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Lichenoid and interface dermatoses

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A B S T R A C T

The terms 'lichenoid' and 'interface' dermatitis are often used interchangeably to describe an inflammatory pattern characterized histologically by damage to the basal keratinocytes in the epidermis. The mechanism of cell damage of such cells is now best understood as apoptosis, or programmed cell death. This inflammatory pattern of dermatoses, is also accompanied frequently by a band of lymphocytes and histiocytes in the superficial dermis, that often obscures the dermal-epidermal junction, hence the term 'lichenoid'. A discussion of the more common lichenoid/interface dermatitides encountered in the routine clinical practice encompasses the following entities: lichen planus, lupus erythematosus, dermatomyositis, erythema multiforme, graft versus host disease, fixed drug reactions, and multiple others.

Introduction

The terms 'lichenoid' and 'interface' dermatitis are often used interchangeably to describe an inflammatory pattern characterized histologically by damage to the basal keratinocytes in the epidermis.[1,2] The mechanism of cell damage of such cells is now best understood as apoptosis, or programmed cell death.[3] The apoptotic cells in the epidermis are also referred to as 'Civatte bodies', and the ones in the dermis as 'colloid bodies'.[4] Another frequent characteristic of this group of disorders is the presence of vacuolar degeneration of the basal keratinocytes. As consequence of such changes, there is variable melanin pigment incontinence in the dermis because of the transfer of the pigment from the keratinocytes. This inflammatory pattern of dermatoses, is also accompanied frequently by a band of lymphocytes and histiocytes in the superficial dermis, that often obscures the dermal-epidermal junction, hence the term 'lichenoid'. A discussion of the more common lichenoid/interface dermatitides encountered in the routine clinical practice is followed thereafter.

Lichen planus and variants

Lichen planus (LP) is a lichenoid and interface dermatosis of unknown etiology.[5–7] The clinical lesions appear as flat-topped papules and plaques with a violaceous appearance with a predilection for the flexor surfaces of the trunk, thighs and genital areas (Fig. 1). The lesions are intensely pruritic, and frequently have fine white lines across them, also referred to as Wickham's striae. Oral lesions are very frequent, and sometimes can be the sole manifestation of the disease.[6] In rare occasions the mucosal surface of the esophagus can be involved.[8–10] The lesions typically last for approximately 12 months, before showing spontaneous resolution, the latter associated with post-inflammatory hyperpigmentation. While LP can be associated with a variety of diseases (autoimmune[11–13], immunodeficiency states[14], malignancy[15,16], etc.), the most notorious association is linked to the infection with HCV virus.[17–19] Indeed, most patients should be screened for HCV infection upon the diagnosis of LP.

LP represents a model of what lichenoid and interface dermatitides are.[2,7,20,21] In LP the epidermis is often times acanthotic, with a somewhat irregular pattern of elongation of the rete ridges (Fig. 1). Such irregular pattern, often adopts a 'sawtooth' configuration. The granular cell layer is often more cellular, and has a wedge-shaped configuration. There is basal cell damage of the keratinocytes with the presence of multiple apoptotic cells, Civatte bodies, and eosinophilic colloid bodies in the dermis. Vacuolar degeneration of the basal keratinocytes is visible, and there is typically a 'lichenoid' band of lymphocytes and histiocytes that obscures the dermal-epidermal junction. Eosinophils are not typically seen, but are more frequent in the hypertrophic forms of LP. Occasional plasma cells can be present, particularly when located in mucosal sites.[22] When the vacuolar changes are very prominent, a cleft between the epidermis and dermis forms, which is often referred to as 'Caspary-Joseph spaces'. The older the lesion is, the more frequent pigment incontinence is noted.

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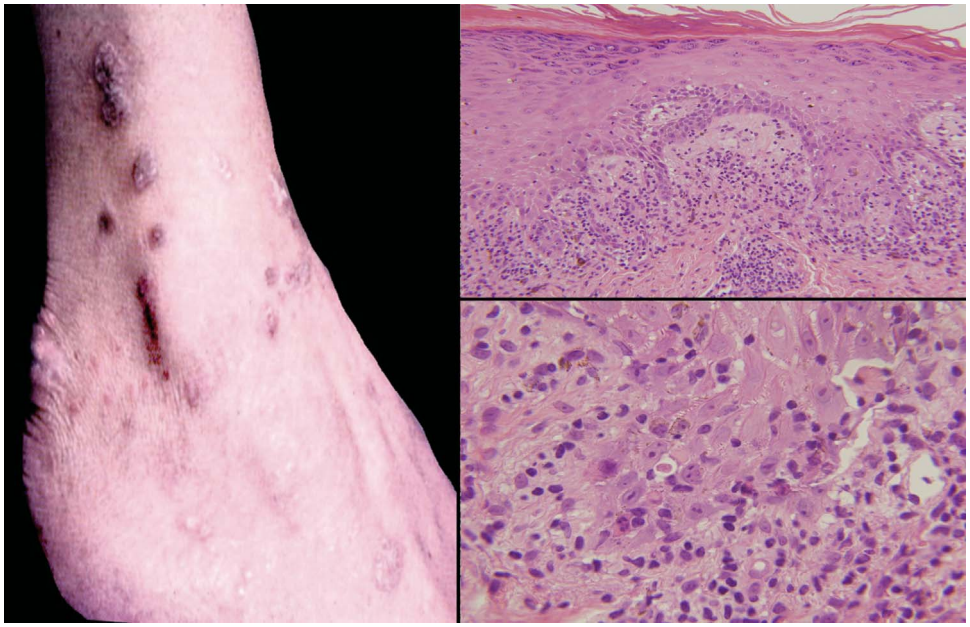

