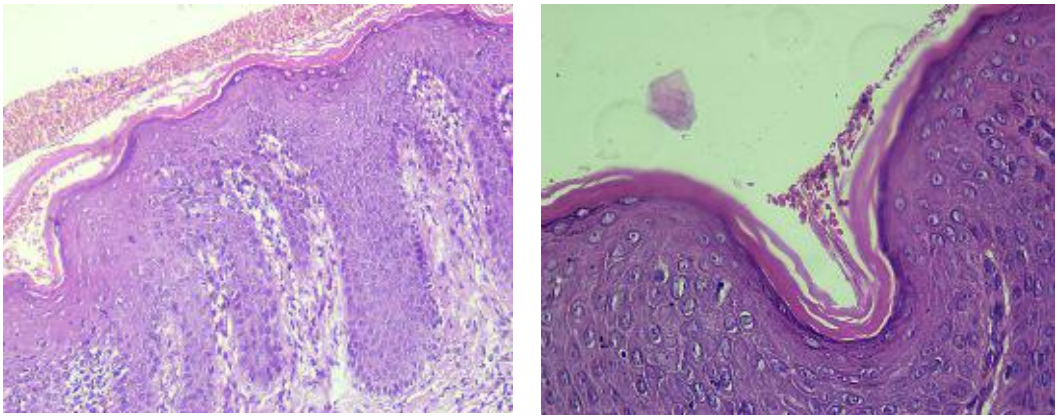
1A 1B

Figure 1. Coexistence of psoriatic and lupus-specific cutaneous lesions. **(A)** Extensor surface of the elbow showing well-demarcated erythematous plaques with thick, adherent silvery-white scale and Auspitz sign on gentle scraping, morphologically consistent with psoriasis. **(B)** Upper back showing confluent erythematous-to-violaceous plaques with ill-defined borders, superficial atrophy, subtle follicular plugging, and fine adherent scale, morphologically consistent with subacute cutaneous lupus erythematosus (SCLE), and contrasting with the well-demarcated, thick-scaled psoriatic plaques illustrated in Figure 1A.

Laboratory tests confirmed active SLE, with pancytopenia, elevated erythrocyte sedimentation rate (70 mm/h), negative proteinuria, normal C3 and C4, negative antiphospholipid antibodies (anti-β2GPI and anti-cardiolipins), high-titer antinuclear antibodies (1:1000, speckled and homogeneous patterns), positive anti-SSA/Ro and anti-SSB/La antibodies, and elevated anti-double-stranded DNA antibodies (134 IU/mL; normal <10 IU/mL). C-reactive protein was within normal limits. The SLEDAI-2K score was 22, indicating severe disease activity attributable to mucosal, cutaneous, articular, headaches, hematological, and immunological involvement.

Sequential skin biopsies were performed to clarify the complex cutaneous presentation. The initial biopsy, taken from an erythematous scaling plaque on the extensor surface, revealed orthokeratotic and parakeratotic hyperkeratosis with neutrophilic infiltration within the stratum corneum (Munro microabscesses), consistent with psoriasis (Figure 2(A)). A subsequent biopsy of a morphologically distinct lesion on the upper back and chest demonstrated epidermal thinning, basal vacuolar degeneration of the basal layer, follicular plugging, and a sparse perivascular lymphocytic interface dermatitis. histopathological features consistent with SLE (Figure 2(B)). These findings confirmed the coexistence of psoriatic and lupus-specific cutaneous pathology in the same patient.

Nail changes, including onycholysis, subungual hyperkeratosis, and discoloration, were attributed to onychomycosis on direct mycological examination (KOH preparation positive). It must be acknowledged, however, that in the context of a confirmed psoriasis/SLE overlap syndrome,this attribution carries diagnostic uncertainty. Nail dermoscopy, which might have disclosed salmon patches, oil-drop sign, or irregular pitting characteristic of psoriatic onychodystrophy, was not performed at this stage, given the clinical priority of managing the severe systemic flare. Similarly, a nail bed biopsy was not obtained. We recognize this as a limitation; concurrent nail psoriasis cannot be definitively excluded, and future cases should include formal nail unit assessment before attributing nail changes to an infectious etiology.



