Hypertrophic lichen planus: masquerading as squamous cell carcinoma

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Summary
We present two cases of hypertrophic lichen planus mistaken for squamous cell carcinomas which were excised on multiple occasions. With appropriate histopathological review and treatment with topical corticosteroids, the ‘lesions’ showed significant improvement with near resolution at six months. This highlights the clinically difficulty with distinguishing hypertrophic lichen planus from squamous cell carcinoma. On literature review, mucosal lichen planus has a well known associated risk of squamous cell carcinoma, however, there have been isolated case reports of squamous cell carcinoma arising from hypertrophic lichen planus.

KEY WORDS: hypertrophic lichen planus; squamous cell carcinoma; keratoacanthoma; topical corticosteroids.

Case based review
Case 1
A 76-year-old male presented to a general surgeon in 2009 with a right pretibial nodule present for several months which was thought to be squamous cell carcinoma (SCC). It was excised and reported as lichen simplex chronicus. In 2010, he had his first recurrence of the right pretibial nodule and underwent another excision, which was reported as an atypical squamous proliferative lesion, clear of resection margins with keratoacanthoma favoured over well differentiated SCC. He then had a second recurrence of a right pretibial nodule in 2012 and had a third excision which was reported as a well differentiated SCC completely excised with changes of lichen simplex chronicus. In February 2013 he then presented with multiple nodular lesions on his left lower leg, with a biopsy reporting well differentiated SCC and a recurrent nodule on the right lower leg which was reported was well differentiated SCC. However due to the unusual presentation of bilateral leg involvement he was referred to the Peter MacCallum Cancer Centre.

A PET scan was performed as is the standard practice for metastatic SCC at this hospital which showed extensive nodular lesions in left lower leg and right medical calf lesion which were FDG avid. There was no evidence of nodal or distant spread.

On dermatology review, he had very little background solar damage or features of such as solar keratoses and SCC-in-situ (Bowen’s disease) and had no predisposing factors such as immunosuppression or drug therapy which would be expected in a patient with multiple SCCs. On further examination, he had multiple pruritic violaceous plaques on bilateral lower legs, arms and scalp (Figure 1). It was felt generalised hypertrophic lichen planus was the unifying diagnosis. There was no evidence of mucosal lichen planus.

Biopsies were then performed of a left arm plaque which was reported as hypertrophic lichen planus and of a right leg nodule as pseudoepitheliomatous hyperplasia associated with hypertrophic lichen planus but well differentiated SCC could not be excluded.

Histopathology review of all the earlier biopsies showed the 2009 biopsy as hypertrophic lichen planus over an old scar, the 2010 right lower leg biopsy as keratoacanthoma which was completely excised, the February 2013 left lower leg biopsy showed hypertrophic lichen planus (Figures 2, 3) and the right lower leg biopsy showed well differentiated SCC with a surface papillary pattern.

Due to the multiple sites involved including arms, legs and scalp, he was treated with topical betamethasone dipropionate in optimised vehicle 0.05% ointment daily and topical salicylic acid 5% daily with significant improvement of the multiple nodular lesions on the lower legs and arms. As the repeat biopsy of the right lower leg in February 2013 was reported as SCC, a repeat incisional biopsy was performed on the proximal tip of the right calf in April 2013 after treatment with topical corticosteroids which excluded malignancy.

He continued to use topical betamethasone dipropionate in optimised vehicle 0.05% ointment daily to all the plaques for five months and had one course of intralesional triamcinolone acetonide 10mg/ml injections to the right calf, left calf and occipital scalp nodules. After six months of treatment, he had significant im-
Improvement of his lower legs, scalp and arms with only small violaceous plaques present on the left leg and right lower leg (Figure 4). No biopsies were performed post treatment.

**Case 2**

This is a 73-year-old male who presented to a surgeon with multiple lesions which have been excised from his lower legs over the past three to four months which were reported to be SCC on histology from an external laboratory. He has no past history of skin cancer.
He also complains of severe pruritus of the lower legs over the last four months. He has had no loss of appetite or weight loss. His other medical problems include ischaemic heart disease, obstructive sleep apnoea, hypertension and pancytopenia.

On examination he has multiple violaceous scaly papules and plaques over the lower limbs and extensor aspects of the forearms consistent with hypertrophic lichen planus. There was no lymphadenopathy or hepatosplenomegaly. There were no suspicious lesions for skin cancer and minimal background sun damage. There was no evidence of mucosal lichen planus.

On histopathology review at the Peter MacCallum Cancer Centre all six excisions from both legs were overturned to hypertrophic lichen planus with marked pseudoepitheliomatous hyperplasia. A representative slide of the lesion excised from the left calf is shown in Figures 5 and 6.

He was treated with topical mometasone furoate 0.1% ointment daily with significant improvement of his symptoms on review at six months. No biopsies were performed post treatment.

**Discussion**

Lichen planus is an autoimmune disorder with characteristically small, violaceous papules with a network of fine white lines (Wickham’s striae) which can be widely dispersed or coalesce into larger plaques. It can be pruritic and Koebner phenomenon can be present. It commonly involves the wrists, dorsum of the hands, anterior aspect of the lower legs, neck and sacrum (1, 2). Hypertrophic lichen planus usually has thicker, pruritic red-brown to purple plaques with follicular accentuation that commonly affects the limbs. Mucosal sites are involved in 30-70% of cases so a thorough oral, hair and genital mucosal examination should occur (3).

The mainstay of treatment is topical, intralesional and systemic corticosteroids. However, resistant cases may require immunosuppressive or immunomodulatory agents such as topical calcineurin inhibitors, systemic retinoids, narrowband UVB, PUVA or antimalarials (2, 3).

These are both interesting cases which demonstrate hypertrophic lichen planus and well differentiated SCC can be difficult to differentiate on histopathology. Typical histology of lichen planus is hyperkeratosis without parakeratosis; focal increase in granular cell layer; irregular, basal cell layer degeneration; band-like lymphocytic infiltrate at the dermo-epidermal junction; colloid bodies in the lower epidermis and pigment incontinence (3).

Pseudoepitheliomatous hyperplasia can be present in hypertrophic lichen planus and keratoacanthoma which can be confused with SCC. This is characterised by gross acanthosis and downward proliferation with horn cysts. However, there is no lymphovascular or perineural invasion and no involvement of deep dermis which are features of malignancy (3).

There have been 7 case reports of pseudoepitheliomatous hyperplasia seen on histopathology on patients with lichen planus-like lesions with minimal background solar damage which were called keratoacanthoma. Three cases were treated with acitretin, two cases with intralesional steroids, two cases with surgical excision and one case with etretinate and topical corticosteroids. Both hypertrophic lichen planus and keratoacanthomas are responsive to retinoids and corticosteroids so this cannot be used as a distinguishing feature (4-9).

Interesting to note, mucosal variants of lichen planus have a known association with transformation to SCC however, this is not the case for generalised cutaneous lichen planus (3). However on review of the literature, there are 91 single reported cases or small case series demonstrating SCC in cutaneous lichen planus (8-32). The significance of this is still yet to be determined with no link found in large studies. However, due to the immediate response and resolution of both cases we present with topical corticosteroids, it is likely that the underlying pathology was lichen planus rather than metastatic SCC.

In conclusion, the similarities of hypertrophic lichen planus to squamous cell carcinoma can cause confusion on clinical examination and histopathology.
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These cases highlight identifying the correct diagnosis of hypertrophic lichen planus can avoid unnecessary cutaneous surgery and earlier intervention with the appropriate treatment.

References