Fig. 1. Lichen planus. Flat topped papules with a violaceous appearance on the ankle. The epidermis is acanthotic with hyperkeratosis on the surface. The acanthosis is irregular and has a saw-tooth appearance. Areas of wedge-shaped hypergranulosis are present. The lichenoid and interface changes are illustrated by the presence of 'civatte bodies' (apoptotic cells). A lichenoid band of lymphocytes and histiocytes is also present.

In ***atrophic lichen planus***[23–25]**,** the epidermis is thinned and there is loss of the normal rete pattern. The degree of inflammation is also less prominent than typical lesions of LP. Changes of atrophic LP can clinically resemble porokeratosis. In ***hypertrophic lichen planus (HLP)***, the lesions are more typically described as plaques on the lower extremities, and sometimes have a verrucous appearance. Hypertrophic changes occur in long-standing lesions of LP. The association between HLP and viral infections (HIV, HCV) is higher than classic LP lesions.[26,27] The other important feature to remember of HLP is the higher predisposition to develop squamous cell carcinoma and keratoacanthomas of the skin, similarly to what occurs in hypertrophic lupus erythematosus.[28–31] HLP is characterized, as the name implies, by epidermal hyperplasia with marked hyperkeratosis. Changes of lichen simplex chronicus are often admixed with HLP. It is also important to be careful when diagnosing a squamous cell carcinoma on a superficial shave biopsy of a patient with long-standing history of LP, as the treatment is widely different.

Annular lichen planus is more of a clinical, rather than a histologic diagnosis.[32] This is a rare variant of LP with lesions presenting in the axilla, penis, extremities and groin, where clinically resembles lesions of morphea, mycosis fungoides or gyrate erythemas. Similarly, ***linear lichen planus*** shows a distinctive distribution along the lines of Blaschko, and many of these cases can

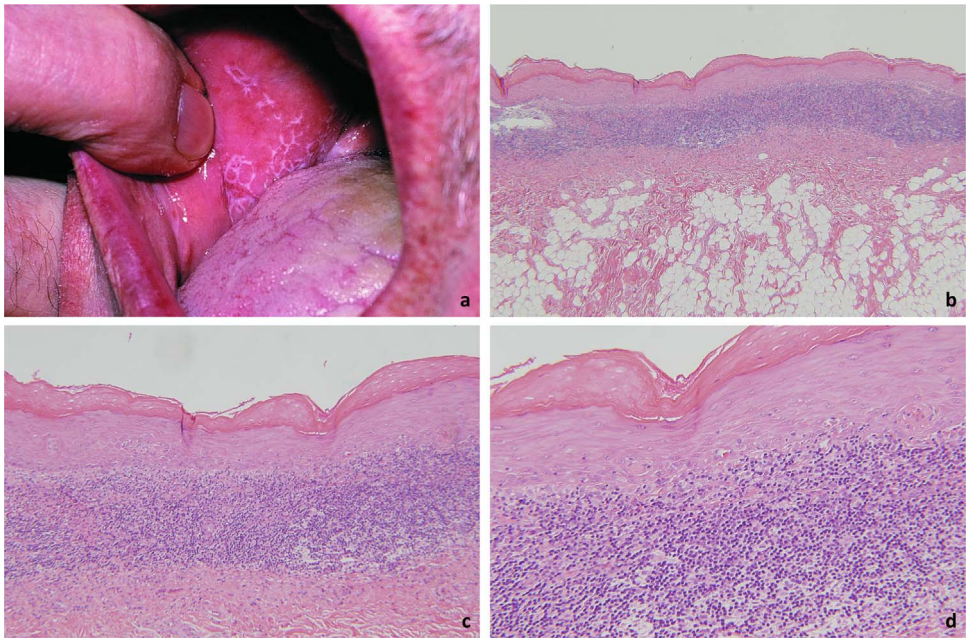

Fig. 2. Oral lichen planus. The presence of white linear striae are seen in the mucosal cheek (2a). The oral mucosa is acanthotic, hyperkeratotic and shows a lichenoid band (2b, 40x). This interface and lichenoid mucositis has scattered plasma cells in the infiltrate. Note the absence of the hypergranulosis that is frequently seen in the skin (2c and 2d, 100x and 200x).

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