



Hypertrophic lichen planus mimicking squamous cell carcinoma: The importance of clinicopathologic correlation

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INTRODUCTION

Hypertrophic lichen planus (HLP) and squamous cell carcinoma (SCC) share many clinical and histopathologic characteristics, making them difficult to distinguish. HLP may undergo malignant transformation, leading to the development of SCC within HLP. We present a case of HLP that mimicked SCC and was referred for surgical intervention. This case underscores how increased awareness of subtle clinical and histopathologic features that distinguish HLP from SCC can help providers promote timely initiation of appropriate treatment.

CASE REPORT

An 83-year-old woman was referred for Mohs micrographic surgery for a biopsy-proven, well-differentiated, invasive SCC on the left midpretibial area. Five months before presentation, she noticed an erythematous plaque on her left lower extremity that subsequently enlarged over the next few weeks and became hyperkeratotic. Inflammatory papules appeared around the plaque and grew progressively more erythematous with associated pain and pruritus. She denied bleeding or ulceration. Initial biopsy was consistent with hypertrophic actinic keratosis with superimposed marked inflammatory infiltrate including eosinophils. Subsequent biopsy result was consistent with well-differentiated, invasive SCC, prompting referral. History was notable for significant sun exposure, including blistering sunburns and a prior SCC on her left cheek. Physical examination found an ill-defined scaly, erythematous 5.5-cm plaque on the left midpretibial area surrounded by

Abbreviations used:

HLP: hypertrophic lichen planus
LP: lichen planus
PEH: pseudoepitheliomatous hyperplasia
SCC: squamous cell carcinoma

multiple discrete erythematous scaly papules and plaques on the left medial and lateral pretibial areas (Fig 1). Examination of the skin, nails, and oral mucosa was otherwise unremarkable.

Given the ill-defined borders of the plaque, scouting punch biopsy sections were obtained to help delineate the borders of the presumed malignant process. Biopsy of the plaque on the left midpretibial area, which was initially read as SCC, showed mild hyperkeratosis, wedge-shaped hypergranulosis, and irregular acanthosis with adjacent well-differentiated endophytic squamous epithelium and associated marked lichenoid chronic inflammation including eosinophils and numerous plasma cells (Fig 2). Biopsy of an adjacent plaque on the left posterior pretibial area found an endophytic squamous proliferation with pseudohorn cysts and a lichenoid infiltrate. Biopsy of the surrounding inflammatory papules showed lichenoid dermatitis with eosinophils. No perforating elastic fibers were seen on elastic stains. After careful clinicopathologic correlation, re-review of the initial biopsy, and discussion with the dermatopathology service, HLP was diagnosed. The plaque initially diagnosed as SCC was treated with high-potency topical steroid ointment (betamethasone dipropionate) under

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Fig 1. Hypertrophic lichen planus clinical presentation. Erythematous hyperkeratotic plaque (arrow) surrounded by violaceous papules on the left pretibial area before treatment.



Fig 3. Hypertrophic lichen planus posttreatment. Decreased thickness, erythema, and scaling of the plaque on the left pretibial area (arrow) after topical and intralesional steroids.

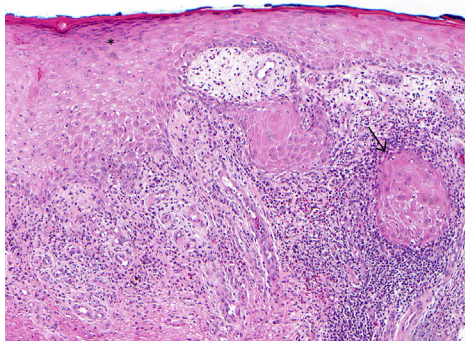


Fig 2. Hypertrophic lichen planus histopathology. Left midshin skin punch biopsy result shows hyperkeratosis, wedge-shaped hypergranulosis (asterisk), irregular epidermal hyperplasia, and endophytic well-differentiated squamous epithelium (arrow) with associated lichenoid inflammation and eosinophils. (Hematoxylin-eosin stain; original magnification: $\times 100$.)

occlusion with an Unna Boot as well as intralesional triamcinolone acetonide (10 mg/mL, total of 3 mL) with subsequent resolution of the plaque and associated pruritus (Fig 3).

