Psoriasiform lichen planopilaris: Unusual variant or coincidence

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Key words: apremilast; cyclosporine; lichen planopilaris; methotrexate; psoriasis; risankizumab; scarring alopecia; treatment.

INTRODUCTION
While psoriasis can involve nearly any cutaneous surface, the scalp is considered to be the most commonly affected area, with some studies reporting a prevalence as high as 50% to 80%. Untreated scalp psoriasis can potentially lead to alopecia, which is typically nonscarring and limited to the area affected by psoriasis. Rare cases of scarring alopecia have also been described. Whether the scarring is due to scratching, secondary infection, or untreated inflammation is still unclear. We report a case of a patient with long-standing isolated scalp psoriasis, which rapidly progressed to cicatricial alopecia. Based on her clinical findings, histopathology, and failure to respond to conventional psoriasis treatments, she was ultimately diagnosed with psoriasiform lichen planopilaris. Herein, we discuss diagnostic and treatment considerations for this rare entity.

CASE REPORT
A 41-year-old Caucasian woman presented to our clinic with a 20-year history of scalp itching, flaking, and hair loss. The patient had a self-reported and family history of psoriasis. On examination, her right parietal scalp was diffusely pink with thick, silvery-yellow adherent scale (Fig 1). There was a noticeable decrease in hair density; however, trichoscopy did not demonstrate any evidence of scarring. The rest of her skin examination, including her nails, trunk, and extremities, was unremarkable. She was prescribed clobetasol 0.05% scalp solution and ketoconazole 2% shampoo.

At the 2-month follow up, she had noticeable progression of her alopecia (Fig 2). Two 4-mm punch biopsies were performed at the periphery and the center of the alopecic patch. Both biopsies exhibited psoriasiform dermatitis with no evidence of scarring or interface changes. The patient was subsequently started on risankizumab 150 mg subcutaneously at weeks 0, 4, and then every 12 weeks. At the 16-week follow up, the patient reported increased itching and worsening hair loss. Two additional 4-mm punch biopsies were taken from
the periphery and center of the alopecic patch. Histopathology was now significant for psoriasiform dermatitis with perifollicular fibrosis and lichenoid inflammation (Fig 3).

Risankizumab was discontinued, and the patient was started on cyclosporine 300 mg daily (4.5 mg/kg/day), which led to a dramatic reduction in her erythema, scaling, and itching in just one month. However, the patient did not want to continue with systemic therapy, preferring a natural alternative approach. She was tapered off of cyclosporine and started on turmeric, curcumin, fish oil, and a Mediterranean diet, which resulted in an abrupt flare of her symptoms and further progression of her hair loss.

The patient was restarted on cyclosporine and gradually transitioned to apremilast 30 mg twice daily. She was still quite symptomatic when her cyclosporine dose was below 100 mg daily, so subcutaneous methotrexate was added and titrated up until her disease was under control. At the time of this writing, the patient is off cyclosporine and maintained on apremilast 30 mg twice daily and methotrexate 20 mg weekly (Fig 4).

**DISCUSSION**

Our case demonstrates the diagnostic and therapeutic challenge of isolated psoriasiform scalp dermatitis with cicatricial alopecia. It is still unclear whether this patient has psoriatic scarring alopecia, an unusual psoriasiform lichen planopilaris, or scalp psoriasis with a coincidental cicatricial alopecia. Given the absence of psoriasis elsewhere on the body, the presence of perifollicular fibrosis and lichenoid inflammation, and her lack of response to risankizumab, we favored “psoriasiform lichen planopilaris”.

Rare cases of concomitant lichen planopilaris in patients with scalp psoriasis have also been described. In both reported cases, there were classic folliculocentric violaceous papules with perihilar scaling at the periphery of the alopecic plaque, which were notably absent in our patient.

One striking finding in this case was the rate at which our patient’s alopecia progressed. In 16 months, the affected body surface area went from approximately 0.3% to 1%, before it finally stabilized. Another notable observation was the lack of obvious scarring on her initial evaluation, even with trichoscopy and histopathology. Therefore, one must maintain a high index of suspicion for this potential complication, particularly if a patient is not responding to conventional psoriasis therapy. In the case reported by Lane et al., the patient also responded to cyclosporine, but there was no mention of maintenance treatment. Our patient promptly flared after tapering her cyclosporine, so apremilast was initially chosen given its efficacy in scalp psoriasis and its successful off-label use for interface and lichenoid dermatitis.
In summary, we report an unusual case of psoriasiform lichen planopilaris, which clinically may appear indistinguishable from scalp psoriasis but which is more refractory to treatment with the potential to rapidly progress to cicatricial alopecia. Histopathology remains critical for diagnosis, and serial biopsies may need to be performed before a diagnosis can be confirmed. Treatment remains a challenge, and the authors suggest that traditional immunosuppressants such as cyclosporine and glucocorticoids should be first-line therapeutics to control the active flare, while methotrexate and apremilast are reasonable choices for long-term maintenance.

Conflicts of interest
Dr Song, MD, has been a consultant, speaker, or investigator for the following companies: AbbVie, Janssen, Amgen, Lilly, SUN, and UCB.

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