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CASE REPORT

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Subacute cutaneous lupus erythematosus with a psoriasiform presentation: A diagnostic clinical conundrum

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Key Clinical Message

For practitioners experiencing worsening psoriasis, subacute cutaneous lupus erythematosus (SCLE) with a psoriasiform presentation should be ruled out. Initial treatment for a presumptive diagnosis of psoriasis using hydroxychloroquine or ultraviolet phototherapy may cause SCLE to worsen.

Abstract

Psoriasiform subacute cutaneous lupus erythematosus is an unusual presentation scarcely reported in literature. We report a case of a 54-year-old man who presented with an itchy, papulosquamous rash of the upper extremities and face for 7 months. The initial physical examination revealed the classical morphology of psoriasis. One and a half years after the diagnosis of clinical worsening, the patient noticed a new papular eruption on the right posterior upper arm. A skin biopsy was performed, confirming a diagnosis of subacute cutaneous lupus erythematosus. This case report highlights the importance of considering rare presentations of cutaneous lupus erythematosus and therapeutic challenges in management.

KEYWORDS

lupus erythematosus, psoriasis, psoriasiform lupus erythematosus, subacute cutaneous lupus erythematosus

1 | INTRODUCTION

Cutaneous lupus erythematosus may also rarely present with psoriasiform lesions; in that instance, the lesion is referred to as psoriasiform subacute cutaneous lupus erythematosus (pSCLE), mimicking psoriasis. The incidence of psoriasis in the United States is 63.8 per 100,000 person-years making it a common initial differential.[1] Whereas the incidence of subacute cutaneous lupus erythematosus (SCLE) is much rarer, occurring in 4.30 per 100,000 cases; similar to the incidence of systemic lupus erythematosus (SLE).[2] Patients with both diseases are more likely to have elevated anti-dsDNA (double-stranded DNA), anti-ENA (extractable nuclear antigens) antibodies, and positive direct immunofluorescence.[3] However, the distinct coexistence of psoriasis with lupus erythematosus (LE) has been scarcely reported.[4,5] The overlapping clinical presentation may make diagnosis a challenge. However, a thorough history and physical exam can aid greatly in differentiating between pSCLE, SCLE, psoriasis, or an overlapping presentation.

2 | CASE REPORT

A 54-year-old man presented with a new onset of well-defined, dry scaly patches, over the extensor surfaces and face over 7 months (Figure 1). The lesions began on the face and upper extremities and later spread to the lower extremities and back. There was no relevant history of photosensitivity, arthritis, renal, or hematologic abnormalities. Differential diagnoses included hypertrophic lichen planus, atopic dermatitis, psoriasis, and SCLE. A presumptive diagnosis of psoriasis was made based on a physical exam and clinical history. The PASI score was 13.8. The patient was initially treated with topical fluocinonide 0.05% cream for 2 weeks; however, his rash continued to worsen at his six-month follow-up. Over the course of 10 months, the patient tried topical 0.05% clobetasol cream for 2 weeks, topical triamcinolone 0.1% ointment as needed, oral hydroxyzine 25 mg as needed, and adalimumab 40 mg every other week for 6 months without symptomatic improvement. Oral methotrexate 12.5 mg daily (five 2.5 mg tablets) and oral folic acid 1 mg daily were added with adalimumab. Due to minimal improvement, the patient began narrowband ultraviolet B (NBUVB) phototherapy twice weekly for 2 months with adalimumab and methotrexate. Paradoxically, the symptoms worsened with a further eruption of erythematous, scaly annular patches associated with pain after starting phototherapy. Phototherapy was discontinued and a differential diagnosis of systemic lupus erythematosus (SLE) was suspected (Figure 2). Biopsy showed marked epidermal hyperplasia, focal interphase papillary dermal elastosis, mild perivascular inflammatory infiltrate, and focal interface dermatitis leading to a working diagnosis of SCLE (Figure 3). There was also a focal vacuolar degeneration of the basement membrane visible on a periodic acid-Schiff (PAS) stain.

