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Clinicopathological analysis of oral lichen planus: A retrospective study

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Aim: Oral lichen planus (OLP), a chronic inflammatory condition affecting the mucous membranes is not contagious but can be persistent, and in some cases, may carry a risk of developing into oral cancer, requiring regular monitoring by healthcare professionals. The prevalence of OLP in the general population is approximately estimated at 1.01%. There are limited studies available on the prevalence of Oral lichen planus in the South Indian Population. This study aimed to identify the most distinctive clinicopathological features of OLP cases.
Materials and methods: Clinical information regarding patients' age, sex, site, laterality, symptoms, provisional diagnosis and the final diagnosis of the lesions were collected from the histopathological report of Oral lichen planus patients seen in a private dental college from Chennai district of Tamil Nadu (South India).
Results: In this study, 99 cases of oral lichen planus (OLP) were analyzed from 3471 histopathological records. OLP was more common in females (60%), with peak incidence at 45 years. The most frequent clinical types were reticular (64%) and erosive (14%), with the buccal mucosa (74%) as the most affected site. Common histopathological features included hyperparakeratosis (76%) and basal cell degeneration (62%). A significant correlation was found between clinically and histopathologically diagnosed OLP, and between basal cell degeneration and subepithelial inflammation. No cases of malignant transformation were reported.
Conclusion: In conclusion, this retrospective analysis of oral lichen planus demonstrated a higher prevalence in females. Histopathologically, hyperkeratosis, basal cell degeneration and subepithelial inflammation were the key features, however, there was no reported malignant transformation.

Keywords: clinical; histopathological features; lichen planus; Oral; South India

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Introduction

Despite being one of the most prevalent chronic oral mucosal diseases, the precise etiology of oral lichen planus (OLP) remains elusive. Improving diagnostic accuracy, early detection and enhancing patient management and treatment options could help in the prevention of malignant transformation as OLP is an oral potentially malignant disorder, with a 1.1 to 1.4% malignant transformation rate.[1] The global prevalence in the general population is approximately estimated as 1.01%, ranging from 0.47% to 1.74% with differences in geographical location. [1-3]

The antigen of OLP may be unmasked by various infectious agents, drugs, mechanical damage or autoimmune responses, whereas in self-peptide antigens are targeted by basal keratinocytes to CD8+ cytotoxic T-cells via IL-2 and IFN-γ, whose activity is further influenced by Th1 and IL-23/Th-17 leading to keratinocyte apoptosis through granzyme and perforin pathways.[4-7] Mast cell degranulation and matrix metalloproteinase (MMP) activation contribute to the nonspecific immune response, resulting in chronic inflammation and basement membrane disruption and contribute to the migration of T-cells into the epithelium.[4] Recent genetic insights highlight the involvement of a combined role of immune dysregulation, epithelial barrier dysfunction and microbial triggers pro-inflammatory cytokines like TNF-α and IFN-γ, chemokines like CXCL9, CXCL10, and CXCL11 in the recruitment of T cells. Epithelial barrier dysfunction was recognized by downregulation of tight junction genes involved in epithelial barrier integrity, including claudin-1 (CLDN1) and occludin (OCLN) suggesting that epithelial cells in OLP are less effective in maintaining a proper mucosal barrier and upregulation of antimicrobial genes such as S100 calcium-binding protein A7 (S100A7) and defensin beta-4A (DEFB4A), indicating an enhanced immune response to microbial pathogen.[4] The deficient immunosuppressive function of transforming growth factor-beta (TGF-β1) may contribute to the chronicity of OLP. [2,4]

Viruses implicated in OLP are Hepatitis C virus (HCV), varicella-zoster virus (VZV), Epstein Barr virus (EBV), human herpesvirus 6 (HHV-6), herpesvirus 7 (HHV-7), human papillomavirus (HPV-11, 16).[8-10] Systemic conditions associated with OLP include diabetes mellitus, metabolic syndrome, hypertension, cholecystitis, dyslipidemia, psychosomatic ailments, thyroid dysfunction, liver dysfunction, nutritional deficiencies and genetic susceptibility to malignancies.[9,11,12] There is the possibility that OLP patients are predisposed to autoaggressive status against different targets, including diabetes mellitus type I, Hashimoto's thyroiditis, multiple sclerosis, systemic lupus erythematosus, Sjogren's syndrome, celiac disease, pulmonary fibrosis and ulcerative colitis.[8,12,13,14] Other genetic alterations involved are the p53 tumour suppressor gene, increased telomerase activity, and loss of c-erbB2 function.[15] Genetic polymorphisms linked with regulation of the immune system in OLP are HLA-DR 1-9, HLA DRB1, HLA DQA1, HLA DQB1.8,16

