Research Article

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A Quantitative Evaluation of Epithelium and Inflammatory Infiltrate of Lichen Planus and Lichenoid Reactions

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ABSTRACT

Background: Lichen Planus and Lichenoid reaction are two different conditions that pose a diagnostic dilemma. Both the diseases are very similar in histology. Being so similar, but still, they have different treatment and have way different prognosis.

Materials and methods: Patients who were suffering from lichenoid reaction (n=10) and oral lichen planus(n=30) and in the age group of 40 to 70 years were selected for the study. Sections were taken for biopsy and stained with hematoxylin and eosin. The thickness of the epithelium and the thickness of the subepithelial inflammatory band was measured at five different places in each of the sections.

Results: The lichen planus group showed a negative correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate. However, in the Lichenoid reaction, there was a positive correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate.

Conclusion: Inflammatory infiltrates more heterogeneous and diffuse. The epithelial thickness was directly proportional to inflammatory infiltrate.

Keywords: Epithelium, inflammation, lichen planus, lichenoid reactions.

INTRODUCTION

Oral lichenoid reactions represent a common endpoint in response to extrinsic agents (drugs, allergens), altered self-antigens, or superantigens.[1] Oral lichen planus (OLP) is a chronic inflammatory disorder of unknown aetiology. Patients experience mild to severe oral pain, which may impede oral intake and compromise nutritional status. The varied morphologies of OLP mandate consideration of a broad differential diagnosis. A complete mucocutaneous examination and extensive review of systems should be performed on all OLP patients in order to identify additional sites of involvement, many of which cause severe morbidity.[2]

Etiopathogenesis of LP is generally dependent upon T cell-mediated immunologic interaction with basal epithelial cells considered as foreign because of altered surface antigenicity.[3] This biochemical alteration in combination with genetic factors appears to make the persons susceptible to Lichenoid lesions. Such a reaction can happen in a plethora of different circumstances.

One of the potential complications of oral lichen planus (OLP) is its malignant transformation – this being a phenomenon that remains subject to important controversy.[4–8] The literature contains an increasing number of series on the potential malignization of OLP, though some authors question

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the existing evidence.[9] In this context, Krutchkoff et al.,[10] in a review of 223 cases reported between 1950 and 1976, found only 15 (7%) to satisfy truly strict criteria of malignant transformation overlying preceding OLP lesions. Posteriorly, van der Meij et al.[11] reviewed the literature between 1977 and 1998 in relation to the malignant transformation of OLP. Of the 98 cases documented, the authors found 33 (34%) to meet the malignization criteria established by Krutchkoff et al.[10] Since 1910 when the first case of squamous cell carcinoma (SCC) in an OLP patient was reported, many series have been published indicating a malignization rate of 0–12.5%.[12–17]

The aim of the study is a) Separating both these entities histopathologically with the use of micrometry. B) to find the role of subepithelial chronic inflammatory infiltrate on overlying epithelium.

MATERIALS AND METHODS

Forty Patients were selected from the outpatient department of our institute. All the patients were explained in details about the study, and written consent was obtained. All the ethical clearances were obtained from the ethical review board for the institute (DWS/OMP/2019/o/23). Patients who were suffering from lichenoid reaction (n=10) and oral lichen planus(n=30) and in the age group of 40 to 70 years were selected for the study. Patients with a recent history of any surgery and/or on antibiotic therapy were excluded from the study.

Sections were taken for biopsy and stained with hematoxylin and eosin. The thickness of the epithelium and the thickness of the subepithelial inflammatory band was measured at five different places in each of the sections. Extreme precautions were taken to include the region to measure should be free from the tissue artefacts. Atrophic and hyperplastic areas and the regions between the two were chosen. Measurements were recorded using an eyepiece graticule.

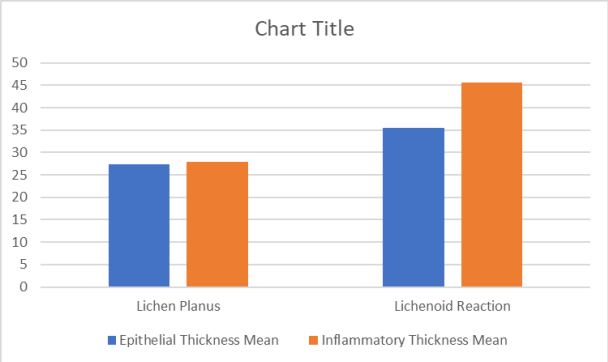
The epithelium was measured from the superficial keratotic areas to the basal cell layer. The mean of these areas was calculated for each lesion. Statistical analysis was done by applying Karl Pearson's correlation coefficient in SPSS software (V21 for windows, NY, USA).