During an extensive clinical history interview, it was revealed that he had a history of joint pain and stiffness localized to both elbows, hips, shoulders, and hands. The patient had a positive family history of SLE and autoimmune disease on his maternal side. He also reported the presence of nonspecific symptoms like fever, fatigue, weakness, headaches, and memory loss. Additionally, surgical history was significant for splenectomy and

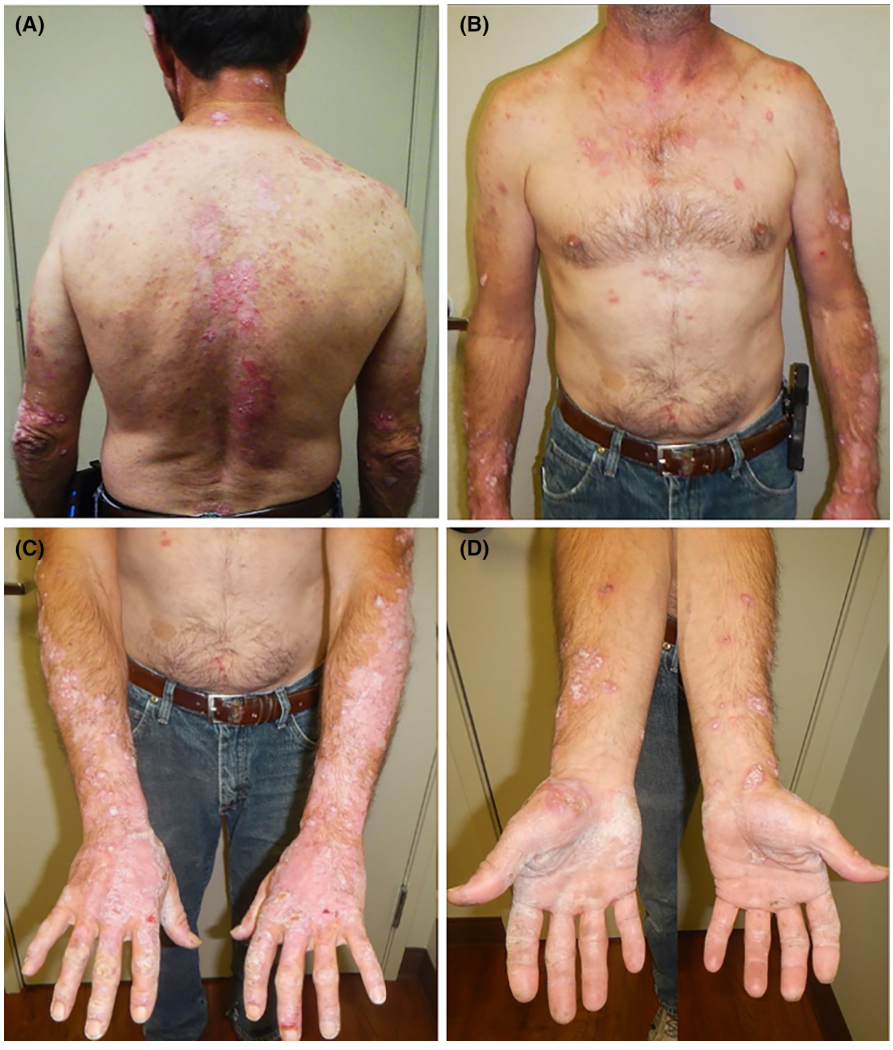

FIGURE 1 Clinical Pictures. (A) Irregular, erythematous, scaly plaques on the back. (B) Irregular, erythematous, scaly plaques on the chest. (C, D) Irregular, erythematous, plaque, and patch-like lesion on the upper extremities.

aortic valve replacement. Chronic synovitis of bilateral wrists, metacarpophalangeal joints, proximal interphalangeal joints, distal interphalangeal joints, elbows, and ankle joints were also noted with tenderness elicited on palpation.

Additional laboratory studies disclosed the following: antinuclear antibody (ANA) of 1:1280 with a speckled pattern of 1:640 (normal ANA 1:40) on indirect immunofluorescence. There was positivity for ribonucleoprotein antibodies and anti-chromatin antibodies (which indicates a 90% chance of drug-induced lupus). Anti-Scl-70 antibody (also called anti-topoisomerase I antibody), double-stranded DNA, and routine chemical analysis were all within normal limits.

After consideration of the histological, clinical, and laboratory results, a diagnosis of psoriasiform SCLE was achieved. Hydroxychloroquine 200 mg was started after consultation with his rheumatologist and finally led to clinical improvement of the cutaneous lesions at his two-month follow-up.

