# A QUANTITATIVE EVALUATION OF EPITHELIUM AND INFLAMMATORY INFILTRATE OF LICHEN PLANUS AND LICHENOID REACTIONS

Usha Balan[1] Nitin Gonsalves[2] Maji Jose[3]

*Departments of Oral & Maxillofacial Pathology, [1]KMCT Dental College, Calicut, Kerala, India*
*[2]AJ Shetty Dental College, Mangalore, [3]Yenepoya Dental College, Mangalore, India*

**Corresponding Author:** Usha Balan, Reader, Dept. of Oral & Maxillofacial Pathology, KMCT Dental College, Mampetta, Calicut. Pin-673602, *Mob:* 9746590178, *E-mail:* tkmithran@yahoo.com

**Abstract**

Lichen planus and Lichenoid reaction are two different entities that pose a diagnostic dilemma. Objectives: Lichen planus and Lichenoid reactions are clinically and histopathologically very similar but have different treatment and prognosis. This particular study is aimed at differentiating both these entities histopathologically with the use of micrometry and to find the role of subepithelial chronic inflammatory infiltrate on overlying epithelium with regard to its thickness.

Methods: In the present study, Lichen Planus (n=30) and Lichenoid reactions (n=10) were studied. Using eyepiece graticule, epithelial thickness and subepithelial inflammatory band thickness were measured in these lesions.

Results: Positive correlation was seen between epithelial and inflammatory band thickness in cases of lichenoid reactions and a negative correlation in cases of lichen planus. Conclusion: The study suggests that the nature and thickness of subepithelial infiltrate has an influence on the overlying epithelium in Lichen Planus and Lichenoid reaction.

Keywords: Lichen planus, Lichenoid reaction, Inflammatory infiltrate

## Introduction

Genetic damage is the match that lights the fire of cancer; some types of inflammation may provide the fuel that feeds the flames. So a study of chronic inflammation is essential to understand the prognosis of any lesion.[1] Lichen Planus is a self-limited chronic inflammatory reaction that normally affects middle-aged adults involving skin, mucous membrane, hair and nails.[2] Lichenoid reaction is a lesion, which resembles erosive lichen planus mainly on buccal mucosa associated with ingestion of some categories of medications and presence of other exogenous materials in the oral cavity.[3]

Etiopathogenesis of Lichen planus is considered to be a T cell mediated immunologic interaction with basal epithelial cells perceived as foreign because of altered surface antigenecity.[3]

Biochemical epithelial alteration coupled with genetic factors related to major histocompatibility profiles appear to render persons susceptible to Lichenoid lesions. Such

reactions can occur under a host of diverse circumstances.[3]

**Premalignant nature of lichen planus?**

Point that is relatively uncontroversial is that if carcinoma arises in lichen planus the likelihood of it doing so is most certainly small.[3] Previously published malignant transformations of oral lichen planus occurred in patients with a known history of exposure to carcinogens. It was concluded that atrophic or erosive oral lichen planus have more tendency towards malignant transformation, may be due to increased vulnerability of the epithelium to the effect of carcinogens. Diagnostic errors like mistaken or poorly documented original diagnosis may constitute the strongest argument against a precancerous potential of lichen planus[4]. Many authors have concluded the risk of malignant transformation is more for oral lichen planus compared to cutaneous counterpart. WHO states that malignant transformation has been observed in as many as 2-3% of oral lichen planus patients in several studies.[4,5]

Lichenoid reaction occurs in association with use of medication, dental restorative materials and often resolves without recurrence following discontinuation of identified medication or change of restorative materials. Attempt to differentiate between the two conditions histologically may be difficult as in common with clinical findings those features considered to be characteristic of an lichenoid drug eruption can also be identified in some cases of idiopathic oral lichen planus.[6]

Lichen planus and Lichenoid reactions are clinically and histopathologically very similar but have different treatment and prognosis.

This particular study is aimed at

1) Differentiating both these entities histopathologically with the use of a micrometry.

2) To find the role of subepithelial chronic inflammatory infiltrate on overlying epithelium with regard to its thickness.

**Material and Methods**

Diagnosed cases of oral lichen planus ( n=30) and lichenoid reactions (n=10) were selected. Sections were taken and stained with hematoxylin and eosin.

