

## Pantomime Inflammation or Neoplasm: Lichen Planus-Like Keratosis

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### Preface

Lichenoid keratosis is a benign, localized dermatosis which can also be designated as lichen planus -like keratosis. Lichen planus-like keratosis was initially described in 1966 as an incomplete or frustrated form of lichen planus. However, various demarcations on clinical grounds have defined it as a distinct entity. Lichen planus -like keratosis commonly implicates middle aged or elderly Caucasians. The condition can be considered as a variant of seborrheic keratosis with preponderant inflammation. Lichen planus -like keratosis can appear as an indeterminate lesion or simulate benign or malignant lesions on clinical representation and on dermoscopic assessment, hence an adequate tissue sample is necessitated for evaluation [1,2].

### Disease characteristics

Lichen planus -like keratosis is cogitated as a rapidly progressive, solitary, inflammatory lesion with a magnitude varying betwixt of 5 millimetres to 20 millimetres and a predilection for upper extremities, face and anterior torso. Lichenoid keratosis is frequently denominated in fair skinned individuals usually betwixt fifth to seventh decade and demonstrates a female predominance. Lichenoid keratosis can appear contingent to exposure to ultraviolet light and can be contemplated as a chronic inflammatory reaction of regressing benign epithelial neoplasm such as solar lentigines or a reticulated variant of seborrheic keratosis. Diverse stages of regressive lichenoid keratosis depicts diverse clinical and dermoscopic manifestations. Lichen planus-like keratosis can undergo a comprehensive and spontaneous involution [1,2].

### Clinical elucidation

Typically, lesions appear abruptly and present as a solitary macule, papule or a plaque which undergoes continuous evolution with eventual regression. A singular, asymptomatic papule commonly arises from the torso or upper extremities. An estimated 8% persons can display two and < 1% subjects demonstrate three or more lesions. Lesions vary in dimension up to 20 millimetres, display a light pink or intensely violaceous hue and generally appear on sun-inflicted cutaneous surfaces.

Subjects can demonstrate oval or elliptical, well demarcated, violaceous plaques with extraneous scaling [2,3].

Lichen planus-like keratosis can appear as a papule or plaque demonstrating a smooth or verrucous extraneous surface and hues varying from pink, violaceous, tan or brown. Preliminary lesions represent as erythematous papules which progressively turn darker or violaceous and eventually appear as hyper-pigmented or hypo-pigmented macules [4].

### Histological elucidation

On gross examination erythematous, elevated lesions with superficial scaling are demonstrated.

Tissue samples of lichen planus-like keratosis obtained by punch biopsy exhibits an inflammatory infiltrate with a band-like dissemination. Lichen planus-like keratosis characteristically exhibits a dense, lichenoid inflammatory infiltrate predominantly composed of lymphocytes, appearing at the dermo-epidermal interface which consequently undergoes obscuration. Lymphocytes are devoid of apparent atypia [3,4].

Microscopic elucidation of hyperkeratosis, parakeratosis, band-like inflammatory exudate emerging within the dermo-epidermal junction (lichenoid pattern of inflammation), necrotic keratinocytes enunciating as Civatte bodies and dissemination of dermal melanophages is observed.

Epidermal manifestations are denominated as necrotic keratinocytes within the basal layer, epidermal acanthosis, hyper-granulosis and hyperkeratosis. Parakeratosis and eosinophilic infiltration is exceptional. Atypia of the basal cell layer is absent. Minimal degrees of reactive spongiosis can ensue [5,6].

### Differential diagnosis

As lichen planus-like keratosis is a common, benign lesion, an accurate differentiation is mandated from cogent disorders. Primarily, malignant melanocytic lesions or a basal cell carcinoma requires a distinction from lichenoid keratosis. Clinical distinction of lichen planus-like keratosis is mandated from solar lentigo, seborrheic keratosis, actinic keratosis, Bowen's disease, basal cell carcinoma, squamous cell carcinoma and malignant melanoma [5,6].

Histological segregation is necessitated from lichen planus, lichenoid drug eruption, lichenoid acral lupus erythematosus, lichenoid graft-versus-host (GVH) disease, inflamed seborrheic keratosis, halo nevus and lichenoid regression of melanoma.

Segregation of lichen planus, especially mucosal lesions, requires appropriate clinical correlation.

