Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Dermatology & Venereology

Coexistence of Alopecia Areata, Lichen Planopilaris, and Lichen Planus Pigmentosus: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i07.014 | **Received:** 08.05.2026 | **Accepted:** 26.06.2026 | **Published:** 13.07.2026

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Abstract **Case Report**

Alopecia areata and lichen planopilaris are immune-mediated alopecias with different follicular targets and prognoses, and their coexistence is rare. We report a 37-year-old woman with alopecia beginning at five years of age and pruritic hyperpigmented patches developing during adolescence. Examination showed diffuse non-scarring alopecia of the frontal and occipital scalp, scar-like temporal patches, subtle follicular papules, and loss of body hair. Trichoscopy demonstrated black and yellow dots in non-scarring areas and cicatricial changes in the temporal regions. A frontal scalp biopsy showed marked follicular miniaturization compatible with chronic alopecia areata, whereas a temporal biopsy showed interface folliculitis, peri-isthmic lymphocytic inflammation, perifollicular fibrosis, and sebaceous gland loss, supporting lichen planopilaris. Biopsy of a pigmented patch demonstrated basal hyperpigmentation, pigment incontinence, and dermal melanophages, consistent with lichen planus pigmentosus. Monthly intravenous corticosteroid pulses combined with methotrexate 20 mg weekly stabilized progression and produced slight scalp and eyebrow regrowth at one year, while pigmentation remained largely unchanged. This case emphasizes the need to examine different scalp zones separately and to target active areas for biopsy, because scarring and non-scarring alopecias may coexist and require different therapeutic priorities.

Keywords: Alopecia areata, lichen planopilaris, lichen planus pigmentosus, cicatricial alopecia, trichoscopy, methotrexate.

INTRODUCTION

Alopecia areata (AA) is a T-cell-mediated non-scarring alopecia that primarily targets the hair bulb and may permit regrowth, whereas lichen planopilaris (LPP) is a primary lymphocytic cicatricial alopecia that targets the infundibulo-isthmic region and may irreversibly destroy follicular stem cells [1,2]. Despite their distinct clinical behavior, both disorders involve disruption of hair-follicle immune privilege. Simultaneous AA and LPP has been described only rarely [3,4]. The additional presence of lichen planus pigmentosus (LPPig) may indicate a broader lichenoid immune spectrum [5]. We report a patient in whom careful regional examination, trichoscopy, and site-directed biopsies identified all three conditions.

CASE REPORT

A 37-year-old woman was evaluated for long-standing alopecia and persistent pruritic hyperpigmented lesions. Hair loss had begun at five years of age and initially involved the frontal scalp. Previous treatments included topical corticosteroids, topical minoxidil, 30 sessions of psoralen plus ultraviolet A photochemotherapy, and traditional practices including scarification and topical plant preparations, without meaningful improvement. At 16 years of age, flat pruritic pigmented lesions appeared on the back and later extended to the limbs. Antifungal treatments were ineffective.

Examination showed diffuse non-scarring alopecia of the frontal and occipital scalp with residual islands of hair. Smooth scar-like patches were present in both temporal regions. Palpation identified small follicular papules in clinically active areas, and diffuse hair loss involved the eyebrows and the remainder of the body. Trichoscopy showed black dots and yellow dots in frontal and occipital regions, supporting AA, whereas temporal areas showed loss of follicular openings and cicatricial change. Hyperpigmented patches were distributed over the back and upper and lower limbs.

