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CASE REPORT

A Case Report of a Rare Subacute Cutaneous Lupus Erythematosus with Chin Prostration

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Abstract: Subacute cutaneous lupus erythematosus (SCLE) is a clinical subtype of cutaneous lupus erythematosus (CLE), an autoimmune connective tissue disease characterized by photosensitive rashes. This report presents a rare case of SCLE involving the chin. The patient presented with an annular, erythematous, scaling plaque on the left chin, which had been repeatedly misdiagnosed. Through integrated analysis of dermoscopic features, reflectance confocal microscopy (RCM) findings, and skin biopsy, a definitive diagnosis was established. Combination therapy with systemic corticosteroids and immunomodulators was initiated. Significant lesion regression was observed at the 19-week follow-up. The combined application of dermoscopy and RCM optimizes clinical management by reducing diagnostic errors and enabling real-time treatment monitoring.

Keywords: subacute cutaneous lupus erythematosus, dermoscopy, reflective confocal microscopy, pathological biopsy

Introduction

Subacute cutaneous lupus erythematosus (SCLE) is a clinical subtype of cutaneous lupus erythematosus (CLE). It is an autoimmune connective tissue disease that is characterized by photosensitive rashes. Ultraviolet radiation, viral infections, tobacco use, environmental pollution, and trauma can trigger SCLE by inducing keratinocytes to express associated antigens. This process activates multiple signaling pathways, promotes autoantigen transfer, and initiates other inflammatory responses.[1,2] There are currently no universally established diagnostic criteria for SCLE. Its diagnosis is primarily based on a combination of clinical presentation, histopathological examination, and serological findings. SCLE typically presents as polycyclic annular and/or psoriasiform papulosquamous lesions. However, due to its morphological similarity to conditions such as granuloma annulare, erythema annulare centrifugum, psoriasis, and tinea corporis, it is frequently misdiagnosed.[3] In addition, SCLE is closely related to systemic lupus erythematosus (SLE). As the disease progresses, SCLE can involve multiple systems and organs, gradually transforming into SLE. Therefore, SCLE is regarded as one of the diagnostic criteria for SLE.[4]

Skin microscopy and RCM are simple, rapid, and non-invasive auxiliary diagnostic tools that have become an important part of the skin disease diagnostic process. They are quite helpful for the early diagnosis and differential diagnosis of skin diseases, in addition to the evaluation of changes in condition and severity.[5,6] They play a crucial role in the guidance of CLE diagnosis and treatment.

This article reports a case of SCLE in the chin area that was repeatedly misdiagnosed over an extended period. Through a systematic analysis of its clinical presentation, dermoscopic and RCM features, histopathological findings, and treatment outcomes, we aim to improve the recognition of atypical SCLE manifestations, enhance diagnostic accuracy, and refine the clinical diagnostic approach for this condition.

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Case Presentation

A 61-year-old female presented to our hospital with a 10-month history of a persistent, pruritic, erythematous patch on the left side of her chin. The lesion, approximately 4–5 cm in diameter, was previously misdiagnosed as eczema or a fixed drug eruption at another hospital, where she received phototherapy. The patient had no underlying diseases, denied a family history of rheumatic diseases, and reported no specific food or drug allergies. Dermatological examination revealed a well-defined, slightly raised, circular erythematous plaque on the left chin. The border was edematous and elevated, with fine scaling along the inner margin (Figure 1a).

The diagnosis of SCLE was based on dermatoscopy, RCM, and histopathological examination. Laboratory investigations revealed leukopenia, as evidenced by a reduced white blood cell count of 3.28 × 109/L (reference range: 3.5–9.5 × 109/L), accompanied by absolute neutropenia (1.08 × 109/L; reference range: 1.8–6.3 × 109/L), constituting 33.2% of the total leukocyte differential. Findings consistent with microcytic anemia included decreased mean corpuscular volume (75.2 fL; reference range: 82–100 fL) and mean corpuscular hemoglobin (23.7 pg; reference range: 27–34 pg). Urinalysis indicated trace protein. Serum IgG was elevated at 17.66 g/L (reference range: 8–17 g/L). Antinuclear antibody (ANA) testing was positive, demonstrating a homogeneous nuclear pattern at a titer of 1:100 alongside a concurrent cytoplasmic speckled pattern at the same titer. Comprehensive autoantibody screening returned negative results for anti-dsDNA, anti-nRNP/Sm, anti-Sm, anti-SSA/Ro, anti-SSB/La, anti-Scl-70, anti-centromere, anti-nucleosome, anti-ribosomal P, and anti-Jo-1 antibodies. Liver function tests and electrolyte levels remained within normal limits, including alkaline phosphatase. The patient declined to undergo echocardiography and chest X-ray. Dermatoscopy revealed a red background with focal yellow and white scales, keratin plugs in hair