Epithelial thickness and subepithelial inflammatory band thickness were measured at 5 different representative regions in each section. Regions chosen were devoid of any tissue artifacts. Atropic and hyperplastic areas together with regions intermediate between the two were chosen. Thickness was measured using eyepiece graticule. Epithelium was measured from the superficial keratotic area to the basal layer and thickness of inflammatory band in the subepithelial connective tissue was measured. Mean of these areas were calculated for each lesion. Statistical analysis was done using Karl Pearsons correlation coefficient.

**Results**

The mean and range of the epithelial and subepithelial inflammatory band thickness is shown in the Table I. In lichen planus, there exists a negative correlation between epithelial thickness and thickness of subepithelial inflammatory cell infiltrate. [Fig 1] In lichenoid reaction, there exists a positive correlation between epithelial thickness and thickness of the subepithelial inflammatory cell infiltrate. [Fig 2]

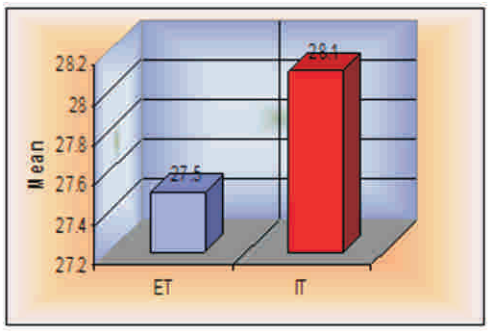


**Fig. 1:** *Graph showing thickness of Epithelium & Inflammatory infiltrate in Lichen Planus. ET: Epithelial thickness IT: Inflammatory band thickness*

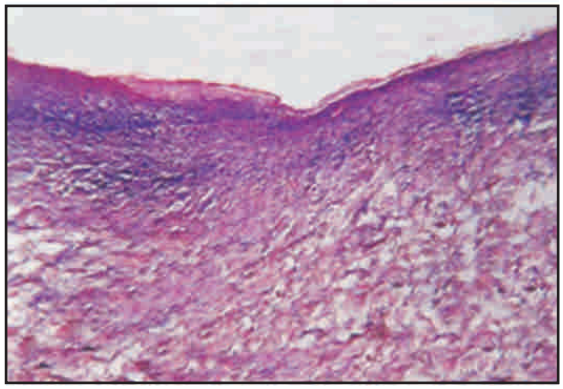

Fig. 4: *Photomicrograph H&E, 10X Lichen Planus*

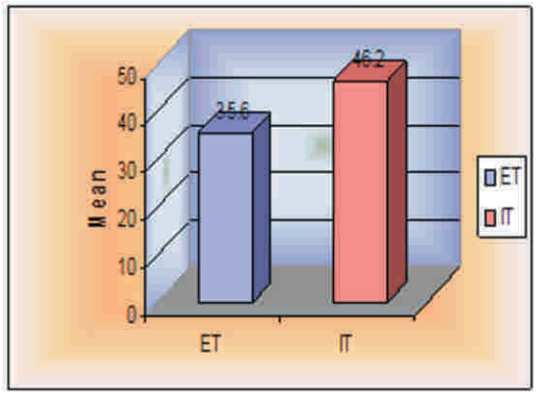


**Fig. 2:** *Graph showing thickness of Epithelium & Inflammatory infiltrate in Lichenoid Reaction. ET: Epithelial thickness IT: Inflammatory band thickness*