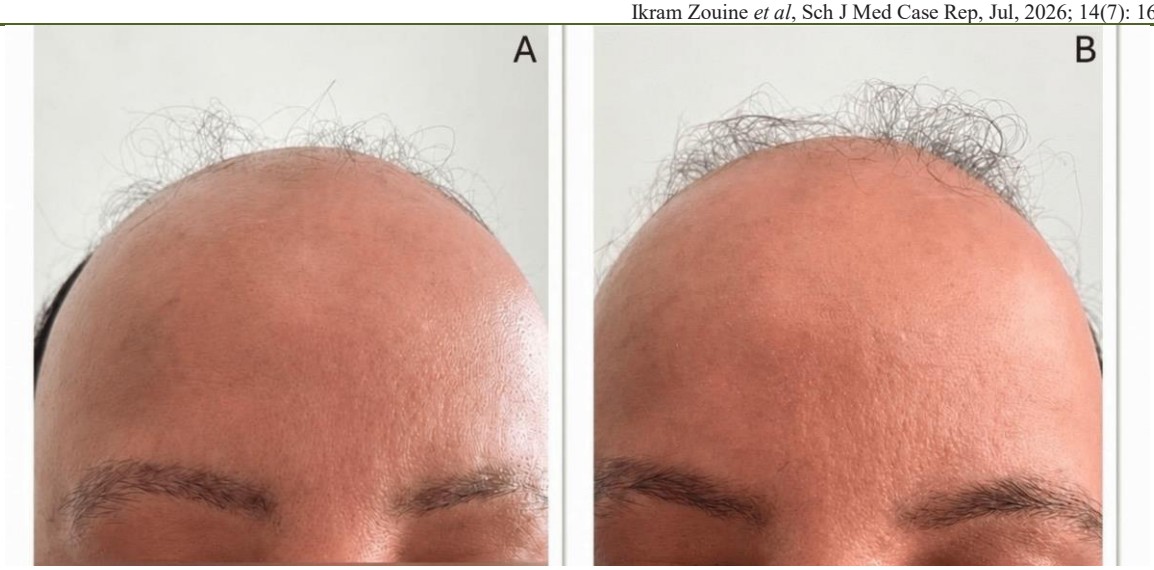

Figure 1: Frontal scalp at baseline (A) and follow-up 6 months after treatment (B), showing modest frontal scalp and eyebrow hair regrowth with overall disease stabilization

Three biopsies were obtained from clinically distinct sites. The frontal scalp specimen showed very sparse atrophic follicles, miniaturized anagen/vellus and telogen follicles, dilated follicular ostia, and perifollicular fibrous streamers, in keeping with chronic AA. The temporal scalp specimen showed an atrophic epidermis, interface inflammation directed at the follicular epithelium, a peri-isthmic lymphocytic infiltrate, cytoid bodies, perifollicular lamellar fibrosis, reduced follicular units, and sebaceous gland destruction, supporting LPP. A biopsy from the back showed mild orthokeratosis, basal hyperpigmentation, marked pigment incontinence, and numerous superficial dermal melanophages, consistent with LPPig. Clinicopathologic correlation therefore supported coexisting AA and LPP associated with LPPig.

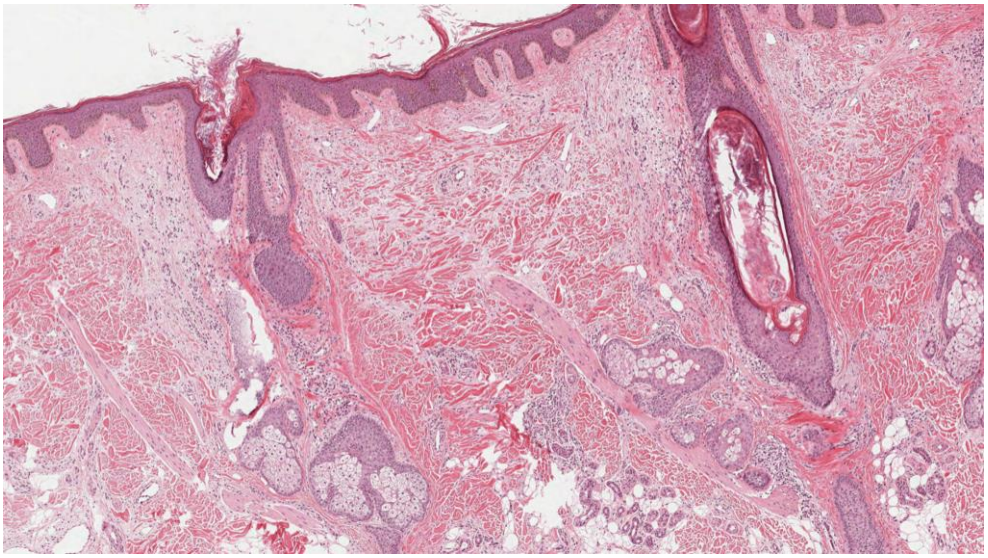

Figure 2: Vertical section from the temporal scalp showing perifollicular lichenoid inflammation and lamellar fibrosis at the infundibulo-isthmic level with reduced sebaceous units, supporting lichen planopilaris (hematoxylin-eosin, original magnification x100)