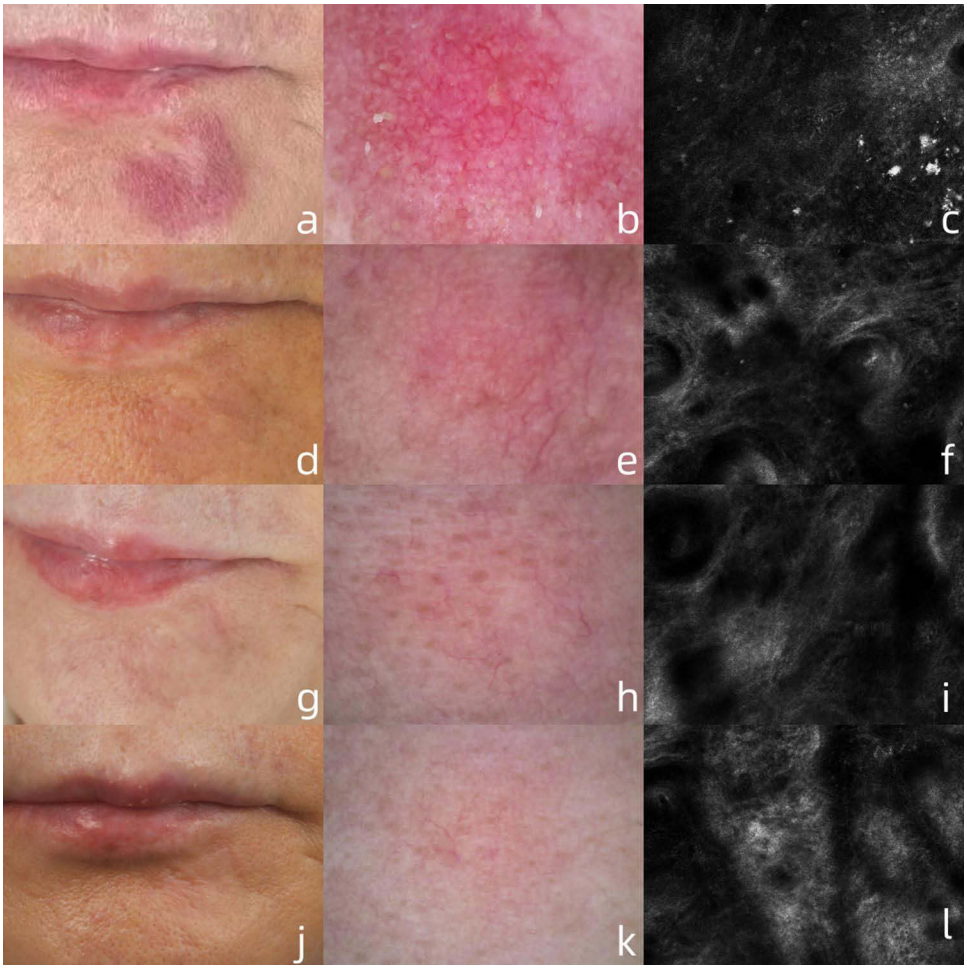

Figure 1 Clinical photography, dermatoscopy and reflectance confocal microscopy of the patient. Baseline presentation (**a–c**). Post-treatment appearances at 11 weeks (**d, g** and **j**); 15 weeks (**e, h** and **k**); and 19 weeks (**f, i** and **l**).

