**Case Report**

**Basal cell carcinoma arising within an alopecic patch of lichen planopilaris**

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**Key words:** basal cell carcinoma; carcinogenesis; corticosteroids; lichen planopilaris; lichen planus; malignant; transformation.

**Introduction**

Lichen planopilaris (LPP) is variant of lichen planus (LP) that affects the hair follicles of the scalp. In its classic form, LPP presents with erythema, hyperkeratotic papules coalescing into plaques, and cicatricial alopecia of the vertex scalp. Chronic mucosal and hypertrophic cutaneous forms of LP may have malignant potential, but the malignant potential of LPP is unknown. Herein, we present a case of a basal cell carcinoma (BCC) arising within a long-standing lesion of LPP.

**Case**

A 36-year-old Caucasian (Fitzpatrick II) female presented to a dermatologist with a 4-year history of generalized hair shedding associated with erythema, hyperesthesia, and scaling of the scalp. She had previously been diagnosed with LPP by another dermatologist. On physical examination, a 1.5 cm patch of scarring alopecia with perifollicular scale at the margins was noted on the left parietooccipital vertex scalp. The scalp otherwise appeared normal, and no cutaneous lesions were noted elsewhere on the body. The oral mucosa also appeared normal. One mg of triamcinolone (0.2 mL of 5 mg/mL solution) was injected into the scalp lesion at the initial visit. The lesion remained unchanged at subsequent follow-up 1 month later when 0.15 mL of triamcinolone 5 mg/mL was injected into the same area. Concurrently, the patient was evaluated for worsening dryness and irritation of the eyes. Slit-lamp examination showed subepithelial fibrosis of the left upper palpebral conjunctiva and blunting of the bilateral inferior conjunctival fornices, consistent with ocular manifestations of LP.

Four months after initiation of treatments, the area of scarring alopecia was persistent without appreciable inflammation. A new tan-colored, pearly papule was noted at the anterior aspect of the alopecic patch (Fig 1). A shave biopsy of the papule and a 4 mm punch biopsy within the adjacent area of alopecia were obtained. Histopathology revealed BCC in both specimens (Fig 2). The punch biopsy also demonstrated features of LPP with prominent perifollicular fibrosis, lymphocytic inflammation, and polytrichia (Fig 3). The patient was referred for Mohs surgery. Review of the patient’s medical, social, and family history was significant for melanoma in both parents. The patient did not have a personal history of skin cancer, and she had no history of tanning bed use. After 3 Mohs stages, negative histologic margins were obtained with a final defect of 3.6 × 1.7 cm to a depth of subcutaneous fat. The wound was repaired with simple closure with interrupted 4-0 Nylon sutures (Ethicon) and the patient experienced no complications.

**Abbreviation used:**

BCC: basal cell carcinoma
IL: interleukin
LP: lichen planus
LPP: lichen planopilaris
SCC: squamous cell carcinoma

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DISCUSSION

Malignant transformation is an uncommon complication of LP. The majority of such cases involve squamous cell carcinoma (SCC) arising within LP lesions of the oral, esophageal, or genital mucosa. The lifetime risk of SCC in patients with oral LP is around 1%. One retrospective study reported a 6.1% risk of esophageal SCC in patients with esophageal LP. There have been sporadic reports of SCC developing within cutaneous LP, but such cases are rare and typically occur in long-standing hypertrophic and ulcerated disease. Five cases of SCC in association with LPP have been reported in the literature.

To our knowledge, this is the first report describing a BCC arising within an LP lesion. There is one previously reported case of BCC developing in long-standing cutaneous LP. Chronic inflammation is known to be pro-tumorigenic through the release of Th2 cytokines interleukin 4 (IL-4), IL-6, IL-10, IL-13, and transforming growth factor beta. Interestingly, acute inflammation is antitumorigenic with a Th1 response and high levels of IL-2 and interferon gamma. Medications that target Th1 activity, such as Janus kinase inhibitors, are known to increase the risk of malignancy through blockade of antitumoral immunity. LP is also an Th1, interferon gamma–driven process that resolves with selective targeting of interferon gamma–associated genes. In our case, the response to intralesional triamcinolone possibly

Fig 1. Flesh-colored papule (A) within a patch of alopecia on the patient’s vertex scalp. Perifollicular scale is present at the margins of the lesion. A, Shave biopsy site. B, Punch biopsy site.

Fig 2. Basaloid tumor islands connected to the epidermis with peripheral palisading and fibromucinous stroma in both specimens.

Fig 3. Polytrichia with the prominent perifollicular fibrosis and scattered lymphocytes.
correlates with diminution of the Th1 response and, therefore, the antitumoral response, which may have led to unmasking of a BCC.

The case described herein represents a unique case of a BCC arising in association with resolving LPP. Importantly, in this case, the overlapping lesions occurred on the scalp. While the patient’s LPP did produce a small area of alopecia gradually over a 4-year period, it is possible that the development of the BCC was coincidental given the scalp is a common location for this tumor. However, the region remained generally sun-protected by the surrounding hair, which underscores the potential for additional immunologic changes that may have contributed to the development of BCC. Tissue cytokine and transcriptomic analysis of malignancies arising in LP lesions may offer insight into the mechanisms behind malignant transformation in LP and should be explored in future studies.

Conflicts of interest
None disclosed.

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