Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies NLM ID: 101716117

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr Indian Citation Index (ICI) Index Copernicus value = 100

(e) ISSN Online: 2321-9599;

(p) ISSN Print: 2348-6805

Original Research

Histopathological features of Oral Lichen Planus: A Retrospective Study

¹Piyush Gandhi, ²Harkanwal Preet Singh, ³Haneet Kaur, ⁴Alka Sharma

¹Associate Professor, ²Professor, Department of Oral Pathology and Microbiology, ³Former B.D.S. student, ⁴B.D.S. Intern, Dasmesh Institute of Research and Dental Sciences, Faridkot, Punjab, India

ABSTRACT:

Background: This study was conducted to assess the Histopathological features of oral lichen planus and its response to corticosteroid therapy.**Material and methods**: Patients who had their first diagnosis of oral lichen planus were eligible for inclusion in this study. All cases were classified using the updated 2003 World Health Organization criteria. All individuals complained of suffering from pain as well as difficulty when consuming food. Selected cases had classic features on histopathology (basal layer hydropic degeneration, band-like subepithelial chronic lymphocytic inflammatory infiltrate, absence of epithelial dysplasia) and classic clinical features (bilateral and roughly symmetrical lesions, white-grey papules in a reticular pattern, and occasional presence of erosive-ulcerative, vesicular, and/or plaque-like lesions).**Results**: All patients had hydropic degeneration of the basal layer and a band-like subepithelial chronic lymphocytic inflammatory infiltration at the time of diagnosis, according to the histological findings. 67% of the patients had plasma cells in the connective tissue (in the band-like infiltration of T lymphocytes). Epithelial hyperkeratosis (86%) was also observed, along with epithelial hyperplasia (23%), epithelial acanthosis (46%), hypergranulosis (49%), civatte bodies (4%), epithelial crest flattening (20%), and fibrin deposits in the epithelium (3%). **Conclusion**: It's possible that fewer flare-ups and a greater response to standard treatment with topical corticosteroids are linked to the existence of plasma cells in OLP. **Keywords**: Oral lichen planus, histopathology, corticosteroid.

Received: 26 June, 2023

Accepted: 29 July, 2023

Corresponding author: Harkanwal Preet Singh, Professor, Department of Oral Pathology and Microbiology, Dasmesh Institute of Research and Dental Sciences, Faridkot, Punjab, India

This article may be cited as: Gandhi P, Singh HP, Kaur H, Sharma A. Histopathological features of Oral Lichen Planus: A Retrospective Study. J Adv Med Dent Scie Res 2023;11(8):133-135.

INTRODUCTION

Lichen planus is a chronic inflammatory disease that affects the skin, hair follicles, nails, and mucosa.¹ Mucosal surfaces affected include the oral, genital, ocular, otic, esophageal surfaces, and in rarer instances, the bladder, nasal, laryngeal, and anal surfaces. The skin and oral mucosa are the major sites that are affected.²

The oral variant, termed oral lichen planus (OLP), is a chronic condition with periods of relapses and remissions, requiring long-term symptomatic treatment and surveillance monitoring. About 15% of patients with oral lichen planus (OLP) develop cutaneous lesions, and 20% develop genital lesions.³

Cutaneous involvement is usually self-limiting and characterized by violaceous, pruritic papules with overlying reticular white striae, known as Wickam striae. Lesions most commonly appear on the trunk or extremities, such as the wrists and ankles.⁴

Genital involvement in females demonstrates erythema, erosion, white reticulated plaques, agglutination, resorption of the labia, or scarring.⁵ Males may demonstrate annular, papulosquamous lesions on the glans penis. Associated symptoms may include dysuria and dyspareunia.⁶ More than a quarter OLP patients also display of oesophageal involvement, with symptomatic patients complaining of dysphagia and odynophagia. An endoscopic examination may reveal friable mucosa, white plaques, erythema, ulceration, erosions, and stricture formation.7-9

Hence, this study was conducted to assess the Histopathological features of oral lichen planus and its response to corticosteroid therapy.

MATERIAL AND METHODS

Patients who had their first diagnosis of oral lichen planus were eligible for inclusion in this study. All cases were classified using the updated 2003 World Health Organization criteria. All individuals complained of suffering from pain as well as difficulty when consuming food.Selected cases had classic features on histopathology (basal layer hydropic degeneration, band-like subepithelial chronic lymphocytic inflammatory infiltrate, absence of epithelial dysplasia) and classic clinical features (bilateral and roughly symmetrical lesions, white-grey papules in a reticular pattern, and occasional presence of erosive-ulcerative, vesicular, and/or plaque-like lesions).

