Review Article

Oral lichen planus - Review on etiopathogenesis

K. Srinivas, K. Aravinda, P. Ratnakar, Nitin Nigam, Siddharth Gupta

Department of Oral Medicine & Radiology, Career Postgraduate Institute of Dental Sciences & Hospital, Sitapur Road, Lucknow, India

Address for correspondence:
Dr. Konidena Aravinda,
Department of Oral Medicine & Radiology, Career Postgraduate Institute of Dental Sciences & Hospital, Sitapur Road,
Lucknow – 226 020,
Uttar Pradesh, India.
E-mail: karavinda1@gmail.com

ABSTRACT

Oral lichen planus (OLP) is a chronic mucocutaneous disease of uncertain etiopathogenesis. Several factors including stress, genetics, systemic diseases, viruses, dental restorative materials and drugs have been implicated as causative agents. The disease seems to be mediated by an antigen specific mechanism, activating cytotoxic T cells, and non specific mechanisms like mast cell degranulation and matrix metalloproteinase activation. Further clarity on the pathogenesis will aid in modifying therapeutic interventions, thus significantly reducing the morbidity of OLP patients.

Key words: Etiology, oral lichen planus, pathogenesis

Introduction

Oral lichen planus (OLP) is a chronic, autoimmune mucocutaneous disease, occurring most commonly in the middle aged women. Lichen planus may also occur concurrently or independently in the skin and the genital, anal, esophageal, nasal and laryngeal mucosae. The prevalence of oral lichen planus in general population varies from 1-2%.[1] There is no racial predilection, and the disease appears to be pan racial.[1,2] Andreasen reported that the average age of occurrence in males and females is 40-49 years and 50-59 years, respectively.[3] However, few cases have been reported in children as young as 6 months.[4]

Clinically the oral lesions have been grouped into reticular, papular, plaque like, atrophic, erosive and bullous forms.[3] OLP usually occurs in a bilaterally symmetrical pattern, commonly involving buccal mucosa, gingivae and dorsum of the tongue.[5] The lesions are usually painless, though pain and burning sensation are associated with erosive and atrophic lesions.[6]

Access this article online

Quick Response Code:

Website: www.njms.in

DOI: DOI:10.4103/0975-5950.85847

Etiology

The etiology of OLP appears to be multifactorial and complicated. Earlier studies have implicated stress, anxiety, depression as the causes for OLP.[3,5,7,8] However, whether stress is the cause or the consequence, was left undetermined. Familial cases of OLP have been reported and role of genetic predisposition was considered. Watanabe T *et al.* concluded that human leukocyte antigen (HLA) associated genetic factors play a certain role in the pathogenesis of OLP.[9] Hedberg and associates reported that epithelium involved by OLP was consistently positive for HLA-DR.[10]

Lodi G *et al.* reported that lichen planus is sometimes associated with infections or auto immune diseases and / or neoplasia, but the association had not been established.[11] Certain systemic diseases like diabetes mellitus, hypertension, ulcerative colitis, myasthenia gravis, lupus erythematosus, etc were considered to be associated with OLP.[12,13] A more consistent association was found between chronic liver disease and erosive form of OLP.[14] Recent studies indicate an association between Hepatitis C Virus (HCV) and OLP.[15-21] HCV is a hepatotrophic Ribonucleic acid (RNA) virus, which possibly alters the antigenecity of the epidermis, causing an interaction with activated T- cells, or acts through a modulation of the quality of host immune response.[18]

Oral lichenoid reactions caused by drugs and dental restorative materials have been considered as variants of OLP. Drugs implicated are non – steroidal anti inflammatory agents, sulfonyl ureas, beta blockers, oral hypoglycemic agents, dapsone, pencillamine. Dental restorative materials like amalgam, composite, acrylic, gold have been reported to cause lichenoid reactions. Lichenoid lesions have also been reported in tobacco chewers; however, the causative role of tobacco in the pathogenesis of OLP has not been identified.[22]

Pathogenesis

Current literature suggests that OLP is caused by cluster of differentiation 8 (CD-8) cell mediated damage to the basal keratinocytes leading to apoptosis. The antigen inciting the cytotoxic T cells could be any of the above mentioned factors including stress, chronic liver disease, HCV virus, dental restorative materials and/or drugs.[22] The main event in the pathogenesis appears to be increased production of cytokines leading to the recruitment of Langerhans cells and clonal expansion of cytotoxic cells. Langerhans cells produce increased amounts of interferon-alpha (IFN - α), which further activates cytotoxic cell mediated apoptosis, via the keratinocyte caspase cascade.[1,23] Intercellular adhesion molecules enhance the attraction of cytotoxic T cells. Interferon –γ production increases the apoptosis through the up regulation of p53 and matrix metallo proteinase -1 (MMP-1).[1] Non specific mechanisms like mast cell degranulation and MMP -1 activation further aggravate the T cell accumulation, basement membrane disruption by mast cell proteases and keratinocyte apoptosis (triggered by basement membrane disruption). The chronicity of the OLP lesions might be partly explained by the fact that the basement membrane disruption triggers keratinocyte apoptosis and apoptotic keratinocytes are unable to repair the breach in basement membrane.[23]

Conclusion

Therefore, interaction of various factors is probably responsible for the initiation, aggravation and persistence of OLP. The current treatment modalities are not only inadequate in treating all patients and preventing recurrences, but also have significant side effects. Further clarity on the pathogenesis will aid in modifying therapeutic interventions, thus significantly reducing the morbidity of OLP patients.