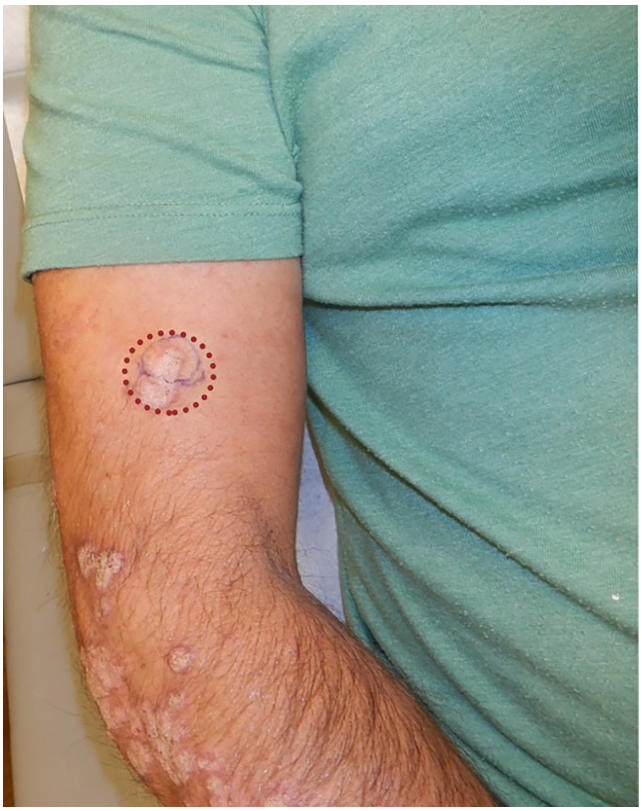

FIGURE 2 Outlined area represents new plaque formation on the right arm after one and a half years of therapy. Biopsy of the plaque was consistent with lupus erythematosus.

3 | DISCUSSION

Given the heterogeneous clinical presentation of psoriasis, other papulosquamous dermatoses such as eczema, pityriasis rosea, actinic keratosis, lichen planus, and SLE should also be considered.[6] Initially, this patient did not meet the American College of Rheumatology (formerly American Rheumatism Association) criteria for SLE, only having met one criterion: rash. However, over the course of his disease, he reported a clinical history of arthritis and photosensitivity, and subsequent laboratory workup confirmed the diagnosis of SCLE. In addition, the majority of patients with only cutaneous involvement fulfill these criteria but with limited systemic involvement.[7] A workup for recalcitrant cases of psoriasis should include biopsy and ANA serologic profiles.[8] SCLE is a rare form characterized by photosensitivity and symmetrical distribution over sun-exposed areas. There are many multifactorial associations with SCLE that include genetic, environmental, and immunologic. The associations include HLA1, B8, DR3, HLA1, B8, DR3, DQ2, DRw52, DR3, and C4 null ancestral haplotype, as well as deficiencies of C2 and C4 components of complement proteins. The increased

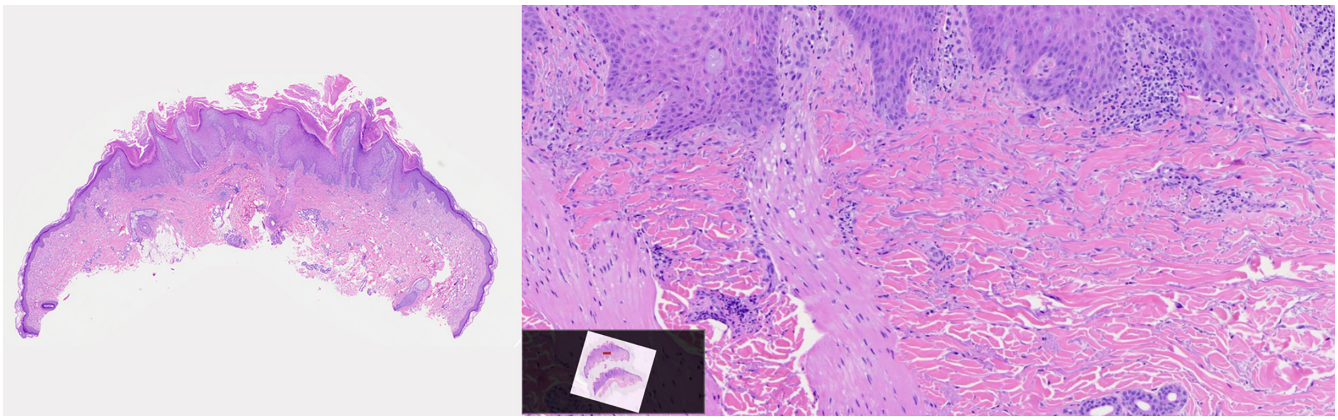

FIGURE 3 Biopsy of the right arm showing marked epidermal hyperplasia, focal interphase papillary dermal elastosis, mild perivascular inflammatory infiltrate and focal interface dermatitis, hematoxylin–eosin ×40.

presence of anti-Ro/SSA antibodies, type I interferon-related proteins, and chemokines have also been associated with SCLE. Environmental associations largely consist of ultraviolet light exposure and certain drugs, such as antihypertensives, hydrochlorothiazide, and terbinafine.