The Koebner phenomenon occurs in both cutaneous and oral lichen planus. Mechanical trauma, smoking, mucosal injury from sharp teeth and oral habits like lip chewing are Koebnerogenic factors that can aggravate OLP.[7]

Due to the unclear understanding of its pathogenesis, the clinicopathological diagnostic criteria for OLP have been subjected to repeated revisions. In 2016, the American Academy of Oral and Maxillofacial Pathology (AAOMP) revised the WHO diagnostic criteria for OLP. The key AAOMP criteria include a clinical presentation involving the multifocal, symmetric distribution of white and red

lesions exhibiting reticular, Atrophic, plaque-like, papular, bullous and erosive patterns. Its histopathological criteria include Hyperkeratosis, band-like / patchy, predominantly lymphocytic infiltrate in the lamina propria confined to the epithelium–connective tissue interface, basal cell liquefactive degeneration, lymphocytic exocytosis, absence of verrucous epithelial architectural change and absence of epithelial dysplasia.[17]

Direct immunofluorescence (DIF) and microscopy can also be utilized as an adjunct to clinical and histopathological diagnosis.[1,18,19] And immune reactants C3, immunoglobulin IgG, IgM and IgA are the reliable markers for fibrin deposition at the submucosal-mucosal interface and in cytoid bodies.[1] PCNA and P21 are two promising predictive markers requiring further validation for evaluating the malignant transformation risk, and also Immunohistochemistry has a limited utility in diagnosing OLP.[19,20] Managing OLP needs multiple disciplinary collaboration, as proper diagnosis and treatment are crucial due to the potential for extraoral lesions and the risk of oral malignancy.[1] The primary therapeutic options for OLP are topical and systemic corticosteroids, immunomodulatory, antidepressants, antiepileptic and antimalarials light-based therapies like photo biomodulation, photodynamic therapies, alternative therapies with natural agents like Nigella sativa, curcumin, aloe vera.[21-25] The Oral health-related quality of life in OLP patients is generally poor before treatment for OLP. After the treatment of OLP, the oral hygiene of the patient has considerably improved.[26,27]

Limited studies are available on the prevalence of Oral lichen planus in the South Indian population.[28] This study aimed to identify the most distinctive clinicopathological characteristics of OLP patients in the South Indian population

Materials and methods

Clinical and histopathological records from January 2020 to December 2023 were analyzed. Histopathologically diagnosed oral lichen planus cases were screened out of all cases reported in the Department of Oral Pathology and Microbiology, Saveetha Dental College and Hospitals. Prior to the study, clearance was obtained from the institutional ethical committee (IHEC/SDC/OPATH-2105/24/148).

Clinical information regarding patients' age, sex, site, laterality, symptoms, provisional diagnosis and the final diagnosis of the lesions were collected from the histopathological report.

Age was categorized into seven groups (10-19, 20-29, 30-39, 40-49, 50-59, 60-69, 70-79). Anatomical sites were grouped into 7 (Buccal mucosa, retromolar trigone, tongue, gingiva, alveolar mucosa, palatal mucosa). Clinically the cases were provisionally diagnosed and categorized into 9 (Oral lichen planus- Erosive LP, Desquamative gingivitis, leukoplakia, lupus erythematosus, pemphigus, melanoma, candidiasis, squamous cell carcinoma) and histopathological diagnoses were categorized into 5 (OLP, erosive OLP, compatible with lichen planus, or lichenoid dysplasia and systemic lupus erythematosus). Symptoms mentioned were grouped into 7 (burning sensation, non-scrapable lesion, redness, ulceration, pigmentation, desquamation, pain), and the presence of about seven histopathological features (hyperparakeratosis, basal cell degeneration, subepithelial inflammation, max Joseph spaces, acanthosis, melanin incontinence saw tooth rete ridges) was assessed from the histopathological slides.