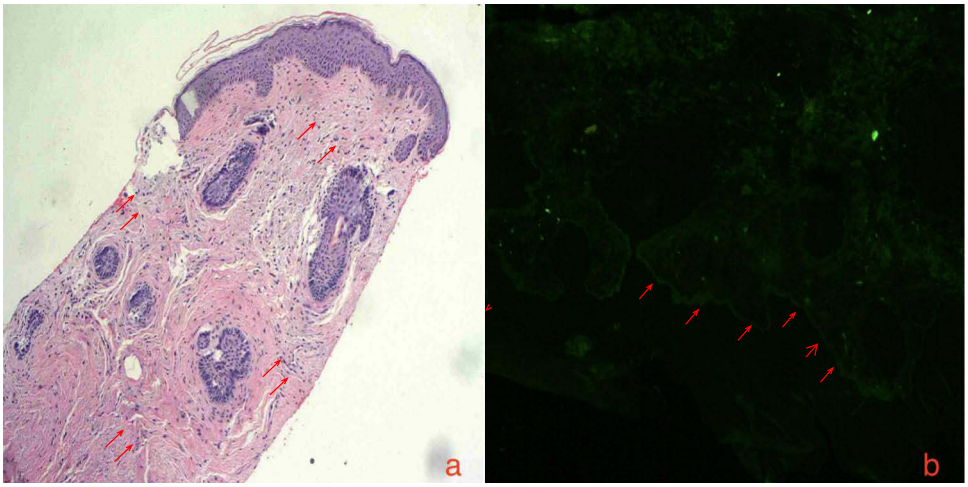

Figure 2 Skin biopsy. Keratin plug formation with dermal perivascular lymphohistiocytic infiltration (**a**). Direct immunofluorescence demonstrated linear Immunoglobulin M deposition along the basement membrane zone (**b**).

follicles, follicular white halos, follicular periphery red dot sign, and irregularly congested blood vessels around the follicles. Blue-gray pigment granules were deposited (Figure 1b). RCM revealed mild epidermal atrophy and thinning, with numerous scattered follicular keratin plugs. The basal layer exhibited significant liquefactive degeneration. Melanin-containing cells and inflammatory cells infiltrated the dermal papilla and superficial dermis. Blood vessels were dilated and congested, with a moderate perivascular and perifollicular inflammatory cell infiltrate (Figure 1c). Skin biopsy showed keratin plug formation with dermal perivascular lymphohistiocytic infiltration. Direct immunofluorescence demonstrated linear IgM deposition along the basement membrane zone, with negative staining for C3, IgG, and IgA (Figure 2a and b).

The therapeutic regimen comprised oral methylprednisolone at 24 mg once daily (0.48 mg/kg/day, based on a 50 kg body weight), Tripterygium glycosides tablets at 20 mg three times daily, and topical halometasone cream. From week 2, the dose of methylprednisolone was tapered to 16 mg once daily (0.32 mg/kg/day). At week 3, Tripterygium glycosides were discontinued, and methylprednisolone was further reduced to 8 mg once daily (0.16 mg/kg/day) for 7 consecutive days, while topical therapy was switched to 0.03% tacrolimus ointment. Beginning week 4, methylprednisolone was maintained at 4 mg once daily (0.08 mg/kg/day) with continued application of 0.03% tacrolimus ointment. At the 19-week follow-up, the patient exhibited significant regression of cutaneous lesions (Figure 1d, g and j). Oral methylprednisolone was subsequently tapered and ultimately discontinued at week 24. Dermoscopy demonstrated marked improvement in vascular congestion and follicular keratin plugs, with reduced pigmentation (Figure 1e, h and k). RCM revealed restoration of epidermal architecture and decreased melanocyte/inflammatory cell density (Figure 1f, i and l). No treatment-related adverse events were documented during the therapeutic course, with ongoing clinical surveillance maintained.