DISCUSSION

HLP and SCC share many clinical and histologic characteristics, making them difficult to distinguish. The task of differentiating HLP from SCC is complicated by concern that HLP may undergo malignant

transformation, leading to the development of SCC within HLP.¹ This case underscores the importance of recognizing HLP as a mimicker of SCC to avoid unnecessary interventions and ensure timely initiation of appropriate treatment. At least 4 patients with HLP mistaken as SCC have been reported in the literature, highlighting the challenge of identifying the correct diagnosis.^{2,3}

HLP is a distinct variant of cutaneous lichen planus (LP), an idiopathic T cell–mediated autoimmune disorder that may affect the skin or mucosal surfaces.⁴ HLP characteristically presents with pruritic violaceous papules and polygonal plaques crossed by fine white lines known as *Wickham striae*. HLP primarily affects the lower extremities, especially the shins, ankles, and interphalangeal joints.⁵ The thick, scaly, hyperkeratotic plaques of HLP may resemble SCC on clinical examination.

The histopathology of HLP is variable. Typical features of LP, including hypergranulosis and basal cell vacuolar degeneration, may not be present.² Like other chronic inflammatory conditions associated with rubbing and irritation, HLP may be associated with features of pseudoepitheliomatous hyperplasia (PEH), which can be difficult to distinguish from SCC.^{2,6} PEH is characterized by hyperplasia of the epidermis and adnexal epithelium, gross irregular acanthosis, and horn cyst formation.^{2,3,6} However, unlike SCC, PEH does not infiltrate into the reticular

Table I. Clinical and histologic features helpful for differentiating hypertrophic lichen planus from squamous cell carcinoma

Clinical	Histologic
Hyperkeratotic plaque(s) on the distal extremities, especially the shins	Hyperorthokeratosis, wedge-shaped hypergranulosis, and irregular psoriasiform hyperplasia of the epidermis
Presence of multiple plaques with follicular accentuation	Lichenoid dermatitis with eosinophils
Pruritus	Classic features of pseudoepitheliomatous hyperplasia
Wickham striae	No cytologic atypia
Typical lichen planus affecting oral mucosa, nails, and skin elsewhere	Absence of marked solar elastosis, no perforating elastic fibers
Negative history of sun damage	No deep extension beyond the superficial dermis
No predisposing factors for multiple SCCs	No lymphovascular or perineural invasion

dermis or invade blood vessels, nerves, or lymphatics.^{2,3}

The relationship between HLP and SCC remains poorly characterized. Although there have been nearly 50 case reports of SCC arising in HLP, a population-based study of 2,071 Swedish patients found no increased frequency of SCC in patients with cutaneous LP.^{5,7} However, the potential for malignant transformation demands careful clinicopathologic correlation to avoid misdiagnosis of SCC, as widely metastatic SCC originating from HLP has been reported.⁸ Patients with SCC arising in HLP tend to present with longstanding nonhealing ulcers⁹ and HLP of long duration; the average time reported between diagnosis of HLP and development of SCC is 11 to 12 years.¹ Malignant transformation of HLP is thought to be associated with arsenic exposure, radiation, and chronic tar application.^{1,8}

HLP should be considered in the presence of several key clinical and histologic features (Table I). HLP should be on the differential diagnosis for any thick, scaly, pruritic, hyperkeratotic plaque on the distal extremities, particularly the shins, and in the presence of multiple lesions, especially in a patient with no history of sun damage or predisposing factors for multiple SCCs. Wickham striae and evidence of typical LP on examination of the oral mucosa, nails, and skin elsewhere further support the diagnosis of HLP. A deep biopsy section should be obtained if HLP is suspected; superficial biopsy specimens may not include a sufficient portion of the dermis to differentiate between HLP and SCC.⁶ Histologic features suggestive of HLP include hyperorthokeratosis, wedge-shaped hypergranulosis, and psoriasiform hyperplasia of the epidermis⁵; lichenoid interface dermatitis with eosinophils¹⁰; presence of typical features of PEH; and absence of cytologic atypia, marked solar elastosis, deep extension beyond the superficial dermis, and lymphovascular

or perineural invasion.^{2,3} Elastic staining to visualize the presence or absence of perforating elastic fibers may assist in distinguishing between SCC and HLP; perforating elastic fibers are rarely present in HLP but may be seen in SCC.^{11,12}

This case highlights the challenge of differentiating HLP and SCC. Awareness of the distinguishing features of HLP is essential for clinicians to provide timely and appropriate treatment to their patients.

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