Data were tabulated in a Microsoft Excel spreadsheet and were analyzed using the statistical software SPSS Statistics. The statistical analysis, whenever applicable, was

performed with the chi-squared test for significance (p-value < 0.05).

Results

A total of 3471 histopathological records cases were screened, out of which 99 cases of oral lichen planus were included in the present study. At the moment of the histopathological diagnosis, 25% of females and 30% of males suffered from systemic diseases like diabetes mellitus and hypertension. No malignant transformation was reported. 4% of cases were reported to have severe epithelial dysplasia after a follow-up of 2 years.

Age ranged from 13 to 78 years, with peak incidence at 45 years; 40% were males and 60% were females. The two clinical manifestations that were most frequently seen were reticular (64%) and erosive (14%) types. The buccal mucosa (74%) and attached gingiva (14%) were the two most frequently impacted locations. The burning sensation (44%) is the most common presenting clinical symptom. Almost 88% of clinically diagnosed OLP cases were consistent with histopathologically diagnosed OLP cases. Hyperparakeratosis (76%), basal cell degeneration (62%) and subepithelial inflammation (59%) were the most common histopathological features (Figure 1-3). 93/99 cases (93.93%) cases were seen bilaterally symmetrical on the buccal mucosa.

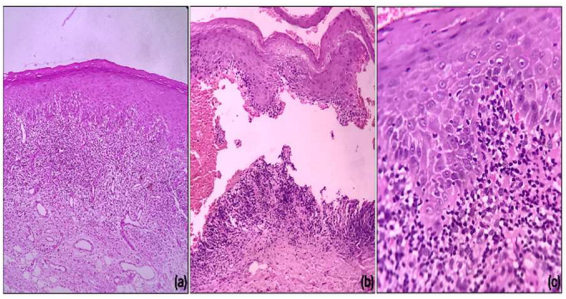

Fig.1: Photomicrograph showing H&E stained sections of Oral lichen planus showing hyperkeratosis and band like subepithelial inflammation (a), Max-Joseph space (b), and basal cell degeneration (c).

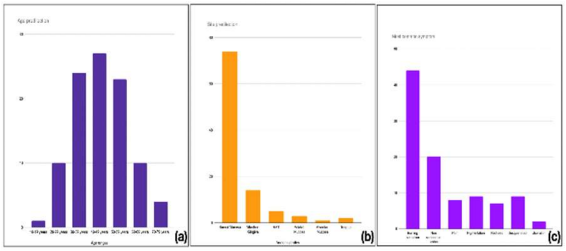

Figure 2: Graphic representation of percentage of age predilection (a), location affected (b), and percentage of predominant clinical symptoms (c).

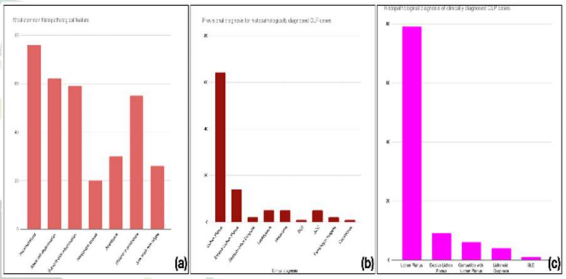

Figure 3: Graphic representation of percentage of prominent histopathological features (a), provisional clinical diagnosis (b), and histopathological diagnosis of cases clinically diagnosed as OLP (c).

When comparing the age distribution of OLP cases, the 40-49-year age group exhibited a statistically significant difference, with a notably higher incidence (p-value < 0.027). Buccal mucosa was found to be the highest number of cases histopathologically diagnosed as OLP, and it was statistically significant when compared to the other sites (p-value < 0.05). There is a statistically significant correlation between basal cell degeneration and subepithelial inflammation in cases of OLP (p-value < 0.01). A statistically significant association was found between clinically and histopathologically diagnosed oral lichen planus (OLP) (p-value < 0.01).

