

Lichen Actinicus

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Lichen actinicus is a sub-type of Lichen ruber planus, which occurs predominantly in dark-complexioned young adults after prolonged exposure to sun and has a distinct photodistribution. The histological findings are as variable as the morphology of the clinical lesions. They are only partly like those of classical Lichen planus. The constant findings are marked pigmentary incontinence and the lymphocytic infiltrate in the papillary dermis. We present a patient with Lichen actinicus with two different types of lesions. We discussed the morphological and histopathologic variants of the disease.

Key words: Lichen actinicus, Lichen ruber planus, morphological types, histopathological findings.

Introduction

Lichen actinicus (LA) is a disease of young people of Oriental extraction with a distinct photodistribution. The lesions develop on the face and on the upper part of the hands. They appear usually in the spring and summer with improvement and even complete remission in winter. The histologic changes are only partly like those as classical Lichen planus (LP). They are as variable as the morphology of the clinical lesions.

Emphasizing the role of sunlight the disease has been defined as a subtype of LP called LA [1]. For a long time it was told to be confined to subtropical countries [1, 2, 3]. Eighty-three per cent of reported patients were of Oriental origin (Iraq, Egypt, North Africa, Afghanistan, Yemen, India, Turkey, Bukhara) [1]. From this point of view it was named Lichen planus subtropicus. Thirty to forty per cent of LP seen in the Middle East were of these subtype [2]. However, the original reports of this condition were clearly Italian in origin [4]. Other authors propose another name of the disease — Summertime actinic lichenoid eruption (SALE) [5]. The point that LP subtropicus only delineates a predominant and not unique geographic distribution and does not stress the role of light in the pathogenesis.

Materials and Methods

Our patient is a 32-year-old white woman. She complains of dark macules, distributed all over her face. The had appeared before one year, after sun exposure in August. She hasn't

pruritus but has been treated for 20 days with topical corticosteroid without effect. In the following winter months she has noticed an improvement as the pigmented lesions became grayish-white. After sun exposure next year the lesions became darker and larger and she also obtained violaceous papules on the dorsum of both hands. She has neither family history of skin disease nor internal disease. She doesn't take any medications. The patient followed laboratory investigations including complete blood counts, ESR, blood sugar, hepatorenal function test, HIV serology, urinalysis and immunological parameters including antinuclear antibodies, anti-Sm, anti-s DNA, anti-n DNA, anti-Ro, anti-La antibodies, C₃ and C₄ complement. Biopsies were taken from a macule on the face as well as the lichenoid papules on the hand. Direct immunofluorescence and phototesting were also performed.

Results

On physical examination it was observed that our patient has a skin phototype IV. Pigmented macules were seen all over her face — on forehead, nose and chin. Violaceous flat topped lichenoid papules arranged linear were distributed on the dorsa of the hands. The mouth, scalp and nails were normal. Routine investigations were in normal limits. Immunological parameters were negative except the antinuclear antibodies which were in titer 1:40. Minimal erythema dose was elevated in phototesting. Direct immunofluorescence was negative. Hematoxylin-eosin section from the face showed an epidermal atrophy, hyperkeratosis and smooth dermo-epidermal junction (Fig. 1). Melanin uncontinence was prominent. Lichenoid lymphocytic infiltrate could be seen in the papillary dermis. There was no deep dermal infiltrate or follicular plugging. The biopsy of the lichenoid papule from the hand revealed typical picture of so-called interface dermatitis (Fig. 2). Pseudoacanthosis of the epidermis and obliterated dermo-epidermal junction could be seen. Some colloid bodies were present in the lower epidermis. A high-hugging lymphocytic infiltrate could be seen in the upper dermis obscuring the dermo-epidermal junction.

