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Photosensitive psoriasis mimicking cutaneous lupus erythematosus: a case report

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Abstract

Photosensitive presentations of psoriasis are often under-recognized and may closely mimic other photodistributed dermatoses. We report the case of a 39-year-old male patient with no history of other photodermatoses who developed a progressive, non-pruritic rash, localized in sun-exposed skin areas, with marked exacerbation after phototherapy. Pronounced photosensitivity and atypical lesion distribution, involving the malar and nasal regions, complicated the initial differential diagnosis between photosensitive plaque psoriasis and cutaneous lupus erythematosus (CLE). Only further clinical evaluation, alongside histopathological analysis and immunological testing, helped to confirm the final diagnosis of photosensitive plaque psoriasis. Treatment with systemic and topical corticosteroids, vitamin D analogues, and calcineurin inhibitors resulted in gradual clinical improvement. This case report highlights photosensitive psoriasis as an important cause of photodistributed eruptions and a rare but significant mimic of CLE.

Introduction

Psoriasis is a chronic immune-mediated inflammatory disease with diverse cutaneous and systemic manifestations involving the skin, nails, joints, and other organ systems. Following atopic dermatitis, psoriasis ranks as the second most prevalent inflammatory skin disorder, affecting approximately 2-4% of the global population.[1] Although psoriasis is inherently inflammatory, it is not traditionally regarded as a photosensitive disease. Phototherapy is a well-established and generally well-tolerated treatment method, known to reduce affected skin area and improve skin appearance, with narrowband UVB (311 nm) serving as the first-line option for stable plaque psoriasis. Second-line treatments include PUVA, often used for palmoplantar or refractory cases, and pulsed dye laser therapy, which is particularly suitable for nail psoriasis.[2] Nevertheless, a subset of patients with psoriasis may experience paradoxical worsening of psoriatic lesions following ultraviolet exposure.

In contrast to psoriasis, cutaneous lupus erythematosus (CLE) is an autoimmune inflammatory skin disease characterized by pronounced photosensitivity and the classic malar rash. Because certain CLE subtypes share overlapping morphologic features with psoriatic plaques, UV-induced exacerbation of psoriatic lesions may create diagnostic confusion.

We herein present an atypical case of psoriasis manifesting with marked photosensitivity, clinically mimicking CLE, highlighting the diagnostic difficulties in distinguishing between these two entities when evaluating photosensitive eruptions.

Case Report

A 39-year-old male patient was hospitalized due to a widespread, progressively worsening, non-pruritic, erythematous rash. Initial inpatient dermatological examination revealed multiple infiltrated erythematous plaques, several centimeters in diameter, on the scalp, face, trunk, and extremities, with areas of confluence and light scaling (Figure 1 A,B). Diffuse erythema was apparent on the face and neck. The lesions were most prominent in the malar and nasal regions of the face, clinically resembling CLE (Figure 1C). Digital dermoscopy of the lesions revealed erythema, scaling and regularly distributed dotted vessels.

According to the patient, the lesions initially appeared three months prior, following a sunburn sustained during outdoor work in summer. They were first noted on the scalp and subsequently spread to the face, neck, shoulders, and upper extremities. The patient denied any comorbidities apart from gastroesophageal reflux disease, as well as any known drug allergies or a family history of psoriasis. Before hospitalization, the patient underwent multiple treatment attempts to manage the disease. At disease onset, the patient consulted an allergologist and was initiated on oral methylprednisolone (16 mg daily), but no clinical improvement was observed. A month later, dermatological assessment revealed erythematous-squamous plaques on the scalp, chest, and back, with dermoscopy showing capillary loop formations. Due to suspected psoriasis, emollients, mometasone furoate 1 mg/g solution for the scalp, and a calcipotriol/betamethasone 50 µg/0.5 mg/g ointment for body lesions were prescribed. Additionally, narrowband UVB (311 nm) phototherapy was initiated. However, after three phototherapy sessions, the patient's condition notably worsened: pronounced erythema of the face and trunk, as well as new lesions on the lower extremities, emerged. Two months after disease onset, the patient was referred to a day hospital for further evaluation. Histopathological examination revealed epidermal parakeratosis, irregular acanthosis with minimal focal spongiosis, slight focal vacuolization of the epidermal basal layer, and superficial perivascular dermatitis. In the papillary dermis, moderately abundant perivascular lymphocytic infiltration with occasional eosinophils was observed. The patient was started on intravenous dexamethasone (8 mg), which only led to partial improvement, with reduced erythema. Outpatient treatment included oral prednisolone, starting with 30 mg per day, mometasone furoate 1 mg/g cream for body lesions, and pimecrolimus 10 mg/g cream for facial lesions. The desired clinical outcome was not achieved, prompting hospitalization one month later.

