A Case of Actinic Lichen Planus

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Citation


Abstract

The condition known as actinic lichen planus (LP) is a rare variant of LP. The high incidence of the disease develops during spring and summer, its involvement mainly of the sun-exposed parts, and its occurrence mainly in tropical areas provides good evidence for the influence of sunlight. A 38-year-old man presented with asymptomatic, violaceous-colored plaques with atrophic center on the forehead. We report a case of actinic LP with a review of the literature.

INTRODUCTION

LP, while relatively common in occurrence, have a variable clinical morphologic diversity and bring a diagnostic difficulty to the physician. Actinic lichen planus (LP), an established variant of lichen planus, is reported under a variety of names: LP atrophicus annularis, LP in subtropical countries, LP subtropicus, LP tropicus, lichenoid melanodermitis, and summertime actinic lichenoid eruption. The majority of cases have come from the tropical country, however only five patients from Korea have been reported (table1). So herein we report a case of actinic lichen planus as interesting case and review of the literature.

CASE REPORT

A 38-year-old male presented with an asymptomatic atrophic change on the forehead that first appeared 2 months ago (Fig.1). The lesion was violaceous in color, had an irregular margin. He supervises the work of construction in the outdoor field for about 7 years. The patient was otherwise healthy and denied any history of infection or trauma. On laboratory examination, complete blood count and blood chemistry were within normal range. Also antinuclear antibody and other systemic lupus erythematosus profiles were all negative.

A punch biopsy was taken from the margin of lesion on the forehead to aid in the diagnosis. Low power magnification of the specimen showed hyperplasia of the epidermis with a band-like inflammatory infiltration in the upper dermis (Fig.2A). Higher magnification showed dyskeratosis and hypergranulosis of the epidermis and vacuolar degeneration of the basal layer (Fig.2B). Focal infiltration of inflammatory cells was observed in the upper dermis, the majority of which were lymphohistiocytes. There were no atypical cells or fibrosis (Fig.2C).

Based on the histological findings, a final diagnosis of LP was made. Additionally, because the lesion developed on the forehead, which is a sun exposed area, and was atrophic in appearance, it was further classified as actinic LP. The patient was instructed to apply topical steroids and sun screen on a regular basis, but he was lost to follow-up.

Figure 1

Table 1. Summary of previously reported actinic lichen planus cases in Korea in the literature.
Figure 2
Fig. 1. A few, grouped, violaceous atrophic change on the forehead (A) and a magnified view (B).

Figure 3
Fig. 2A. Hyperplasia of the epidermis with a band-like inflammatory infiltrate in the upper dermis was seen (H & E stain, x40).

Figure 4
2B. Hypergranulosis and dyskeratosis of the epidermis were observed. Also vacuolar degeneration of the basal layer in the epidermis was seen. (H & E stain, x200).

Figure 5
2C. Focal band-like infiltrations of inflammatory cells were observed in the dermis, the majority of which were lymphohistiocytes. (H & E stain, x200).
DISCUSSION

LP is a disease with a diverse clinical presentation. It can be classified by its morphological characteristics, site of occurrence, or cause. Actinic LP is a photosensitive variant of LP that occurs primarily on sun exposed areas. Four morphologic patterns have been clinically described in the literature. The atrophic type is the most common form and is accompanied by hyperpigmentation. The second type, which is the dyschromic type, shows small, white angular papules that coalesce into plaques on the neck and dorsa of the hands. The classic plaque-like form presents as violaceous papules, and the pigmented form can be seen as melasma-like patches on the face and neck. Our case might be compatible to the atrophic form according to clinical manifestations of the lesion.

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The patient with actinic LP was mostly young, nine of 16 cases were younger than 30 years of age and the mean age was 14 years, but there was no sex predilection. The lesion was usually asymptomatic and appeared mostly during spring and summer. In this case, the lesion developed in summer and the patient was relatively young. The favored sites were sun-exposed areas such as the face and the dorsal aspect of the forearms. Unlike classic LP, pruritus, the Koebner phenomenon, and mucous membrane involvement are not commonly seen in all types of actinic LP.

Clinically, actinic LP lesions resemble the lesions found in discoid lupus erythematosus (DLE), melasma, morphea, LP pigmentosus and lichenoid drug eruption. So it should be differentiated by histopathologic and immunofluorescence (IF) findings. First, in DLE, the liquefaction, degeneration or thickening of basal layer were more prominent and IF finding or autoantibody in a blood test could be conclusive factor. Second, in melasma, melanin synthesis in epidermal layer and the number of melanocytes were increased. Third, in morphea, epidermal change is not prominent and hyalinized dermis or decreased adnexal structure is seen. However, the pathologic findings with LP pigmentosus were very similar to actinic LP, the only point of difference is the incontinence of pigment and melanophages in upper dermis. So, clinical manifestations such as the site of lesion, age or the history of sun exposure should be examined for final diagnosis. Finally, the patient with lichenoid drug eruption should have a history of medication.

The term lichenoid tissue reaction and interface dermatitis would be used synonymously in the review of literature. And lichenoid tissue reaction or interface dermatitis skin disease could be subdivided into two groups, those that display a high-density inflammatory infiltrate and a low-density infiltrate. Clinical examples of cell-rich interface dermatitis include LP and its variant, lichenoid drug reactions, lichen nitidus, lichen striatus, and some forms of autoimmune connective tissue disease such as DLE. Another example of a cell-poor interface dermatitis include virus or drug induced morbilliform exanthems, other forms of autoimmune connective tissue diseases, acute graft-versus-host skin disease and erythema multiforme. Our case might coincides with cell-rich interface dermatitis. Additionally, lichen nitidus and lichen striatus could be excluded by the absence of the findings such as focal ball shape infiltrate in papillary dermis or infiltrate around sweat gland. The histopathologic findings of actinic LP also show similarity to classic LP, such as hyperkeratosis, wedge-shaped hypergranulosis, necrotic keratinocytes in epidermis and a band-like lymphocytic infiltration in dermis. However, atrophic change might be more prominent.

The cause of actinic LP is still unknown but sunlight might be the major precipitating factor. Van der Shroeff et al., induced actinic LP by using repeated doses of UVB radiation on the back. So the sunscreen is necessary for treatment. Several modalities such as topical corticosteroid, intralesional corticosteroid injection, grenz ray or antimalarial agent could be applied for the treatment of actinic LP. Recently topical 0.1% pimecrolimus cream have been reported to be successful in a case of actinic LP.

In conclusion, LP, while relatively common in occurrence, can sometimes have a variable clinical presentation, posing a
diagnostic challenge to the dermatologist. The actinic LP was an example of such variants, and should be kept in mind when dealing with lesions of a similar nature.

References

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