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# Differentiating and Managing Cutaneous Lupus Erythematosus-Associated Alopecia and Patchy Alopecia Areata: Therapeutic Insights From Case Studies

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## ABSTRACT

Cutaneous lupus erythematosus (CLE) is an autoimmune disease with diverse clinical manifestations, including patchy hair loss resembling alopecia areata (AA). This report describes two cases of CLE presenting as AA mimickers, emphasizing the need to consider CLE in differential diagnosis for patchy hair loss. Early and accurate diagnosis is crucial for effective management and preventing scarring alopecia.

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## INTRODUCTION

Cutaneous lupus erythematosus (CLE), a group of autoimmune skin diseases characterized by skin eruptions including, but not limited to photosensitivity, malar rash, discoid lupus erythematosus (DLE), and subacute cutaneous lupus erythematosus (SCLE), as well as generalized non-scarring alopecia, which manifests in up to 85% of systemic lupus erythematosus (SLE) patients.[1]

SLE diagnostic criteria, as established by prominent health organizations like the Systemic Lupus International Collaborating Clinics (SLICC), European League Against Rheumatism (EULAR), and American College of Rheumatology (ACR), incorporate non-scarring alopecia, potentially complicating the differential diagnosis for patients with both SLE and hair loss.[2,3] Differentiating between solely alopecia areata (AA), AA concomitant in the setting of SLE, or other conditions initially resembling AA, such as DLE or syphilitic alopecia, may present a clinical challenge, necessitating comprehensive patient history and diagnostic tools.[4-6]

This article illuminates the need for a multidisciplinary approach in managing CLE-associated alopecia and patchy AA through the examination of two case studies. With an emphasis on diagnosis and treatment, this study accentuates the critical importance of a comprehensive understanding of these conditions, collaboration among healthcare professionals, and ongoing research to refine treatment strategies and ultimately improve patient outcomes.

### Case 1

A 36-year-old Asian male with a 22-year history of Systemic Lupus Erythematosus (SLE) presented to a dermatology clinic in 2016, reporting rapid hair loss and intense headaches (Figures 1 and 2). His SLE history included Discoid Lupus Erythematosus

**FIGURE 1 AND 2.** Multiple round alopecic patches on the bilateral parieto-temporal scalp in patient with SLE (Case 1). The bilateral manifestation of hair loss is a common feature of cutaneous lupus erythematosus.

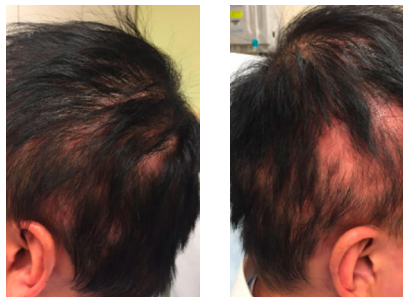


**FIGURE 3.** CT Scan Illustrating Subcutaneous Fat Stranding in patient with SLE (Case 1). This CT scan image reveals extensive subcutaneous fat stranding in the skull of a 36-year-old SLE patient, presenting with rapid hair loss and intense headaches. This finding suggests the impact of systemic inflammation associated with SLE.

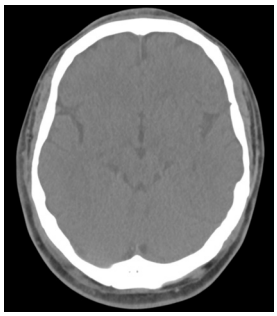
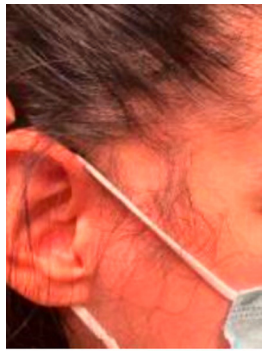


**FIGURE 4.** Distinct alopecic patch in right preauricular area within the temple in patient with no known prior autoimmune disease history (Case 2).



(DLE) confirmed by scalp biopsy in 2014, antiphospholipid antibodies, pyoderma gangrenosum-like lesions, and chronic kidney disease.

In December 2016, he experienced a significant increase in hair loss, with no scalp symptoms. His medical records indicated previous SLE-related alopecia. Despite using clobetasol solution, his condition did not improve. Given his history of intermittent thrombocytopenia (ITP) linked to cyclosporine, his platelet and white blood cell counts were monitored upon restarting cyclosporine therapy.

A CT scan showed extensive subcutaneous fat stranding above the skull (Figure 3). He had a prior diagnosis of patchy Alopecia Areata (AA) two decades earlier, responding well to intralesional steroid injections. Physical examination revealed multiple round alopecic patches with erythema, decreased surrounding hair density, and a negative pull test (Figures 1 and 2). A scalp punch biopsy confirmed lupus panniculitis.