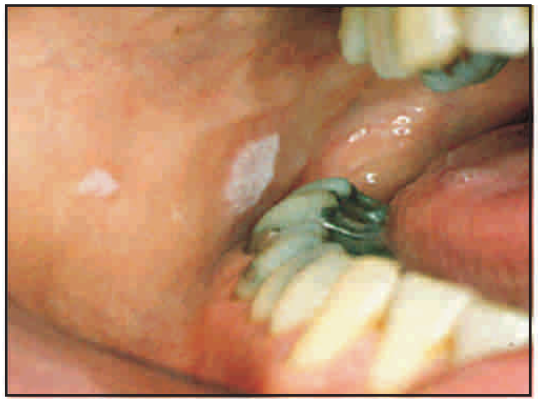

Fig. 5: *Clinical photograph of Lichenoid Reaction*

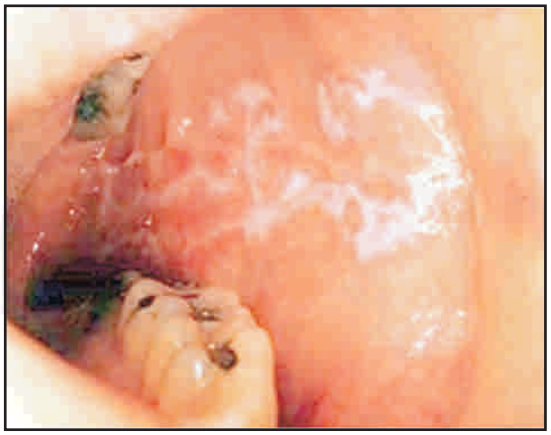

Fig. 3: *Clinical photograph of Lichen Planus*

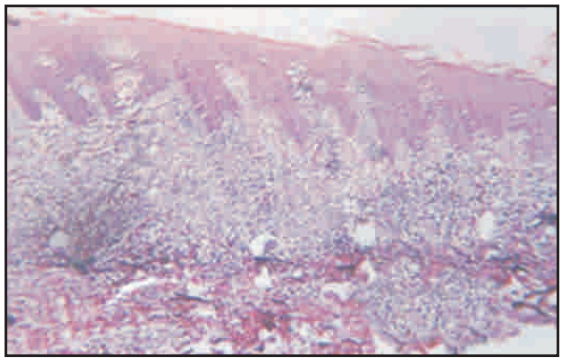

Fig. 6: *Photomicrograph H&E, 10X Lichenoid Reaction*

| Lesion | Lichen Planus (n-30) | Lichenoid Reaction (n-10) |
|---|---|---|
| *Epithelial thickness* | | |
| Range | 15 - 48.4 | 20 – 49 |
| Mean | 27.5 | 35.6 |
| *Inflammatory band thickness* | | |
| Range | 16 - 46 | 28 – 71 |
| Mean | 28.1 | 46.2 |

Table I: *Table showing Quantitative thickness of epithelium and subepithelial inflammatory band in Lichen Planus and Lichenoid lesions.*

## Discussion

There is a good deal of overlap of both clinical and histopathological features in many lichenoid processes.
Modified WHO criteria of oral lichen planus and oral lichenoid lesions.[6]

**Clinical criteria:**

- Bilateral more or less symmetrical lesion with a lacy white keratotic plaque and striae on an erythematous background. [Fig 3]
- Erosive, atrophic, bullous and plaque type lesions accepted as a subtype in presence of reticular lesions elsewhere in the oral mucosa.
- All other lesions that resemble oral lichen planus but did not complete the above criteria should be termed as clinically compatible.

**Histopathologic criteria**

- Presence of well-defined band like zone of cellular infiltration confined to superficial part of connective tissue consisting predominantly of lymphocytes. [Fig 4]
- Sign of liquefaction degeneration in basal cell layer.
- Absence of epithelial dysplasia.
- Less obvious histopathologic features should be termed as histopat hologically compatible with.

Diagnosis of oral lichen planus should fulfill both clinical and histopathologic criteria. The term Oral lichenoid lesions should be used otherwise.[7] Lichenoid reaction may clinically and histoligocally mimic Lichen planus. [Fig 5,6]

Consideration of case history plus knowledge of pertinent clinical factors (biopsy site, distribution & specific appearance of lesions) are valuable adjuncts that must be applied in order to better evaluate lichenoid lesions histologically.[8]

Association of chronic inflammation with a variety of epithelial malignancies have been recognized for centuries. Chronic inflammatory cell infiltrate showed significant differences in various lesions. Hyperkeratotic lesions without acanthosis showed a significantly smaller number of mononuclear cells. Increase in number of mononuclear cells with development of acanthosis and a progressive increase in epithelial atypia and carcinoma in situ.[9]

