HYPERKERATOTIC ORAL LICHEN PLANUS ROLE OF VITAMIN A

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Chronic tobacco chewing and smoking have been implicated as precipitating factors for the development of oral lichen planus (OLP). We here report a case of chronic cigarette smoker who developed progressive OLP plaque, without any other clinical pattern of OLP and skin involvement, which did not respond to conventional corticosteroid treatment and dapsone, but responded when vitamin A was added.

Key Words : Hypertrophic oral lichen planus, Smoking, Vitamin A

Introduction

Mucous membrane (m.m) involvement in lichen planus is common, occurring in 30 - 70% of cases. In 15 - 20% it may be the only manifestation of the disease. OLP may be found in any location of the mouth but favoured sites are the buccal mucosa, the tongue and gingiva, whereas palatal lesions are uncommon. OLP may be divided into 6 clinical patterns. [1] Reticular OLP is the commonest form and predominantly affects the buccal mucosa, appearing as a network of white or gray threads (Wickham's striae), interspersed with papules or rings. On the tongue the lesions are in the form of fixed, white plaques often slightly depressed below the surrounding mucosa. Elevated plaques over the mucosal surface usually have a grainy, coarse texture and reticular lesions may be seen surrounding the periphery of the plaque.[2] Local and systemic steroids remain the mainstay of treatment. Dapsone has been tried for the treatment of OLP by several authors.[3] Topical or systemic vitamin A can be given alone or in combination with corticosteroids.

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Case Report

A 44-year-old man presented with a 4 cm x 3 cm whitish plaque with thick velvety surface and irregular spreading border on right side of mouth (Fig.1) and a similar but smaller plaque of 1 x $^1/_2$ cm on the left side. It started as a fissure in the right commissure and extended inward towards the buccal mucosa and outward towards the labial mucosa and took the present position. Lesion on the left side started $2^1/_2$ years later.Rest of the mucosa was normal and no submucosal fibrosis or atrophy was felt. There was no skin involvement. Patient used to smoke 2 packets of cigarette daily for the last 15 years without

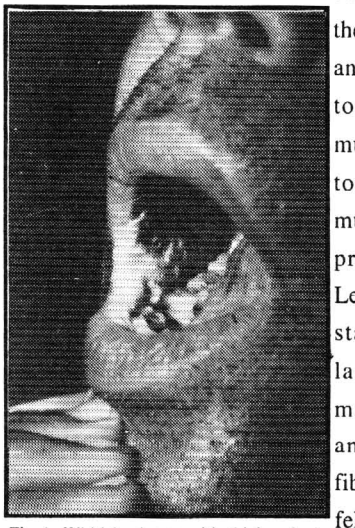

Fig 1. Whitish plaque with thick velvety surface and irregular spreading border.

tobacco chewing. Oral lesions were not associated with hypertension, diabetes mellitus, liver disease or any other autoimmune disease. Direct smear from the plaque was negative for candida. All routine investigations were normal. Biopsy from the lesion showed hyperkeratosis, papillomatosis, acanthosis, liquefaction degeneration of the basal layer and chronic inflammatory infiltrate in the subepithelial region in a band -like fashion suggesting the diagnosis of hypertrophic lichen planus which is a rare presentation in oral mucosa. Patient was advised to stop smoking and was put on tab dapsone 150mg daily orally with hamycin 1 vial mixed with 4 soluble tablets of betnesol for local application for 1 month but he showed no response. Then oral prednisolone 20mg was added. Even then no improvement was noted after $1^1/_2$ months. Vitamin A, 3 tablets (50000 IU each) daily was added along with the previous treatment. After 1 month he showed improvement in the form of regression in the size and thickness of the lesion.

Discussion

Pindborg et al conducted a survey among Indian villagers in Kerala[4] and found that tobacco chewing and smoking were related with the occurrence of OLP. Higher prevalence of OLP was found among tobacco chewers than in smokers, the number of patients with involvement of the right buccal mucosa were more than those with involvement of left side and location of OLP was in the posterior buccal mucosa because they used to keep tobacco in the posterior buccal mucosa and gum margin. But in our patient anterior buccal mucosa, anterior commissure and portion of labial mucosa of both upper and lower lips were involved . Several different forms of OLP may be seen in the same mouth. However our patient had exclusively elevated plaque type of lesions. The plaque type of OLP was significantly more prevalent among smokers than among non-smokers and tobacco chewing, and especially smoking may cause a superimposed leucoplakia and / or transformation of other types of OLP to plaque type, which in turn if untreated can change into squamous cell carcinoma.[5] Our patient did not respond to corticosteroids and dapsone alone but responded well when vitamin A was added. Same observation has been documented by Gunther who treated 17 patients of OLP with 30mg of vitamin A daily for 5-7 weeks.[6] Itin used 30mg acitretin with 15mg oral prednisolone daily in a patient having isolated LP of lip and noted complete remission after 10 weeks.[1] We are reporting this case because of rarity of isolated (hyperkeratotic) elevated plaque type of OLP and rewarding role of vitamin A.

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