2A 2B

Figure 2. Histopathological findings from sequential skin biopsies (haematoxylin-eosin stain). **(A)** Biopsy from an erythematous scaly plaque on the extensor surface (×20): regular epidermal acanthosis with elongated club-shaped rete ridges, thinning of the suprapapillary plate, parakeratosis, and neutrophilic infiltration within the stratum corneum forming Munro microabscesses, consistent with psoriasis. **(B)** Biopsy from a morphologically distinct lesion on the upper back (×40): epidermal atrophy, vacuolar degeneration of the basal layer, follicular plugging with corneal ostium dilatation, thickened basement membrane zone, and sparse perivascular lymphocytic interface dermatitis, consistent with lupus erythematosus-specific cutaneous pathology.

Methotrexate was increased to 25 mg weekly without clinical improvement. Given the severity of systemic SLE activity and the fact that histopathological confirmation of a psoriatic component was still awaited, hydroxychloroquine was subsequently escalated to 600 mg daily, a step consistent with EULAR 2023 recommendations for refractory cutaneous and systemic SLE activity (16). However, this escalation paradoxically worsened the cutaneous eruption, prompting dose reversion and retrospectively confirming the psoriatic nature of the skin involvement. This sequence illustrates the diagnostic and therapeutic pitfall of escalating HCQ before histopathological characterization of the skin eruption is complete.

Direct immunofluorescence (DIF) of the SCLE lesion revealed granular deposits of IgG and C3 at the dermoepidermal junction (positive lupus band test (11)), supporting the lupus-specific nature of that lesion. Dermoscopic examination of psoriasiform lesions revealed regularly distributed dotted vessels with white scales, while lesions suspected of cutaneous lupus showed telangiectatic vessels and follicular plugs.

The clinical course and histopathological evidence underscored the diagnostic challenge posed by overlapping manifestations of psoriasis and SLE. Given the partial response to methotrexate and the paradoxical worsening with HCQ escalation, there was a need to explore biologic options targeting shared pathways.

3. DISCUSSION

Epidemiology and Clinical Significance

The coexistence of psoriasis and SLE remains uncommon but is increasingly recognized. Earlier reports by Millns and Muller estimated the coexistence rate at approximately 0.69% (12). More recent population-based cohort studies, including a Swedish registry study, report a prevalence of psoriasis among SLE patients of 3.4% to 4.9%, and, conversely, psoriasis patients have a higher-than-expected incidence of SLE (13). A meta-analysis and case series by Ali et al. further confirm this bidirectional association and illustrate the therapeutic complexity (7). The rarity and diagnostic complexity of this overlap syndrome underscore the need for heightened clinical vigilance.

Shared Immunopathogenic Mechanisms

Emerging evidence outlines a complex immunoinflammatory continuum linking psoriasis and SLE, supported by shared genetic susceptibility and partially overlapping immune pathways. Genome-wide association studies (GWAS) have identified several shared susceptibility loci, including PTPN22, STAT4, TNIP1, NFKBIA, and IL28RA, which are involved in immune activation, cytokine signaling, and regulation of immune tolerance (2,4). The PTPN22 gene encodes lymphoid tyrosine phosphatase (Lyp), a key regulator of T-cell receptor signaling; its variants are strongly associated with multiple autoimmune diseases, including both SLE and psoriasis (14).

Psoriasis pathogenesis involves activation of the IL-23/IL-17/TNF-α inflammatory axis, promoting chronic epidermal inflammation, keratinocyte hyperproliferation, and vascular remodeling (4). SLE is characterized by dysregulation of innate and adaptive immunity, with type I interferons (especially IFN-α) driving immune activation and autoantibody production. The Th17/IL-17 pathway also contributes to SLE pathogenesis, influencing disease severity and organ involvement, particularly renal and cutaneous manifestations (5). This shared cytokine milieu may account for the clinical and histopathological overlap observed in patients with both diseases.

Of particular interest in the present case is the prior exposure to rituximab. B-cell depletion by rituximab is known to perturb the Th1/Th17 cytokine balance, with a relative reduction in regulatory B cells and a consequent augmentation of Th17-mediated inflammation. This mechanism has been proposed in isolated case reports to precipitate psoriasiform eruptions in patients receiving rituximab for autoimmune conditions. While causality cannot be established retrospectively in our patient, this plausible immunopathological link warrants prospective investigation in future SLE-psoriasis overlap cohorts.