Additionally, drug reactions, cutaneous T cell lymphoma, regressing melanocytic lesions particularly malignant melanoma and lichenoid actinic keratosis requires demarcation from lichen planus-like keratosis. Lichenoid actinic keratosis demonstrates atypical, hyperchromatic, basaloid cells particularly disseminated at the lesion perimeter, as cogitated with solar elastosis [6,7].

### Investigative assay

Dermoscopic evaluation reveals peripheral granules, a centric zone of pinkish- brown pigmentation, shimmering, disorganized, pearly articulations and rosette formations.

Foci of coarse, greyish granules, light brown amorphous areas or shimmering, alabaster articulated rosettes superimposed upon a backdrop of light brown pigmentation can be observed on dermoscopy. Aforesaid features can also indicate a melanoma *in situ*.

Dermoscopic evaluation of lichen planus-like keratosis characteristically displays coarse grey- brown granules with specific localized and diffuse granular pattern [8].

Diffuse granular pattern typically exhibits brownish-grey, reddish-brown, bluish-grey or whitish-grey coarse granules admixed with tan-coloured pigmentation.

Localized granular pattern is indicative of preliminary, regressive phase of lichen planus-like keratosis with classical granularity adjacent to the primary epidermal lesion.

Granular appearance is contemplated as a regressive feature and can be associated with adjunctive melanocytic or non melanocytic lesions. Thus, diffuse granular pattern requires a segregation in lichen planus-like keratosis from malignant lesions or malignant melanoma. An adequate cutaneous tissue sampling is recommended in ambiguous instances [1,2].

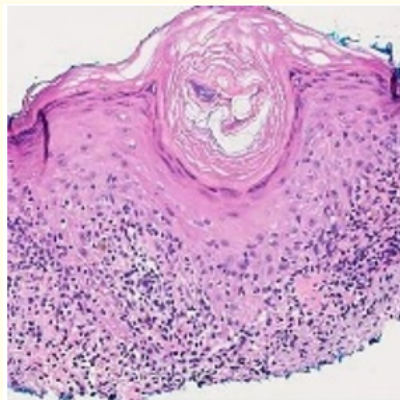
Reflectance confocal microscopy (RCM) is a cogent technique for non invasive cutaneous histological analysis which can be applied to the diagnosis of lichen planus-like keratosis. Reflectance confocal microscopy can be performed on specimens obtained by deep shave or excisional biopsy. A spinous granular layer of superimposed epidermis with typical honeycomb configuration and several whitish, ovoid articulations along with milia- like cysts are indicative of the condition. Dark-hued, depressed configurations or comedo-like apertures are noted. The lesion is devoid of pagetoid or dendritic cells [1,2].

Dermo-epidermal junction displays expansive cellular cords and bulbous cellular projections. Numerous bright, plump or spherical cells and stellate spots are enunciated within the interpapillary spaces of the superficial dermis. An absence of bright nucleated or dendritic cells in the spinous granular layer, bright dendritic cells, spindle or atypical cells within the dermo-epidermal junction and tumour cell nests within the superficial dermis is exemplified. Aforementioned findings are diagnostic of lichen planus-like keratosis.

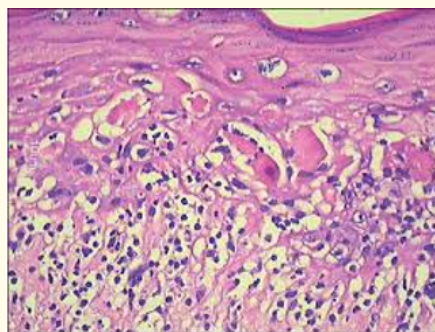
Reflectance confocal microscopy can adequately categorize lichen planus-like keratosis in around three fourths (71.4%) instances [1,2].

**Therapeutic options**

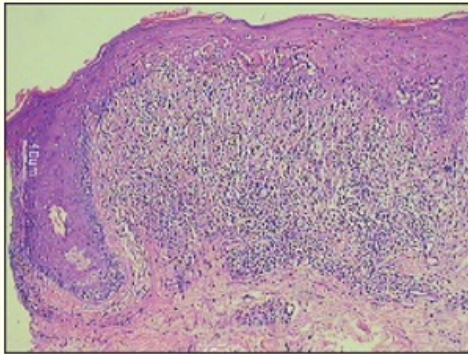
Usual treatment of lichenoid keratosis includes employment of diverse modalities such as cryotherapy, curettage, electro-surgery and/ or shave resection. Lesions can also be treated with topical steroids. Sequential monitoring exhibits a partial regression of lesions at three months and complete resolution at six months [8].