Patient age, sex, medical history, OLP lesion location, OLP type, OLP clinical features (erosion, ulceration, plaque, papular), OLP diagnosis date, histopathological findings, number of annual exacerbations, treatment in each exacerbation was recorded. All the results were assessed by SPSS software.

RESULTS

Table 1	: Gend	ler-wise	distributi	on of	f subj	jects
---------	--------	----------	------------	-------	--------	-------

Gender	Number of subjects
Males	15
Females	85
Total	100

One hundred patients with OLP participated in the trial. All patients were considered. There were 15 males and 85 females in total. 89 of them had never smoked. Clinically, reticular OLP accounted for 38% of patients, atrophic-erosive for 35%, plaque for 25%, and papular for 2%. The buccal mucosa was the most common site (36%).

Histopathological features	Number of subjects
Plasma cells	67%
Epithelial hyperkeratosis	86%
Epithelial dysplasia	23%
Epithelial acanthosis	46%
Hypergranulosis	49%
Civatte bodies	04%
Epithelial crest flattening	20%
Fibrin deposits in epithelium	03%

All patients had hydropic degeneration of the basal layer and a band-like subepithelial chronic lymphocytic inflammatory infiltration at the time of diagnosis, according to the histological findings. 67% of the patients had plasma cells in the connective tissue (in the band-like infiltration of T lymphocytes). Epithelial hyperkeratosis (86%) was also observed, along with epithelial hyperplasia (23%), epithelial acanthosis (46%), hypergranulosis (49%), civatte bodies (4%), epithelial crest flattening (20%), and fibrin deposits in the epithelium (3%).

DISCUSSION

Lichen planus is a chronic inflammatory disease that affects the skin and the mucus membrane. Oral lichen planus (OLP), the mucosal counterpart of cutaneous lichen planus, presents frequently in the fourth decade of life and affects women more than men in a ratio of 1.4:1.¹⁰ The disease affects 1–2% of the population.^{11,12} It is seen clinically as reticular, papular, plaque-like, erosive, atrophic or bullous types. Intraorally, the buccal mucosa, tongue and the gingiva are commonly involved although other sites may be rarely affected.¹³ Oral mucosal lesions present alone or with concomitant skin lesions. The skin lesions present as violaceous flat-topped papules in

ankles, wrist, and genitalia, but characteristically the facial skin is spared.

OLP is a T-cell-mediated immunological disease to an unknown antigenic change in the skin or oral mucosa in genetically predisposed patients.^{14,15} Several studies have analyzed those genes that are potentially involved in the pathogenesis and evolution of OLP include JUN, EGFR, FOS, IL2 and ITGB4 and other MHC genes such as HLA B 27, HLA B 51, HLA BW-57, HLA-DR1 and HLA-DR6 also play a role.¹⁶⁻¹⁸

In a genetically predetermined individual, various triggering factors cause unmasking of lichen planusspecific antigen, which is displayed by MHC class 1 molecules. This, in turn, favors the recruitment of CD8+ T-cells (cytotoxic) and it is activated. This aids in the release of tumor necrosis factor (TNF)- α , and various other cytokines and chemokines leading to release of mucous membrane pemphigoid (MMP), eventually causing basement membrane disruption resulting in migration of T-cells into the epithelium thus resulting in keratinocyte apoptosis. Other nonspecific immune responses such as chemokine ligand activation and mast cell degranulation also play a role in basement membrane disruption in the pathogenesis of OLP. Cytokine polymorphisms TH1 and TH2 determine the appearance of lesions in the oral mucosa (interferon - associated) or on the skin

(TNF- α associated).¹⁹Hence, this study was conducted to assess the Histopathological features of oral lichen planus and its response to corticosteroid therapy.

In this study, all patients had hydropic degeneration of the basal layer and a band-like subepithelial chronic lymphocytic inflammatory infiltration at the time of diagnosis, according to the histological findings. 67% of the patients had plasma cells in the connective tissue (in the band-like infiltration of T lymphocytes). Epithelial hyperkeratosis (86%) was also observed, along with epithelial hyperplasia (23%), epithelial acanthosis (46%), hypergranulosis (49%), civatte bodies (4%), epithelial crest flattening (20%), and fibrin deposits in the epithelium (3%).