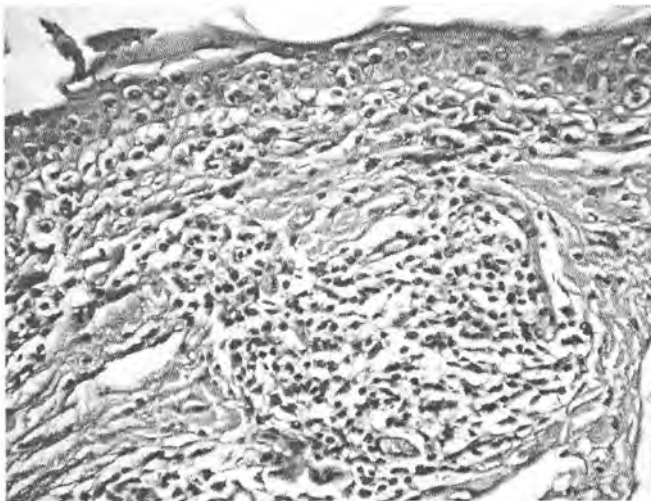


Fig. 1. Skin biopsy from lesion on the face: epidermal atrophy, hyperkeratosis, smooth dermo-epidermal junction, marked melanin uncontinence. Lichenoid lymphocytic infiltrate in papillary dermis ($\times 100$)

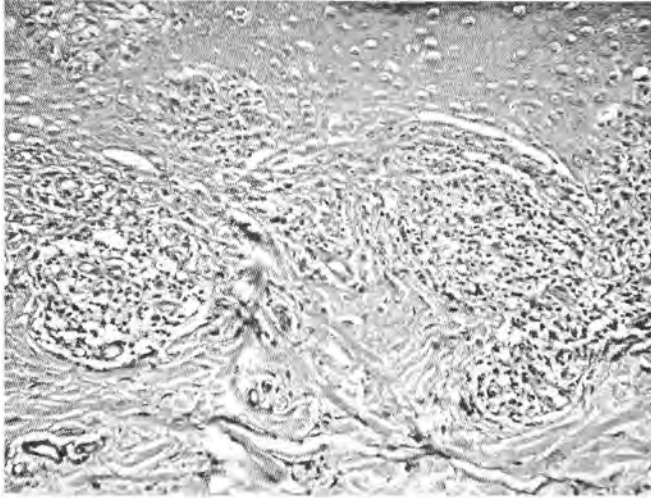


Fig. 2. Skin biopsy of the lichenoid papule from the hand: typical picture of so-called interface dermatitis (× 50)

The patient was treated with whitening lotion with kojic acid and a cream with Mexoryl XL for the lesions involving the face and with Methylprednisolon aceponat for the lesions distributed on the hands. Two months later she was slightly improved and she was recommended to use all the seasons photoprotecting cream.

Discussion

LA occurs or exacerbates after sun exposure, with recurrences in the spring and summer, and improvements or spontaneous remissions in the winter months. It affects almost exclusively the photoexposed parts of the skin, mainly face and dorsa of the hands. Morphologically, LA can be quite variable with several types of lesions:

1. Annular hyperpigmented plaques. This is the most common variety.
2. Pigmented form. This appears as chloasma-like lesions.
3. Dyschromic type. This consists of whitish pinhead papules with a tendency to coalesce.
4. Typical lichenoid papules. They are invariably present.

The histopathologic findings in LA differ according to the morphological type.

Typical lichen planus histology has been described in some of reported articles [2, 6, 7]. Some of the epidermal cell degenerated in so-called colloid bodies.

This change, which proceeded the formation of the dermal infiltrate, together with the liquefaction necrosis of the basal-cell layer, are the most characteristic features of early LP. Some colloid bodies are seen also in the biopsy specimen taken from the papules from the hand of our patient. According to K a t z e n e l l e n b o g e n [1] histologic features showed an overlap pattern between LP, lupus erythematosus, actinic dermatitis and irradiation dermatitis. In another group of patients lichenoid and non-specific inflammatory reactions were observed [8]. The only feature common to all types was marked pigmentary incontinence. Our case confirmed this finding.

It is important to differentiate LA from lupus erythematosus and polymorphous light eruption as histologic differentiation may be somewhat more difficult. Microscopically

LA shows no thickening of the basement membrane, and the infiltrate is confined to the upper part of the dermis without involvement of adnexal structures. LA differs from polymorphous light eruption by the absence of the papillary dermal edema and the deep dermal infiltrate seen on it later.

Having in mind the clinical and especially the histologic findings of LA it is more than clear that there is a close relationship between these disease and LP. More investigations will help us to understand why exactly the dark people suffer and what is the role of the light of the pathogenesis of this entity.

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