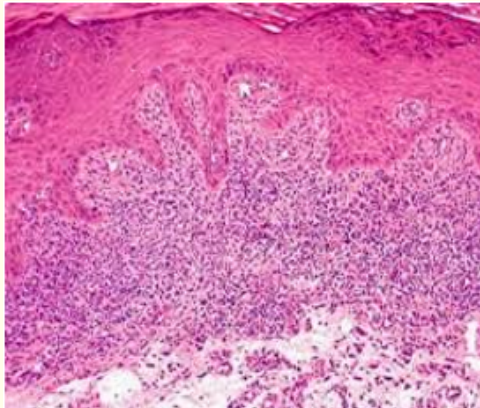
**Figure 1:** Lichenoid keratosis with band like lymphocytic infiltrate in the upper dermis and dermal-epidermal interface along with acanthosis and hyperkeratosis [9].



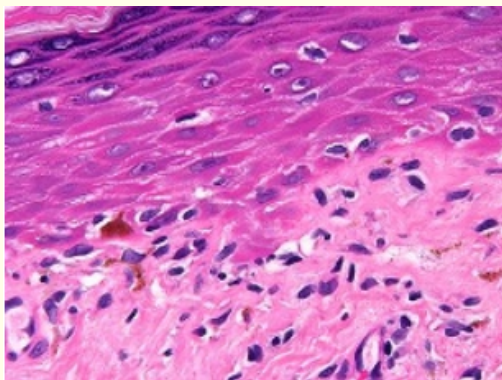
**Figure 2:** Lichenoid keratosis with junctional lymphocytic infiltrate, marked acanthosis and mild hyperkeratosis [10].



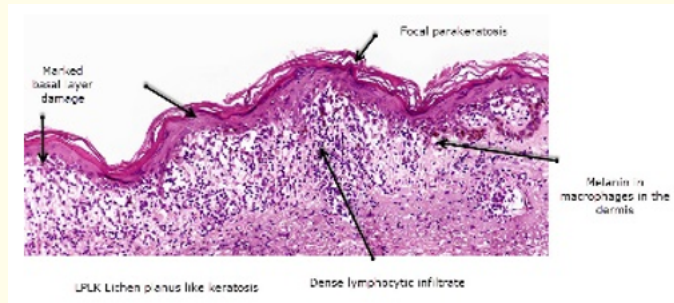
**Figure 3:** Lichenoid keratosis with dense lichenoid, lymphocytic inflammation, hypergranulosis and moderate acanthosis [10].



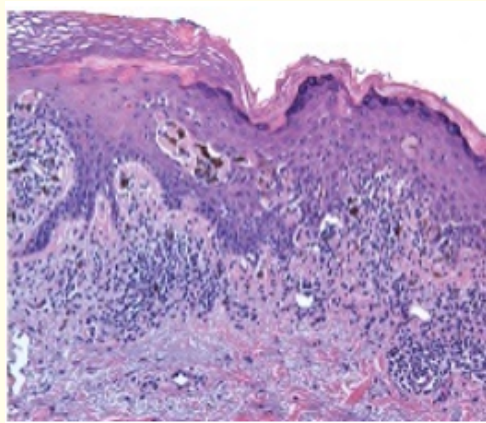
**Figure 4:** Lichenoid keratosis with intense junctional inflammatory exudate, marked acanthosis, hypergranulosis and mild hyperkeratosis [11].



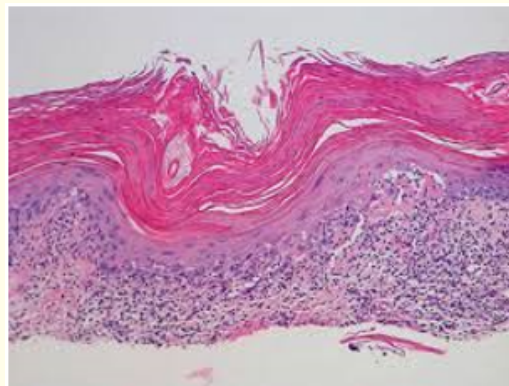
**Figure 5:** Lichenoid keratosis with necrotic, basal keratinocytes, Civatte bodies, acanthosis, hyperkeratosis and mild dermal inflammation [11].