Treatment for SCLE includes various lifestyle and pharmacological therapies. By implementing proper sun protection techniques, such as using sunscreen appropriately and wearing photoprotective clothing when outdoors, the patient would be able to minimize the potential trigger of sun exposure. In addition, the use of tobacco has been shown to worsen the disease process and can lead to worse responses to certain pharmacological treatments in comparison to nonsmokers, which is why tobacco cessation is important for treatment. With the pharmacological approach, there are multiple routes of treatment. The first-line treatment typically consists of the use of topical corticosteroids and calcineurin inhibitors. If the condition is widespread, the use of systemic therapies may be implemented instead of topical therapies. It is also recommended to use antimalarial drugs (hydroxychloroquine, chloroquine, or quinacrine) due to their anti-inflammatory and photoprotective properties. If the patient is not responding to the first-line treatment, immunosuppressants such as methotrexate, dapsone, mycophenolate, azathioprine, and thalidomide which can be used to reduce the number of autoantibodies or inhibit the synthesis of tumor necrosis factor-α. Other treatments include belimumab, intravenous immunoglobulin (IVIG), and rituximab. Belimumab is an IgG1 monoclonal antibody that has been shown to reduce SLE-related flares. Rituximab is a chimeric anti-CD20 monoclonal antibody and is a first choice for patients with severe autoimmune diseases that have been resistant to conventional treatments. There have also been case studies where IVIG showed promising effects on SCLE with limited side effects.

Other differentials include the rare presentation of psoriasis in unison with SCLE, which can cause a challenging treatment paradox. Phototherapy is beneficial for psoriasis but may exacerbate SCLE, as in our case. In cases of psoriasis and SCLE overlap, severe disease flares can occur after exposure to ultraviolet phototherapy.[8] In addition, the standard pharmacologic management of SLE with systemic steroids or hydroxychloroquine can cause pustular flares in psoriasis.[9] However, tumor necrosis factor-alpha (TNF-α) inhibitors have been reported to successfully treat overlap, despite the induction of antinuclear antibodies.[10,11] Varada et al. reported that in patients receiving TNF-α inhibitors, only 5% of SLE patients flared, whereas there were none in the psoriasis or psoriatic arthritis groups.[4] However, TNF-α inhibitors may also aggravate preexisting SLE. Thus, it is essential to recognize the nuances between psoriasiform SCLE versus psoriasis and SCLE overlap. Treatment of psoriasis may cause patients with concomitant SCLE to flare, whereas SCLE medications are not efficacious for psoriasis and may exacerbate the disease.

4 | CONCLUSION

We report a rare but relevant case of psoriasiform SCLE and discuss the importance of distinguishing this diagnosis from SCLE with psoriasis overlap. Initially, the patient was clinically diagnosed with psoriasis without clinical improvement over the course of a year. Worsening of lesions, especially with phototherapy exposure, should prompt a reevaluation of differentials. After reaching the correct diagnosis, the response to therapy was rapid. Although rare, practitioners should be cognizant of psoriasiform SCLE versus psoriasis and SCLE overlap and be mindful of adverse effects when treating coexistent psoriasis and SCLE as therapy used for treating SCLE may cause psoriasis to worsen, whereas phototherapy used for treating psoriasis may cause SCLE to flare.

AUTHOR CONTRIBUTIONS

Shazli Razi: Conceptualization; investigation; methodology; writing – original draft; writing – review and editing. **Thu M.Truong:** Methodology; writing – original draft; writing – review and editing. **Palveen Sekhon:** Data curation; investigation; writing – review and editing. **Samantha Ouellette:** Writing – original draft; writing – review and editing. **Babar K Rao:** Project administration; supervision; validation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

Authors have no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

Data supporting the research is indicated in the references.

ETHICS STATEMENT

Does not apply.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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