Discussion

CLE is subclassified into four clinical variants based on disease chronicity and lesion morphology: acute, subacute, chronic, and intermittent. Accurate clinical subtyping is crucial, as it informs both prognosis and guides treatment strategies. SCLE accounts for approximately 8% of all CLE cases in clinical practice.[7] SCLE presents in both typical and rare forms. Rare variants include erythrodermic lupus erythematosus, lupus erythematosus tumidus, and annular erythema of lupus. Typical SCLE lesions are usually distributed on the upper trunk and extremities but typically spare the face and scalp.[8,9] Currently, there are no standardized diagnostic criteria for SCLE.[10] SCLE must be differentiated from erythema gyratum repens, psoriasis, granuloma annulare, erythema annulare centrifugum, and tinea corporis.[4]

A multicenter clinical study (n=553) revealed a median diagnostic delay of 59 weeks from symptom onset to histopathologically confirmed CLE, with 19 cases misdiagnosed prior to biopsy.[11] This prolonged diagnostic interval and frequent misdiagnosis highlight persistent challenges in clinical practice. Moreover, histopathological examination

alone is often insufficient for precise CLE subclassification.[9] Therefore, integrating serological profiles with dermatologic imaging and histopathological findings is essential to improve diagnostic specificity. Notably, SCLE can present initially with atypical symptoms (eg, erythematous or bluish-red edematous rashes) or in atypical locations, which is a major contributor to misdiagnosis.[12]

Dermatologic imaging techniques, such as dermoscopy and RCM, are valuable in dermatological diagnosis due to their convenience and noninvasive nature. A key advantage is their capacity for longitudinal monitoring of disease progression and therapeutic response, which is not feasible with conventional histopathological examination alone. These imaging modalities significantly enhance the differential diagnosis of SCLE from conditions such as eczema, contact dermatitis, cutaneous lymphoma, basal cell carcinoma, and postinflammatory hyperpigmentation.[13] Dermatologic imaging techniques are also useful in the subclassification of CLE. The reported sensitivity and specificity of RCM for diagnosing chronic CLE are 62.96% and 94.53%, respectively.[14] Compared to ACLE and CCLE, the dermoscopic features of SCLE are characterized by white scales, polymorphic vessels (including linear, linear-irregular, and branched forms, as well as sparsely distributed punctate vessels), and a pinkish background. Notably, punctate vessels and peripheral pigmentation are observed more frequently in SCLE than in other CLE subtypes.[15] Reflectance confocal microscopy (RCM) of SCLE lesions reveals dense, dendritic inflammatory cell infiltrates in the stratum spinosum/granulosum and at the dermoepidermal junction, along with variable disruption of the epidermal honeycomb pattern. Notably, the density of these inflammatory infiltrates is significantly higher than that in chronic lesions of discoid lupus erythematosus.[16]

In the treatment of CLE, systemic therapy involving antimalarials and/or corticosteroids is recommended.[10] Given the detection of proteinuria in our patient, regular urine protein testing has been advised to monitor for the potential development of lupus nephritis (LN) during the course of SCLE. It is important to emphasize that LN necessitates a more aggressive therapeutic approach compared to CLE, including intervention with biologic agents such as belimumab and rituximab.[17]

This case presented with cutaneous lesions at atypical locations, manifesting as lupus erythematosus serpiginosus which contributed to repeated diagnostic delays during the disease course. Definitive diagnosis of SCLE was achieved through integrated analysis of clinical manifestations, dermatologic imaging, serological profiles, and histopathological examination. Dermatologic imaging served as a pivotal tool for objectively monitoring therapeutic efficacy.

In summary, the absence of standardized diagnostic criteria contributes to diagnostic challenges in SCLE. For patients with atypical presentations, integrating multimodal diagnostic approaches is recommended to enhance diagnostic sensitivity.

Ethics Statement

The publications of images were included in the patient's consent for publication of the case. The Hospital Ethics Committees of the Fifth People's Hospital of Hainan Province approved to publish the case details.