Treatment adjustments included an increased dose of Thalidomide in July 2018, and later discontinuation of Thalidomide and Warfarin in February 2019, with no significant hair regrowth. In 2019, his treatment was intensified with an increase in cyclosporine to 200 mg twice daily and Vitamin D supplementation for low levels. The current regimen includes belimumab, cyclosporine, mycophenolate mofetil, and topical minoxidil. Despite this comprehensive treatment, he continued to experience Cutaneous Lupus Erythematosus on the scalp, with additional hair loss potentially due to androgenetic alopecia. Significant hair regrowth remains an unmet clinical goal.

**Case 2**

A 42-year-old female with no history of autoimmune diseases presented in 2022 with facial skin eruptions, scalp pruritus, and a well-defined alopecic patch on her scalp (Figure 4). Clinical evaluation revealed a round alopecic patch in the right preauricular area. Despite nine intralesional triamcinolone acetonide injections improving her alopecia, she continued to experience facial skin eruptions and scalp pruritus.

Subsequent comprehensive skin examinations showed erythema and fine scaling on her cheeks. After systemic work-up ruled out SLE, a scalp punch biopsy was performed. Histopathology showed dense superficial and deep perivascular and periadnexal lymphocytic infiltrate with vacuolar alteration. Indications of a scarring process were noted, including perifollicular fibroplasia and fibrous tracts, as revealed by an Elastin van Gieson stain showing elastic fiber thickening and clumping. Features characteristic of alopecia areata, like follicular streamers, pigment casts, and eosinophils, were also present.

Based on these findings, she was diagnosed with chronic cutaneous lupus erythematosus presenting in an alopecia areata-like pattern. Her treatment included oral hydroxychloroquine 5 mg/kg daily, topical corticosteroids, and continued intralesional triamcinolone injections. Six months later, her facial eruption and scalp pruritus had resolved, with significant hair regrowth in the alopecic patch, showing no signs of scarring.

## DISCUSSION

This case series underscores the diagnostic and therapeutic intricacies in addressing alopecia, particularly in distinguishing between Cutaneous Lupus Erythematosus (CLE) and Alopecia Areata (AA). Despite their distinct nature, these conditions exhibit overlapping clinical presentations, necessitating a comprehensive diagnostic process that includes clinical, laboratory, and histopathological evaluations (Table 1).

**TABLE 1.**

**Comparative Overview of Cutaneous Lupus Erythematosus (CLE) and Alopecia Areata (AA)**

| Feature | CLE | AA | Citations |
|---|---|---|---|
| Clinical Manifestations | Varied; includes oral/nasal ulcers, photosensitivity, skin infections, scarring | Predominantly hair loss without visible inflammation | 8,9,12 |
| Typical Presentation | Malar/maculopapular rash, annular/papulosquamous dermatitis, DLE, LE profundus | Well-defined, round/oval patches of complete or near-complete hair loss | 4,5,8 |
| Histopathological Features | Lymphocytic inflammation, interface dermatitis, follicular plugs, mucin deposition, basement membrane thickening | Peribulbar inflammatory infiltrate with activated T-lymphocytes, histiocytes, eosinophils | 5,13 |
| Diagnostic Testing | ANA, anti-dsDNA, skin biopsy | Clinical features, dermoscopy, scalp biopsy | 9-11 |
| First-line Therapy | Topical/intralesional corticosteroids, oral antimalarials | Intralesional/topical corticosteroids | 4,10,11 |
| Advanced Therapy | Oral corticosteroids, oral JAK inhibitors, belimumab | Topical DPCP, anthralin, systemic therapy, oral JAK inhibitors | 4,11,14 |

AA: Alopecia Areata; CLASI: CLE Disease Area and Severity Index; CLE: Cutaneous Lupus Erythematosus; DLE: Discoid Lupus Erythematosus; JAK: Janus Kinase; SCLE: Subacute Cutaneous Lupus Erythematosus

The challenge in differentiating AA from CLE stems from their shared symptom of patch-like hair loss and the need for detailed clinical and histopathological assessment (Table 1).[5,8] These conditions also differ significantly in their clinical manifestations, diagnostic testing, and treatment approaches, as summarized in Table 1.

For CLE, the diagnostic approach integrates clinical assessment with tests like ANA and anti-dsDNA, and the treatment focuses on managing the immune response, often involving corticosteroids and antimalarials (Table 1).[4,9] Conversely, patchy AA is diagnosed primarily based on clinical presentation and dermoscopy, with treatments including corticosteroids and oral JAK inhibitors (Table 1).[10,11]

In summary, recognizing the nuances in clinical presentations, pathophysiology, and therapeutic strategies for CLE-associated alopecia and patchy AA is crucial. This case series highlights the value of a broad differential diagnosis and a multidisciplinary approach in patient management, emphasizing collaboration among various specialties to enhance patient care and outcomes (Table 1). The ongoing research in immunology and dermatology continues to enrich our understanding of these conditions and aids in the development of more targeted therapies.

## DISCLOSURES

Dr Shapiro is a consultant for Pfizer, Eli Lilly, Eirion, Follica, and Replicel Life Sciences. Drs Shapiro and Lo Sicco have been investigators for Regen Lab and are investigators for Pfizer. Dr Lo Sicco is a consultant for Pfizer and Aquis. MGB, LA, EM, AA, and AC have no conflicts to disclose.

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