Gao Y et al²⁰estimated the histological features of oral lichen planus (OLP) which underwent malignant transformation and discuss the current problems in OLP pathological diagnosis. Using the modified WHO OLP criteria (2003), reevaluated the pathological changes of cases diagnosed initially as OLP and transformed into squamous cell carcinomas indicated by subsequent biopsies. Among 3721 cases of OLP clinically and pathologically diagnosed during 1984 and 2015, there were 19 cases (0.51%) having underwent malignant transformation. Reevaluation of the initial biopsies revealed that 10 cases did not meet the criteria of OLP, as without characteristic basal cell liquefaction, not exhibiting sufficient band-like lymphocytes infiltration, or presenting with epithelial dysplasia. There were 9 cases of OLP malignant transformation left after the reevaluation. Pathological diagnosis of OLP should fully fit the criteria i.e. basal cell liquefaction, typical band-like lymphocytes infiltration and absence of epithelial dysplasia, which is also a prerequisite for researches in malignant transformation of OLP.

CONCLUSION

It's possible that fewer flare-ups and a greater response to standard treatment with topical corticosteroids are linked to the existence of plasma cells in OLP.

REFERENCES

- Farhi D, Dupin N. Pathophysiology, etiologic factors, and clinical management of oral lichen planus, part I: facts and controversies. Clin Dermatol. 2010 Jan-Feb;28(1):100-8.
- 2. Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal lichen planus: a comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. ScientificWorldJournal. 2014;2014:742826.
- 3. Parashar P. Oral lichen planus. Otolaryngol Clin North Am. 2011 Feb;44(1):89-107, vi. [PubMed]
- 4. Eisen D. The evaluation of cutaneous, genital, scalp, nail, esophageal, and ocular involvement in patients

with oral lichen planus. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 1999 Oct;88(4):431-6. [PubMed]

- Eisen D. The clinical manifestations and treatment of oral lichen planus. Dermatol Clin. 2003 Jan;21(1):79-89.
- 6. Rogers RS, Eisen D. Erosive oral lichen planus with genital lesions: the vulvovaginal-gingival syndrome and the peno-gingival syndrome. Dermatol Clin. 2003 Jan;21(1):91-8, vi-vii.
- Abraham SC, Ravich WJ, Anhalt GJ, Yardley JH, Wu TT. Esophageal lichen planus: case report and review of the literature. Am J SurgPathol. 2000 Dec;24(12):1678-82.
- 8. Fox LP, Lightdale CJ, Grossman ME. Lichen planus of the esophagus: what dermatologists need to know. J Am Acad Dermatol. 2011 Jul;65(1):175-83.
- 9. Katzka DA, Smyrk TC, Bruce AJ, Romero Y, Alexander JA, Murray JA. Variations in presentations of esophageal involvement in lichen planus. Clin Gastroenterol Hepatol. 2010 Sep;8(9):777-82.
- Sugerman PB, Savage NW, Walsh LJ, Zhao ZZ, Zhou XJ, Khan A, et al. The pathogenesis of oral lichen planus. Crit Rev Oral Biol Med. 2002;13:350–65.
- 11. Bouquot JE, Gorlin RJ. Leukoplakia, lichen planus, and other oral keratoses in 23,616 white Americans over the age of 35 years. Oral Surg Oral Med Oral Pathol. 1986;61:373–81.
- Scully C, Beyli M, Ferreiro MC, Ficarra G, Gill Y, Griffiths M, et al. Update on oral lichen planus:etiopathogenesis and management. Crit Rev Oral Biol Med. 1998;9:86–122.
- Ismail SB, Kumar SK, Zain RB. Oral lichen planus and Lichenoid reactions; Etiopathogenesis, diagnosis, management and malignant transformation. J Oral Sci. 2007;49:89–106.
- Gupta S, Jawanda MK. Oral lichen planus: An update on etiology, pathogenesis, clinical presentation, diagnosis and management. Indian J Dermatol. 2015;60:222–9.
- Sameera A, Erugula SR, Rahman MA, Farooq MU, Veldurthi D, Imran S, et al. Oral lichen planus – A review. IOSR JPBS. 2017;12:75–80.
- 16. Shklar G. Lichen planus as an oral ulcerative disease. Oral Surg Oral Med Oral Pathol. 1972;33:376–88.
- Eisenberg E. Clinicopathologic patterns of oral lichenoid lesions. Oral MaxillofacSurg Clin North Am. 1994;6:445.
- Rotaru DI, Sofineti D, Bolboacă SD, Bulboacă AE. Diagnostic criteria of oral lichen planus: A narrative review. Acta Clin Croat. 2020;59:513–22.
- Patil S, Rao RS, Sanketh DS, Sarode SC, Sarode GS. "A universal diagnostic criteria for oral lichen planus: An exigency!," Int J Contemp Dent Med Rev. 2014 Article ID 041214,2014;1-4.
- Gao Y, Luo HY. [Histopathological analysis of oral lichen planus with malignant transformation]. Zhonghua Kou Qiang Yi Xue Za Zhi. 2016 Dec 9;51(12):717-721.