Angiogenesis and innate immune dysregulation represent additional shared mechanisms. Both diseases exhibit aberrant angiogenic signaling driven by vascular endothelial growth factor (VEGF), angiopoietins, and fibroblast growth factors (FGF), contributing to persistent inflammation and tissue remodeling (13).

Autoantibody profiles further support immunological convergence: antinuclear antibodies (ANA) and anti-Ro/SSA antibodies, classically associated with SLE, have occasionally been detected in patients with psoriasis and may correlate with photosensitivity and cutaneous inflammation (2).

Diagnostic Challenges

Psoriasis classically presents as sharply demarcated erythematous plaques with silvery scale, whereas cutaneous lupus presents with polymorphic, often annular, and photosensitive lesions that demonstrate interface dermatitis on histopathological examination (2,4). The coexistence or sequential development of these lesions can complicate diagnosis, often necessitating multiple skin biopsies to distinguish psoriasiform lupus from true psoriasis. Histopathological overlap, including parakeratosis, neutrophilic microabscesses, and epidermal atrophy, requires careful clinicopathological correlation.

Dermoscopy provides additional non-invasive clues: psoriasis typically shows regularly distributed dotted vessels and diffuse white scales, whereas cutaneous lupus more often demonstrates telangiectatic vessels, follicular plugs, and white structureless areas (14). Direct immunofluorescence aids in definitive differentiation, as lupus characteristically shows granular deposition of immunoglobulin

(IgG, IgM) and complement (C3) at the dermoepidermal junction, a feature absent in psoriasis. In the present case, both dermoscopy and sequential DIF were instrumental in establishing the dual diagnosis, illustrating the methodological contribution of this multimodal approach.

Therapeutic Considerations

Management of patients with overlapping psoriasis and SLE is challenging due to therapeutic paradoxes. Hydroxychloroquine, a cornerstone of SLE treatment, can exacerbate or induce psoriasiform eruptions, as supported by a systematic review by Sachdeva et al. reporting 18 affected patients and by case reports of HCQ-induced psoriasiform erythroderma in SLE patients (8,9). In the present case, HCQ dose escalation was initially undertaken in the context of severe systemic SLE activity and before histopathological confirmation of the psoriatic component was available, but that paradoxically worsened the cutaneous eruption. This sequence provides a real-world illustration of the imperative to histopathologically characterize cutaneous lesions before modifying HCQ dosing in patients with phenotypic overlap. Phototherapy, commonly employed in psoriasis, is generally contraindicated in SLE due to photosensitivity and the risk of systemic flares. Conventional immunosuppressants such as methotrexate and cyclosporine remain valuable options with efficacy in both diseases when used cautiously (15,16).

Biologic therapies targeting the IL-17/IL-23 axis, including secukinumab and ustekinumab, are highly effective for psoriasis but require vigilance for lupus-like adverse events, including autoantibody induction and cutaneous lupus (17). A case series by Ali et al. reported promising results with IL-17 and IL-23 inhibitors in controlling psoriatic manifestations in patients with SLE-psoriasis overlap, without triggering lupus flares (7). Rituximab may benefit refractory overlap cases, although responses are variable. In contrast, SLE-specific biologics such as belimumab and anifrolumab currently lack indications for psoriasis, highlighting a persistent therapeutic gap (18,19).

Emerging small-molecule therapies such as TYK2 inhibitors, including deucravacitinib, offer a promising approach by targeting both Th17 and type I interferon pathways simultaneously, making them theoretically attractive for this overlap syndrome; however, clinical data in SLE-psoriasis overlap remain limited (20). Multidisciplinary management involving dermatologists and rheumatologists is essential for tailoring treatment, monitoring disease activity, and mitigating adverse effects. Personalized therapeutic strategies must balance disease control with minimization of paradoxical exacerbations.

4. CONCLUSION

The coexistence of psoriasis and SLE represents a rare overlap syndrome arising from shared genetic susceptibilities and partially convergent immune pathways. Accurate diagnosis requires a high index of clinical suspicion and a multimodal approach; however, the central challenge lies in therapeutic management. Standard treatments for each condition may paradoxically worsen the other. A unified, individualized therapeutic strategy potentially incorporating agents targeting shared pathogenic pathways is needed. Multidisciplinary collaboration between rheumatology and dermatology remains essential. Further prospective research is warranted to establish evidence-based guidelines for this under-recognized overlap syndrome.

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