# Oral lichenoid reactions: A clinico-pathological study

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#### **ABSTRACT**

Eleven hypertensive and nine diabetic patients under treatment with appropriate therapy show oral mucosal lesions mimic lichen planus. The clinical and histological features of the lesions were studied. Stress was made on the type and distribution of the inflammatory infiltrate seen in the histologic section of the lesions. It was found that the infiltrates show features differ from the infiltrate demonstrated in conventional lichen planus.

Key Words: Drug reaction, lichen planus.

الخلاصة

أحد عشر مريضاً يعانون من مرض ارتفاع ضغط الدم وتسعة يعانون من داء السكري وهم تحت العلاج المناسب لكل منهم، ظهرت عندهم أفات في الغشاء المخاطي المبطن للتجويف الفموي شبيهة للحزاز المنبسط. تم دراسة المواصفات السريرية والنسيجية وكان التركيز على نوع وانتشار الخلايا الالتهابية على الشرائح النسيجية لهذه الأفات. لقد تبين أن انتشار الخلايا الالتهابية أظهر مواصفات تختلف عن انتشار الخلايا الالتهابية للحز از المنبسط.

## INTRODUCTION

Oral lichen planus may occur in up to (2%) of a normal population and there may be associated skin involvement (1). Oral lichen planus is characterized clinically by the presence of white areas on the oral mucosa, which appear in reticular, papular, atrophic, plaque-like, erosive or bullous forms (2). The clinical diagnosis is often confirmed histologically by the presence of hyperortho- or hyperpara- keratosis, a band-like subepithelial infiltration of lymphocyte and liquifactive degeneration in the basal cell layer (3). Oral lichen planus like lesions have been associated with systemic diseases, such as diabetes, hypertension and immunologic disorders (4, 5). Several drugs have been implicated in causing lichenoid reaction of the oral mucosa being clinically and histologically identical to lichen planus. The most commonly reported drugs are methyldopa (6) and beta-blockers (7). The lesion of lichen planus most commonly affects the buccal mucosa and lateral border of the tongue, often bilaterally. The ætiology of this condition remains unknown, but dermal and oral lesions, which resemble in appearance to lichen planus occur in association with ingestion of certain drugs. Withdrawal of the suspected drug should produce reduction of the lesions within a few weeks (8).

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Recently, an immunopathogenesis has been proposed in the ætiology of lichen planus based on the findings of T-lymphocyte band as subepithelial infiltrate. In this study, diagnosed cases of lichenoid reaction were retrospectively investigated to find out histologically if there is similarity in the type of the cellular infiltrate and its distribution in the subepithelial area compared to those seen in conventional lichen planus.

# MATERIALS AND METHODS

Twenty patients were admitted to the Oral Medicine Clinic, College of Dentistry, University of Mosul during a period of (4) years with oral mucosal lesions mimic various clinical types of oral lichen planus. The files of the patients are retrieved from the Oral Pathology Unit. The materials consist of (12) females and (8) males aged between (27) to (64) years of mean age (50) years. The patients having chronic systemic diseases under treatment. Eleven were hypertensive and (9) were diabetic. The oral lesions involve buccal mucosa bilaterally in all patients. In addition, the lesions extended to the tongue in (7) patients, alveolar mucosa in (5) patients and lip mucosa in (3) patients. Under local anesthesia biopsy was taken from the lesions and a routine hæmatoxyline and eosin stained histologic sections were prepared. The sections were reviewed and stress was made on the type and distribution of the inflammatory infiltrate. The obtained results were reported and discussed.

#### RESULTS

Following analysis of the clinical and histologic forms of the patients involved in the study, it was found that reticular lichen planus like lesions occurred in (13) patients, crosive type in (5) and papular in (2) patients. The (5) with erosive type are females, whereas patients with papular type are males. The reticular type occurred in (7) females and (6) males. The histopathology of all lesions demonstrate lymphocyte and macrophage cells in the inflammatory infiltrate. Plasma cells are recognized in (15) cases, whereas mast cells are observed in (4) cases, in (3) of which eosinophils are seen as well. Only in one case the inflammatory infiltrate was limited to lymphocyte and macrophages. In the (15) cases which show plasma cells in the infiltrate no mast or eosinophil cells are observed.

## DISCUSSION

On the clinical bases there is no way to separate between lichen planus and lichenoid reaction of the oral mucosa. These lesions are totally similar in their appearance. However, in the histology although some similar features sharing both lesions, some variations in the inflammatory infiltrate at subepithelial areas are observed. Classically, the inflammatory infiltrate in lichen planus located immediately subepithelially, which does not infiltrate deep in the lamina propria. This infiltrate shows cells of similar type said to be T-lymphocytes (3). On the other hand, the observation made in the majority of the cases in this study show some variations in the inflammatory infiltrate compared to those seen in lichen planus lesions. In

lichenoid reaction the infiltrate does not confined only subepithelially but spread deeply in the lamina propria as well. Moreover, the cellular types in the infiltrate are variable. In addition to lymphocytes, macrophages and plasma cells are seen. In some lesions mast cells and eosinophils are also demonstrated. From these observations one can suggest that the pathologic process in lichenoid reaction is differ from that of lichen planus.

The mechanism involved in initiation of lichen planus is said to be cellular hypersensitivity since T-lymphocytes are predominated in the infiltrate <sup>(3)</sup>. Whereas in lichenoid reactive lesions, although allergic reaction is played in the area but the mechanism is differ. In the cases where plasma cells and macrophages are seen, this may indicate that the allergic reaction is of antibody antigen of type II hypersensitivity, since plasma cells are antibody producers. In other cases in which mast cells and eosinophils are seen the mechanism is probably of type I hypersensitivity <sup>(9)</sup>. Therefore, it can be concluded that the pathogenesis of both oral lichen planus and lichenoid reactions are of allergic of various types. Farther advanced studies involving immunocytochemistry to support the above suggestions are recommended.

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