The patient received monthly intravenous methylprednisolone pulses at a dose of 500 mg combined with methotrexate 20 mg weekly. For LPPig, a compounded topical regimen containing a very potent corticosteroid, an emollient, and a depigmenting agent was prescribed. At four months, slight eyebrow regrowth was observed, without significant improvement of the scalp or body hair. At one year, the alopecia was globally stable with no further progression and modest scalp and eyebrow regrowth, while the pigmentation remained largely unchanged. Baricitinib was considered for the severe AA component but was not initiated because of financial constraints. Psychological support was incorporated because of the chronicity and psychosocial burden of the disease.

DISCUSSION

This case illustrates a diagnostically important overlap between a potentially reversible non-scarring alopecia and an irreversible cicatricial alopecia. AA classically affects the bulb, whereas LPP targets the upper permanent follicle, including the bulge region [1,2]. A shared T-cell-mediated loss of immune privilege may help explain coexistence, but the two processes remain anatomically and prognostically distinct. Only a limited number of simultaneous or co-localized AA-LPP cases have been reported [3,4].

The heterogeneous scalp examination was the key diagnostic clue. Black and yellow dots supported AA in frontal and occipital areas, while scar-like temporal patches and absent follicular openings suggested a cicatricial process. Biopsy of a single end-stage area could have missed one component. Sampling separates clinically active regions demonstrated chronic follicular miniaturization compatible with AA and a lichenoid peri-isthmic process with fibrosis and sebaceous gland loss diagnostic of LPP. This case therefore supports combining palpation and trichoscopy to choose biopsy sites and interpreting each scalp zone independently.

The associated LPPig further supports a lichenoid disease spectrum. Although the relationship between pigmentary lichen planus and classic LPP is incompletely understood, their coexistence has been described and may reflect related interface immune mechanisms [5]. In the present patient, the distribution of hyperpigmented patches and histologic pigment incontinence with melanophages supported LPPig rather than a fungal or purely post-inflammatory disorder.

Treatment must prioritize suppression of active LPP because follicular destruction is permanent. High-potency topical or intralesional corticosteroids and systemic agents such as hydroxychloroquine, methotrexate, cyclosporine, and mycophenolate mofetil are used, although the evidence base remains limited and no universally accepted regimen exists [6,7]. Methotrexate and corticosteroid pulses were selected to address both inflammatory components. Stabilization at one year suggests control of the scarring process, while limited regrowth is biologically plausible only in follicles not yet destroyed.

For severe AA, Janus kinase inhibition can produce clinically meaningful regrowth, and phase 3 trials have established the efficacy of baricitinib in adults [8]. Its expected benefit in an AA-LPP overlap is uncertain because it cannot restore follicles already replaced by fibrosis. In resource-limited settings, access to targeted treatment may further restrict therapeutic options. The principal limitations of this report are the absence of standardized activity scores, serial trichoscopic measurements, and a separate published histologic image of the AA component.

CONCLUSION

AA and LPP may coexist in the same patient and even within adjacent scalp regions. Recognition of the cicatricial component is essential because the therapeutic priority is to arrest irreversible follicular destruction. Regional clinical assessment, trichoscopy, and biopsies from appropriately selected active sites are central to diagnosis. The coexistence of LPPig in this patient supports a wider lichenoid immune spectrum and underscores the need for individualized, multidisciplinary management.

Consent for Publication: Consent for publication of the clinical information and images was obtained from the patient.

Conflicts of Interest: The authors declare no conflicts of interest.

Funding: No funding was received for this work.

AUTHOR CONTRIBUTIONS

Ikram Zouine: conception, data acquisition, interpretation, and drafting. Soukaina Chhiti, Achraf Miry, Zineb Mortaji, Nassma Ait Abdelali, and Radia Chakiri: clinical interpretation, critical revision, and approval of the final manuscript. Radia Chakiri: supervision. All authors agree to be accountable for the work.

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