Chronic inflammatory process provides a cytokine based microenvironment which influences cell survival, growth, differentiation, proliferation and movement hence contributing to cancer initiation, progression, invasion and metastasis. Inflammatory factors known to be related with cancer initiation progression and invasion are also expressed by oral lichen planus related chronic inflammatory infiltrate thus contributing to oral lichen planus malignant transformation.[10]

In our study a negative correlation was present between epithelial thickness and subepithelial inflammatory band thickness in lichen planus and a positive correlation was found between epithelial thickness and subepithelial inflammatory band thickness in lichenoid reaction. As similar studies are not reported in earlier literature we are unable to compare our results with previous studies.
Etiopathogenesis of lichen planus suggests that immunologically induced degeneration of basal cell layer of oral mucosa related to a

cell mediated immune process involving Langerhan cell, T lympocytes and macrophages. Thus basal cell degeneration may be responsible for reduction of epithelial thickness in case of lichen planus.[10]

Etiopathogenesis of Lichenoid reaction suggests an immunological response to some species of antigenic modification involving adjacent epithelium. These modifications can range widely in character.[8] Results obtained in our study suggests there may be an attempt to repair shown by basal cells which would result in the increase of epithelium which is directly related to the amount of inflammatory infiltrate.

Interaction between lymphocytes and keratinocytes are important determinants in effector phase of lichenoid lesions. Balance between proliferating and opposing influences of various cytokines have overall effect on keratinocytes thus influencing epithelial atrophy and hyper proliferation, which also reflects peculiar local influences.
Our study reveals that inflammatory infiltrate is indirectly proportional to epithelial thickness in lichen planus. This observation may assist in differentiating these two lesions. Further studies are required to be conducted including more number of cases to confirm our observation.

## Conclusion

Lichen Planus and Lichenoid reaction differ in amount of inflammatory infiltrate. In Lichen Planus average thickness of epithelium was less. Subepithelial band of mononuclear infiltrate is lesser but thicker and epithelial thickness was indirectly proportional to subepithelial inflammatory infiltrate.

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

## References:


1. *Mignogna MD, Fedele S, Russo LL, Muzio LL, Bucci E. Immune activation and chronic inflammation as the cause of malignancy in oral lichen planus. Is there any evidence? Oral Oncology 2004;40: 120-130*
2. *Boyd AS, Kenneth HN. Lichen planus. J Am Acad Dermatol 1991; 25(4):*
3. *Eisenberg E, Krutchkoff DJ. Lichenoid lesions of oral mucosa. Oral Surg Oral Med Oral Pathol 1992;73: 699-704*
4. *Hietanen J, Paasonen MR, Kuhlefelt M, Malmstrom M. A retrospective study of oral lichen planus patients with concurrent or subsequent development of malignancy. Oral Oncology 1999;35:278 – 82.*
5. *Rajendran R. Diseases of the skin. In, Rajendran R, Shivapathasundaram B (ed). Shafer's Textbook of Oral pathology, 6th edition. Elsevier, 2009;169-173*
6. *Rice JP, Hamburger J. Oral Lichenoid Drug Eruptions. Their recognition and management. Dent Update. 2002;29:442 – 47.*
7. *Meij VEH, Waal VI. Lack of clinicopathologic correlation in the diagnosis of oral lichen planus based on the presently available diagnostic criteria and suggestions for modifications. J Oral Pathol Med 2003 ; 32 : 507-512*
8. *Eisenberg E: Oral lichen planus: A benign lesion: J Oral Maxillofacial Surg 2000: 58: 1278-1285*
9. *Lehner T & Shillitoe E. Immunological aspects of cancer. In Cohen B & Kramer's Scientific foundations of Dentistry. William Heinemann Medical Books Ltd. Pg No: 163 – 171.*
10. *Mollaoglu N. Oral lichen planus: a review. British Journal of Oral and Maxillofacial surgery. 2000: Pg No: 370-377.*


Source of Support - Nil Conflict of Interest - None declared

***How to cite this article:***
Usha Balan, Nitin Gonsalves, Maji Jose: A quantitative evaluation of epithelium and inflammatory infiltrate of Lichenplanus and Lichenoid reactions, Oral Max Path J, 3(2), Jul-Dec 2012 : 233-237