Colocalisation of alopecia areata and lichen planus

Sir,

Frequent associations between alopecia areata and immune-mediated cutaneous disorders have been reported.[1] Being common skin disorders, lichen planus and alopecia areata may rarely coexist. We report a case of co-localization of lichen planus and alopecia areata.

A 42-year-old man presented with a single patch of non-scarring hair loss of 4 months' duration over the right parieto-occipital region. Alopecia areata was diagnosed and he was treated with topical betamethasone dipropionate. With that the lesion became static. He had no other lesion on any hair-bearing area.

Three months after the appearance of the initial lesion he developed a solitary violaceous papule in the center of the patch (Figure 1). The lesion was pruritic and mildly scaly. No mucosal lesion was present. A biopsy from the patch of alopecia revealed a typical perifollicular "swarm of bees" type lymphocytic infiltrate consistent with a diagnosis of alopecia areata (AA) (Figure 2). A biopsy from the central papular lesion showed hyperkeratosis, wedge-shaped hypergranulosis, irregular acanthosis, basal cell liquefaction and a band-like lymphocytic infiltrate and pigment incontinence in the superficial dermis suggestive of a diagnosis of lichen planus (LP) (Figure 3).

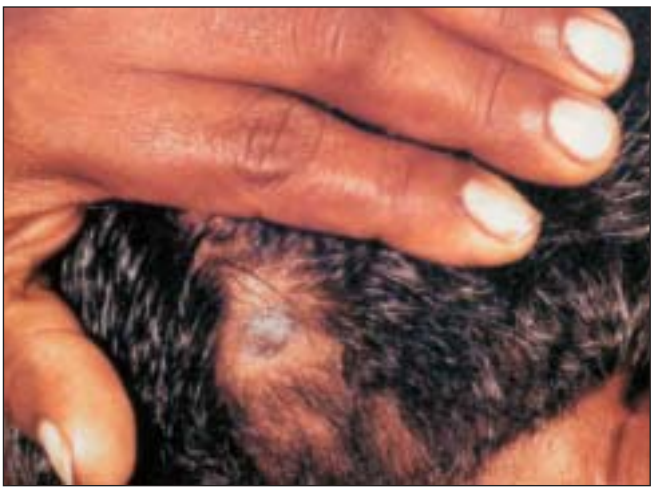

Figure 1: A patch of alopecia areata on the right parieto-occipital region with a centrally located papule of lichen planus

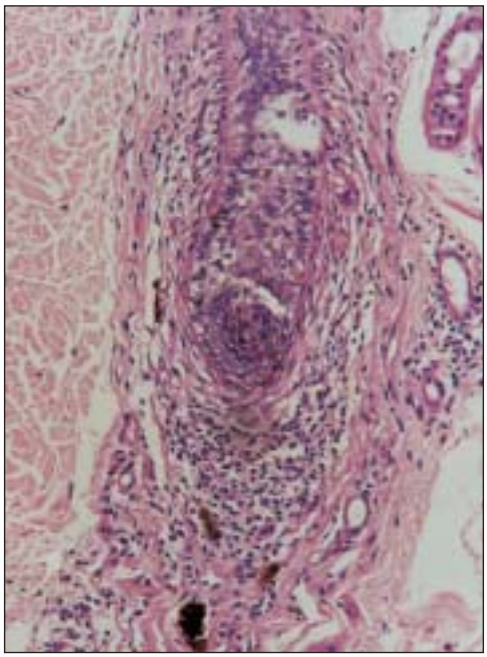

Figure 2: Perifollicular lymphocytic infiltrate of alopecia areata (H&E, 200x)