RESULTS

The mean thickness of the epithelial band in lichen planus was 27.3 micron and 35.5 in Lichenoid reaction lesion. At the same time, the mean thickness of the inflammatory band in lichen planus was 27.9 micron and 45.6 in Lichenoid reaction Table 1, Figure 1). Lichen planus group showed a negative correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate. However, in the Lichenoid reaction, there was a positive correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate.

Table 1: Showing quantitative thickness of the epithelium and subepithelial inflammatory band.

	Lichen Planus	Lichenoid Reaction
Epithelial Thickness Mean	27.3	35.5
Inflammatory Thickness Mean	27.9	45.6



Graph 1: Graphic representation of thickness of the epithelium and subepithelial inflammatory band.

DISCUSSION

There was the presence of many features that were common in both the group. Initially, in 1978 WHO gave guidelines to diagnose the oral lichen planus. However, it was modified in2003 by the same institution. The present criteria that were followed in the study are mentioned below.[18]

Modified WHO diagnostic criteria of OLP and OLL (2003)

Clinical criteria

(1) Presence of bilateral, more or less symmetrical lesions

(2) Presence of a lacelike network of slightly raised grey-white lines (reticular pattern)

(3) Erosive, atrophic, bullous and plaque-type lesions are accepted only as a subtype in the presence of reticular lesions elsewhere in the oral mucosa. In all other lesions that resemble OLP but do not complete the aforementioned criteria, the term "clinically compatible with" should be used

Histopathologic criteria

(1) Presence of a well-defined band like zone of cellular infiltration that is confined to the superficial part of the connective tissue, consisting mainly of lymphocytes

(2) Signs of liquefaction degeneration in the basal cell layer

(3) Absence of epithelial dysplasia

**When the histopathologic features are less obvious, the term "histopathologically compatible with" should be used

**Final diagnosis criteria OLP or OLL (OLP: oral lichen planus, OLL: oral lichenoid lesion)

• OLP – a diagnosis of OLP requires fulfilment of both clinical and histopathologic criteria

• OLL – the term OLL will be used under the following conditions

(1) Clinically typical of OLP but histopathologically only compatible with OLP

(2) Histopathologically typical of OLP but clinically only compatible with OLP

(3) Clinically compatible with OLP and histopathologically compatible with OLP

Diagnosis of oral lichen planus should fulfil both clinical and histopathologic criteria. The term Oral lichenoid lesions7 should be used otherwise. Lichenoid reaction may clinically and histologically mimic Lichen planus.[18] Consideration of case history plus knowledge of pertinent clinical factors (biopsy site, distribution & specific appearance of lesions) is valuable adjuncts that must be applied in order to better evaluate lichenoid lesions histologically.[9]

It has been long recognized that there is an association of chronic inflammation with a variety of epithelial malignancies. The quality of chronic inflammatory cell infiltrates differ in different types of lesions. Hyperkeratotic lesions without acanthosis showed a significantly smaller number of mononuclear.[19] Chronic inflammatory process provides a microenvironment that is base on cytokines. This promotes cell survival, differentiation and proliferation hence contributing to cancer initiation.[19] A study by Mollaoglu showed that inflammatory factors are related to the cancer initiation progression and invasion are often expressed by the oral lichen planus.

In our study Lichen planus group showed a negative correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate. However, in the Lichenoid reaction, there was a positive correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate as similar studies are not done earlier, so it's very difficult to compare the results of this study to others. Etiopathogenesis of Lichen Planus shows that degeneration of the basal cell layer of oral mucosa occurs due to the immune system. This degeneration of oral mucosa is related to a cell-mediated immune process involving Langerhans cells, T Lymphocytes and macrophages. Hence, basal cell degeneration can be responsible for the reduction of epithelial thickness in the case of lichen planus.[9] Interaction between lymphocytes and keratinocytes are important determinants in the effector phase of the lichenoid lesion.

CONCLUSION

In Lichenoid reaction thickness of epithelium was more. Inflammatory infiltrates more heterogeneous and diffuse, and epithelial thickness was directly proportional to inflammatory infiltrate. So this finding may be considered as an additional feature in differentiating these two entities.

CONFLICTS OF INTEREST

The authors declare they have no potential conflict of interests regarding this article.

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