During hospitalization, routine laboratory testing showed no clinically significant deviations. Strong suspicion of CLE remained, but all antinuclear antibodies (ANA) and extractable nuclear antigen antibodies (ENA) (including nRNP/Sm, Sm, dsDNA, SS-A, Ro-52, and others) were negative. Repeat histopathology revealed epidermal parakeratosis and irregular acanthosis, lacking the distinct

interface dermatitis suggestive of CLE. Direct immunofluorescence (DIF) was performed and demonstrated only a weak, focal granular deposition of IgG and C3 at the basement membrane zone. Although classic psoriatic signs such as Munro microabscesses were not identified, the persistence of parakeratosis and acanthosis without specific lupus features supported the exclusion of CLE. Consequently, a final diagnosis of psoriasis vulgaris was established. The patient was treated with intravenous dexamethasone (8 mg/day for 5 days), followed by oral prednisolone therapy (starting dose of 25 mg/day). Topical therapy included clobetasol propionate 500 µg/g ointment, fusidic acid/betamethasone 20 mg/1 mg/g cream, and emollients. Within one week of inpatient treatment, marked improvement was observed, including a noticeable reduction in pruritus and erythema. The patient was discharged and recommended to continue oral prednisolone and topical mometasone furoate 1 mg/g ointment to affected areas.

At the five-month follow-up visit, during the winter season, significant clinical improvement was observed, with fewer lesions and reduced erythema (Figure 2 A,B). Facial lesions had diminished, appearing lighter in color, with ill-defined borders (Figure 2C). For further disease management, the patient was prescribed topical tacrolimus monohydrate 0.03% ointment for the face and mometasone furoate 1 mg/g solution for the scalp.

Discussion

We presented a diagnostically challenging case of photosensitive psoriasis mimicking CLE, which responded well to systemic corticosteroid therapy. The initial presentation was atypical of psoriasis, as both the clinical distribution of lesions and the reported photosensitivity more closely resembled CLE.

CLE is a chronic inflammatory skin disorder with a broad spectrum of clinical manifestations, commonly grouped into acute, subacute, and chronic forms, although universally accepted diagnostic criteria for these subtypes have not been established. Erythema accompanied by a malar rash, similar to the eruption observed in the presented patient, is a characteristic feature of acute CLE and reflects the condition's inherent photosensitivity. Subacute CLE may likewise manifest as erythematous, psoriasiform plaques with fine scaling, closely resembling those noted in this case.[3] Histologically, acute CLE may present with mild basal layer degeneration, superficial dermal oedema, and interface, perivascular, and periadnexal lymphocytic infiltrate. In contrast, chronic CLE, specifically discoid lupus erythematosus, may display hyperkeratosis, keratin-filled dilated follicles, vacuolar basal keratinocyte degeneration, and a dense, often deep, inflammatory dermal infiltrate.[4] The classic lupus band test seen in DIF is characterized by a continuous granular band of localized immune deposits (IgG, IgM, IgA, C3) located along the basement membrane zone, which supports the diagnosis of

CLE.[5] In this patient, however, DIF demonstrated only weak and focal deposition of IgG and C3. Furthermore, the absence of distinct interface dermatitis, together with the presence of parakeratosis and irregular acanthosis in histopathology, negative serologic tests for ANA and ENA antibodies, and classic dermoscopic features of psoriasis, collectively supported the diagnosis of photosensitive plaque psoriasis rather than CLE.[6]

Photosensitive psoriasis is a rare condition, typically affecting young, fair-skinned women with a family history of psoriasis.[7] The reported case differs from this classical presentation, given the patient's male sex and the absence of a family history of psoriasis. The reported prevalence of photosensitivity is estimated to range from 5.5% to 24% among patients with psoriasis. Photoaggravation of psoriatic lesions is also not uncommon in individuals with coexisting photodermatoses, such as previously discussed CLE, porphyria, or polymorphic light eruption, all of which were ruled out in the reported patient.[8]

Phototherapy is widely used in the treatment of psoriasis due to its proapoptotic and immunosuppressive properties. The beneficial antipsoriatic effects of UV radiation include apoptosis of keratinocytes and lymphocytes, induction of regulatory T cells and B cells, Langerhans cell alterations, production of anti-inflammatory cytokines (interleukin [IL]-4, IL-10), as well as the downregulation of the T helper (Th)-17/IL-23 axis. However, UV radiation may also induce psoriatic lesions through damage to self-coding RNA, increased damage or pathogen-associated molecular patterns (DAMP/PAMP), activation of Toll-like receptors, microRNA dysregulation, release of inflammatory cytokines (IL-6, tumor necrosis factor [TNF]-α), and other effects. It is speculated that low doses of UV may suppress, whereas higher doses may trigger, psoriatic lesion formation. It should also be noted that the clinical antipsoriatic effect of phototherapy is confined to the sites of direct UV exposure. The appearance of new lesions in sun-exposed areas may also be attributed to the Koebner phenomenon, in this case due to UV damage.[8]