References

1. Carazzo M, Thorpe R. Oral lichen planus: a review. Minerva Stomatol 2009;58:519-37.
2. Femiano F, Scully C. Functions of the cytokines in relation oral lichen planus-hepatitis C. Med Oral Patol Oral Cir Bucal 2005;10:E40-4.
3. Andreasen JO. Oral lichen planus. 1. A clinical evaluation of 115 cases. Oral Surg Oral Med Oral Pathol 1968;25:31-42.
4. Strauss RA, Fattore L, Soltani K. The association of mucocutaneous lichen planus and chronic liver disease. Oral Surg Oral Med Oral Pathol 1989;68:406-10.
5. Scully C, El-kom M. Lichen planus: Review and update on pathogenesis J Oral Pathol 1985;14:431-58.
6. Brown KS, Bottomley WK, Puente E, Larigne GL. A retrospective evaluation of 193 patients with oral lichen planus J Oral Pathol Med 1993;22:69-72.
7. Mc Cartan BE. Psychological factors associated with oral lichen planus J Oral Pathol Med 1995;24:273-5.
8. Shah B, Ashok L, Sujatha GP. Evaluation of salivary cortisol and psychological factors in patients with oral lichen planus. Indian J Dent Res 2009;20:288-92.
9. Watanabe T, Ohishi M, Tanaka K, Sato H. Analysis of HLA antigens in Japanese with oral lichen planus J Oral Pathol 1986;15:529-33.
10. Hedberg NM, Hunter N Expression of HLA –DR on keratinocytes in oral lichen planus. J Oral Pathol 1987;16:31-5.
11. Lodi G, Porter SR. Hepatitis C virus and lichen planus: A short review. Oral Dis 1997:3:77-81.
12. Lozada- Nur F, Miranda C. Oral lichen planus: epidemiology, clinical characteristics and associated diseases. Semin Cutan Med Surg 1997;16:273-7.
13. Scully C, Potts AJ, Hamburger J, Wiesenfeld D, McKee JI, el Kom M. Lichen planus and liver disease: how strong is the association. J Oral Pathol 1985:14:224-6.
14. Bagán JV, Aguirre JM, del Olmo JA, Milián A, Peñarrocha M, Rodrigo JM, *et al.* Oral lichen planus and chronic liver disease: A clinical and morphometric study of oral lesions in relation to transaminase elevation. Oral Surg Oral Med Oral Pathol 1994:78:337-42.
15. Al Robaee AA, Alzolibani AA. Oral lichen planus and hepatitis C virus: Is there real association? Acta Dermatoven 2006:15:15-9.
16. Lodi G, Giuliani M, Majorana A, Sardella A, Bez C, Demarosi F, *et al.* Lichen planus and hepatitis C virus: A multicentre study of patients with oral lesions and a systematic review. Br J Dermatol 2004;15:1172-81.
17. Sanchez – Perez J, De Castro M, Buezo GF, Fernandez-Herrera J, Borque MJ, Garcio- Diez.A. Lichen planus and Hepatitis C virus: Prevalence and clinical presentation of patients with lichen planus and Hepatitis C virus infection. Br J Dermatol 1996:134:715-9.
18. Prabhu S, Pavithran K, Sobhanadevi G. Lichen planus and hepatitis C virus (HCV) - Is there an association? A serological study of 65 cases. Indian J Dermatol Venereol Leprol 2002;68:273-4.
19. Gimenez-Garcia R, Perez-Casrillon JL. Lichen planus and hepatitis C virus infection. J Eur Acad Dermatol Venereol 2003:17:291-5.
20. Mignogna MD, Lo Muzio L, Lo Russo L, Fedele S, Ruoppo E, Bucci E. Oral lichen planus: different clinical features in HCV – positive and HCV – negative patients. Int J Dermatol 2000:39:134-9.
21. Aravinda K, Pavani B.V. Hepatitis C virus infection in patients with oral lichen planus. Nigerian J Clin Pract 2011 In-press.
22. Ismail SB, Satish KS, Zain RB. Oral lichen planus and lichenoid reactions: etiopathogenesis, diagnosis, management and malignant transformation J Oral Sci 2007:49:89-106.
23. 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.

How to cite this article: Srinivas K, Aravinda K, Ratnakar P, Nigam N, Gupta S. Oral lichen planus - Review on etiopathogenesis. Natl J Maxillofac Surg 2011;2:15-6.

Source of Support: Nil. **Conflict of Interest:** None declared.