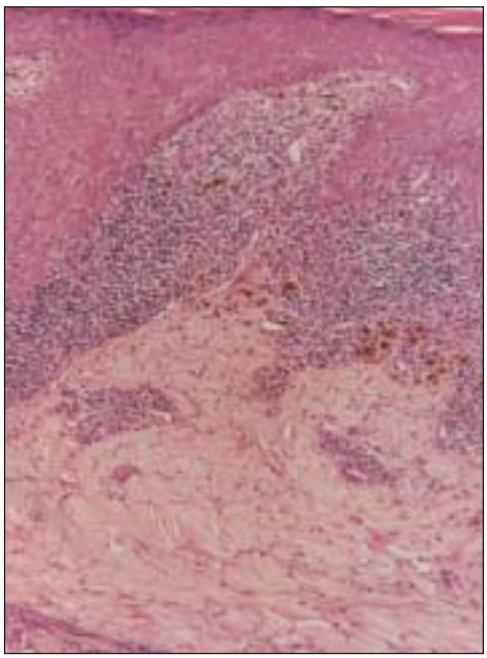

Figure 3: A band of lymphocytic infiltrate in the upper dermis with plenty of melanin incontinence suggestive of lichen planus (H&E, 100x)

Kanwar et al reported 20-nail dystrophy in a patient of AA due to LP.[2] Brenner et al reported a case of coincidence of five dermatological disorders: vitiligo, AA, onychodystrophy, morphea and LP.[3] Similarly, ulcerative colitis, myasthenia gravis, LP, AA and vitiligo were present in a single patient reported.[4] Patients with AA were found to be at a higher risk for developing LP (RR=2.7; 95% confidence interval, 1.1 to 6.5).[5] However, co-localization is very rare. Dhar et al had reported one child with co-localization of lesions both conditions.[6] The incidence of AA in the Indian population is 0.7%7 whereas it is 0.8% for LP.8 The coexistence of these disorders may be purely coincidental. Gilhar et al found that induction of AA was possible with injection of CD8+ cells cultured with follicular homogenate but not with cultured CD4+ cells.[9] The T lymphocyte is also pivotal in regulating epidermal cell recognition and epithelial destruction in lichen planus. T cells become activated via antigen-presenting cells such as Langerhans cells in conjunction with epidermal keratinocytes and co-stimulatory molecules. Though both CD4+ and CD8+ T cells are found in the lesional skin of LP, progression of disease leads to the preferential accumulation of CD8+ cells.[10] The majority of the lymphocytes in the infiltrate of LP are CD8+ and CD45RO (memory)-positive cells and express the g/d T-cell-receptor. The ensuing immune reaction by CD8+ T lymphocytes against activated keratinocytes results in epidermal cell damage and development of the lichenoid reaction that is the hallmark of lichen planus.

Further studies might clarify whether co-localization of lichen planus and alopecia areata is a mere coincidence or represents a common pathogenic mechanism in these two predominantly CD8+ T lymphocyte-mediated disorders.

Bikash Ranjan Kar, Gigi Ebenezer*, C. K. Job*
Departments of Dermatology and *Pathology, Schieffelin Leprosy Research & Training Centre, Karigiri, Vellore, India.

Address for correspondence: Dr. B. R. Kar, Dept of Dermatology, SLR & TC, Karigiri, India. E-mail: karbikash@hotmail.com

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ANA-negative systemic lupus erythematosus

Sir,

We report a case of ANA-negative systemic lupus erythematosus diagnosed on the basis of ARA criteria.

A 27-year-old female presented with fever and bilaterally symmetrical dusky erythematous, scaly papules and patches with pigmentation over the scalp,

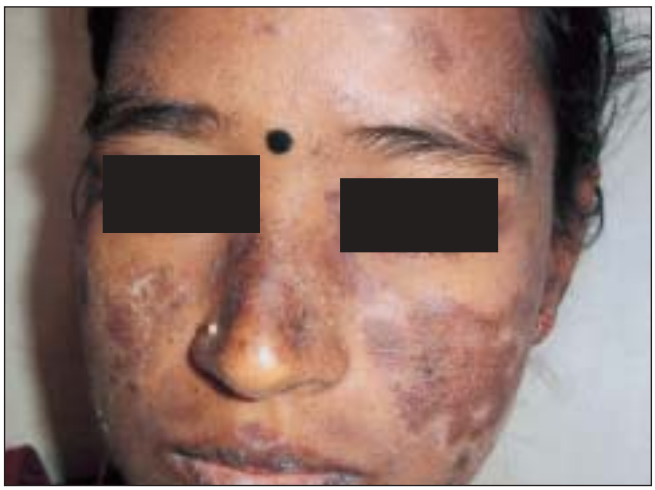

Figure 1: Scaly skin lesions over malar area of the face