Consent Statement

Informed consent was provided by the patient for publication of the case.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Shapiro M, Sosis AC, Junkins-Hopkins JM, Werth VP. Lupus erythematosus induced by medications, ultraviolet radiation, and other exogenous agents: a review, with special focus on the development of subacute cutaneous lupus erythematosus in a genetically predisposed individual. *Int J Dermatol*. 2004;43(2):87–94. doi:10.1111/j.1365-4632.2004.02013.x
2. Wang XL, Du JF, Zhao JT, Pei GD. Screening and bioinformatics analysis of differential genes in subacutecutaneous lupus erythematosus based on GEO database. *Chin J Health Lab Tec*. 2022;32(21):2640–2643.
3. Aringer M, Costenbader K, Daikh D, et al. 2019 European League Against Rheumatism/American College of Rheumatology classification criteria for systemic lupus erythematosus. *Arthritis Rheumatol*. 2019;71:1400–1412. doi:10.1002/art.40930
4. Vale ECSD, Garcia LC. Cutaneous lupus erythematosus: a review of etiopathogenic, clinical, diagnostic and therapeutic aspects. *An Bras Dermatol*. 2023;98(3):355–372. doi:10.1016/j.abd.2022.09.005
5. Wang ZY, Sheng YJ, Chen KJ, Cui Y. Progress of artificial intelligence in the application of reflectanceconfocal microscopy for the diagnosis of skin diseases. *Med J Wuhan Univ*. 2024;45(12):1419–1422+1427. doi:10.14188/j.1671-8852.2024.0255
6. Wu W, Deng DQ. *Preliminary Study of Dermoscopy in Differential Diagnosis of Lupus Erythematosus and Dermatomyositis*. Kunming Medical University; 2020. doi:10.27202/d.cnki.gkmyc.2020.000075
7. Filotico R, Mastrandrea V. Cutaneous lupus erythematosus: clinico-pathologic correlation. *G Ital Dermatol Venereol*. 2018;153:216–229. doi:10.23736/S0392-0488.18.05929-1
8. Herzum A, Gasparini G, Cozzani E, Burlando M, Parodi A. Atypical and rare forms of cutaneous lupus erythematosus: the importance of the diagnosis for the best management of patients. *Dermatology*. 2022;238(2):195–204. doi:10.1159/000515766
9. Bitar C, Menge TD, Chan MP. Cutaneous manifestations of lupus erythematosus: a practical clinicopathological review for pathologists. *Histopathology*. 2022;80(1):233–250. doi:10.1111/his.14440
10. Lu Q, Long H, Chow S, et al. Guideline for the diagnosis, treatment and long-term management of cutaneous lupus erythematosus. *J Autoimmun*. 2021;123:102707. doi:10.1016/j.jaut.2021.102707
11. Wu MY, Wang CH, Ng CY, et al. Periorbital erythema and swelling as a presenting sign of lupus erythematosus in tertiary referral centers and literature review. *Lupus*. 2018;27(11):1828–1837. doi:10.1177/0961203318792358
12. Wu RR. Analysis of the causes of misdiagnosis of subacute cutaneous lupus erythematosus. *Zhejiang Prev Med*. 1998;(07):432–433.
13. Lacarrubba F, Ardigò M, Stefani A, Di Verzì AE, Micali G. Dermatoscopy and reflectance confocal microscopy correlations in nonmelanocytic disorders. *Dermatol Clin*. 2018;36:487–501. doi:10.1016/j.det.2018.05.015
14. Koller S, Gerger A, Ahlgrimm-Siess V, et al. In vivo reflectance confocal microscopy of erythematosquamous skin diseases. *Exp Dermatol*. 2009;18:536–540. doi:10.1111/j.1600-0625.2008.00822.x
15. Żychowska M, Reich A. Dermoscopic features of acute, subacute, chronic and intermittent subtypes of cutaneous lupus erythematosus in caucasians. *J Clin Med*. 2022;11(14):4088. doi:10.3390/jcm11144088
16. Mazzilli S, Vollono L, Diluvio L, et al. The combined role of clinical, reflectance confocal microscopy and dermoscopy applied to chronic discoid cutaneous lupus and subacutus lupus erythematosus: a case series and literature review. *Lupus*. 2021;30(1):125–133. doi:10.1177/0961203320958141
17. Chan EY, Marks SD. Childhood-onset lupus nephritis: unique aspects and challenges in management. *Kidney Int*. 2025;108(5):799–810. doi:10.1016/j.kint.2025.05.039