**Figure 6:** Lichenoid keratosis with hyperkeratosis, hypergranulosis acanthosis and intense lichenoid inflammation [12].

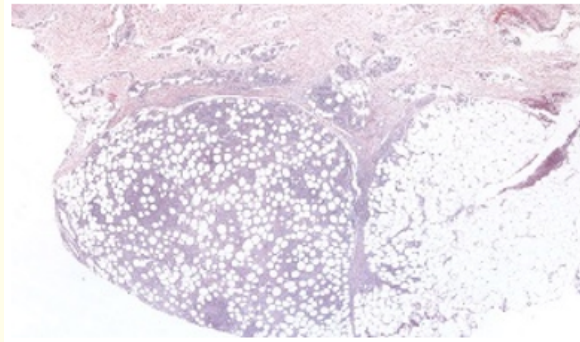


**Figure 7:** Lichenoid keratosis with dense, dermal-epidermal lymphocytic inflammatory infiltrate, prominent acanthosis, hyperkeratosis and hypergranulosis [13].

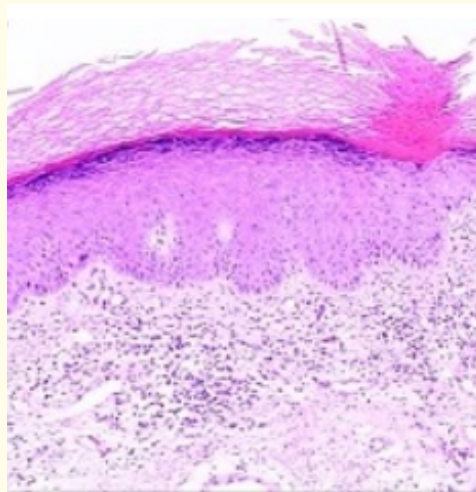


**Figure 8:** Lichenoid keratosis with intense acanthosis, hypergranulosis, hyperkeratosis, necrotic basal keratinocytes and dense lymphocytic exudation of dermal interface [14].

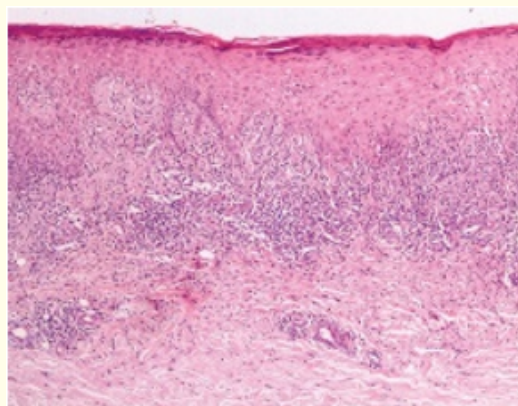




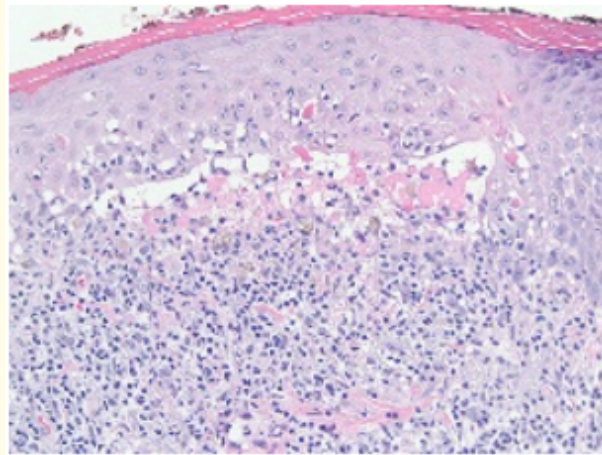
**Figure 9:** Lichenoid keratosis with epithelial islands of inflammatory exudate composed of lymphocytes with circumscribing adipose tissue [15].



**Figure 10:** Lichenoid keratosis with marked acanthosis, hyperkeratosis, necrosis of keratinocytes and an intense inflammatory exudate of lymphocytes at the dermal-epidermal junction [16].



**Figure 11:** Lichenoid keratosis with preponderant lymphocytic infiltrate at the dermal interface, necrosis of keratinocytes and mild acanthosis with hyperkeratosis [17].



**Figure 12:** Lichenoid keratosis with predominant chronic inflammation comprised of lymphocytes, Civatte bodies and moderate acanthosis with mild hyperkeratosis [18]. acanthosis with hyperkeratosis [17].

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