Nonetheless, the exact cause of photosensitivity in psoriatic patients is unknown, and studies on photosensitive psoriasis are limited. According to a study conducted by Rutter *et al.*, photosensitive psoriasis has a strong association with HLA-Cw*0602 and can be linked to changes in T lymphocyte subsets, namely an increase in dermal $CD8^+$ cell counts after photoaggravation. In their study, broadband UVA elicited the strongest response in patients with severely photosensitive psoriasis.[9] In contrast to these observations, our reported patient experienced marked worsening of lesions specifically following exposure to narrowband UVB radiation at 311 nm, rather than UVA. According to an older study conducted by Ros *et al.*, oral photochemotherapy with trimethylpsoralen and UVA (PUVA) has a positive prophylactic effect for patients with photosensitive psoriasis and may serve as an effective alternative treatment option.[10]

Besides phototherapy, there are several different treatment options available for psoriasis. Topical therapy for the analyzed patient included corticosteroids (mometasone furoate, betamethasone dipropionate, and clobetasol propionate) to reduce inflammation and proliferation of psoriatic lesions. Topical calcineurin inhibitors (tacrolimus monohydrate) helped decrease disease activity by blocking the transcription of interleukins IL-2, IL-4, and IL-10. Topical vitamin D analogues (calcipotriol) aided keratinocyte growth inhibition and cell differentiation. Keratolytic agents and emollients were used alongside these therapies to support skin barrier restoration and scale reduction.[11]

In this case, the patient's skin condition responded well to systemic corticosteroid therapy – during hospitalization, he received intravenous dexamethasone followed by oral prednisolone. Although systemic corticosteroids are generally not recommended for the treatment of psoriasis due to the well-recognized risk of rebound flares, their use in this patient was warranted by the atypical presentation and acute severity. Given the initial clinical ambiguity and strong suspicion of CLE, a deviation from standard protocols was necessary to ensure prompt anti-inflammatory control.[12] The patient was administered oral prednisolone at the onset of the disease, but intravenous corticosteroids proved to be more effective. Five months following hospitalization, the patient's skin condition had notably improved; however, it may be argued that reduced UV exposure during the winter months may also be a significant contributing factor to lesion regression.

Conclusions

This case highlights the importance of recognizing photosensitive psoriasis as a potential mimic of CLE. Accurate differentiation relies on careful clinical, dermoscopic, and histopathological evaluation, particularly when phototherapy worsens symptoms. Early identification allows for appropriate management and helps prevent misdiagnosis alongside unnecessary interventions.

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Figure 1. A) Anterior view with multiple erythematous lesions. **B**) Posterior view, highlighting rash localization on the upper back. **C**) Facial view with lesions concentrated in the malar and nasal regions.

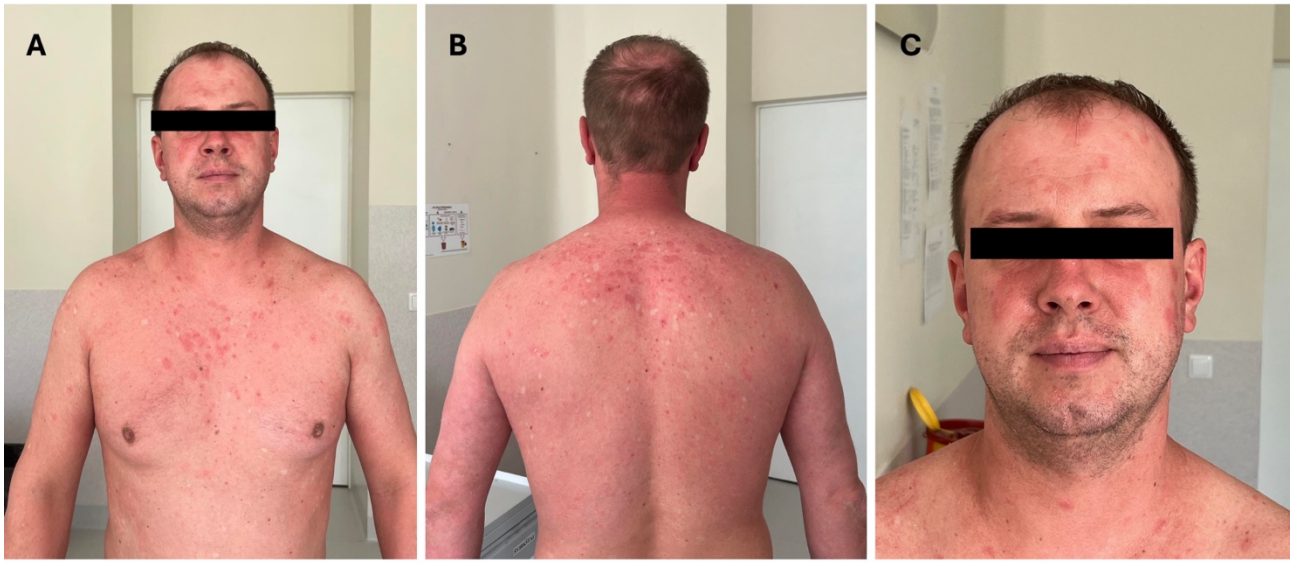

Figure 2. A) Anterior view, showing fewer erythematous plaques. **B**) Posterior view, highlighting reduced lesions and milder erythema on the upper back. **C**) Facial view with ill-defined lesion borders and marked regression of erythema.