Discussion

Oral lichen planus is a chronic inflammatory mucosal condition characterized by distinctive white striae, patches, erosions, or ulcers in the oral cavity.

OLP is reported to have a potential association with systemic diseases, including Hepatitis C and autoimmune disorders. Recent advances in diagnostic techniques and treatment approaches have emphasized the importance of both symptom relief and targeting the underlying pathogenesis. This research aimed to identify the most distinctive clinicopathological features of oral lichen planus.

In the present investigation, reticular and erosive lesions, along with a burning sensation in the buccal mucosa, were the most common clinical manifestations. Among the histopathological characteristics, basal cell degeneration was predominantly associated with chronic inflammatory infiltration. Our presentation was similar to a study by Shen ZY et al, a retrospective analysis of 518 OLP patients in China.[29] It consisted of 353 females and 165 males, with an average age of 46.3 years (range 9-81 years). The buccal mucosa was the most commonly affected site (87.8%). Clinically, white lichen and red lichen presentations were observed in 52.3% and 47.7% of patients.[29]

It was demonstrated that buccal mucosa is the most typical site for OLP. This is consistent with the previous studies where the buccal and labial mucosa (79.92%) were the commonest.[30] This could be attributed to the constant irritation and trauma, varying epithelial thickness, and, additionally, the non-keratinized epithelium is more prone to damage.[31] Also, buccal mucosa has a rich immune surveillance mechanism as its constant barrier to oral pathogens.[32] OLP, being an autoimmune condition, may affect buccal mucosa often due to its higher susceptibility to immune attack, leading to chronic inflammation and lesion development.[32] Previous studies indicated a male predominance among most patients in the third decade of life.[30] Our present study showed that females have slightly higher predilection than males, similar to previous studies.[28,33,34] Women are generally susceptible to autoimmune diseases due to their stronger immune response and hormonal influences. X- Chromosome inactivation and other epigenetic mechanisms may also contribute to this.[35]

Among the histopathological findings, hyperkeratosis is noted in almost all OLP cases, which is similar to previous studies.[28,31,33,36] Hyperkeratosis is a compensatory response to epithelial damage and chronic inflammation as the tissue attempts to protect itself and repair the damaged epithelium. There is increased keratinocyte proliferation and differentiation and cellular turnover, resulting in a thicker keratinized layer on the mucosal surface.[36]
In our study, basal cell degeneration and subepithelial inflammation were observed to coexist in most cases. This may be explained by antigen-specific and nonspecific inflammatory mechanisms that lead to the increased localization of CD8+ T cells subjacent to the epithelium, leading to apoptosis of keratinocytes.[28] And vice versa, the degeneration of basal epithelial cells may alter the basal lamina, which stimulates the macrophages by epithelial antigens, and these macrophages then transfer immunological information to lymphocytes, activating the inflammatory process.[37,38]

Sawtooth rete ridges are common histopathological features in cutaneous lichen planus (CLP) in contrast to OLP.[39] This could be attributed to the differences in the thickness of the epithelium, keratinization and inflammatory response between skin and oral mucosa. OLP displays hyperparakeratosis, a less prominent granular layer, and atrophy of the oral epithelium. Further, the oral mucosa is more delicate and less keratinized compared to the skin, and it reacts differently to chronic inflammation. Instead of developing acanthosis as in cutaneous LP, the oral epithelium develops

atrophy, leading to a comparative flatter interface between the epithelium and connective tissue.[39,40] Our study result showed around 26% of cases showed sawtooth rete ridges, and this is in contrast to a previous study where 60% of cases showed sawtooth rete ridges and epithelial atrophy was not observed in the reticular forms studied.[33]

No malignant transformation was reported in our study, with four percentage cases of OLP with severe epithelial dysplasia. The persistent chronic inflammatory microenvironment in OLP is itself a predisposing factor for carcinogenesis.[41,42] Pimolbutr et al 2024, stated that a pre-existing OLP is associated with two times increased risk of developing epithelial dysplasia than without OLP, [42] but there was no evidence of a greater risk of OLP with oral epithelial dysplasia to oral squamous cell carcinoma. This could be because the malignant transformation of OLP is a slow process, and it requires a longer duration of at least 20 years.[42,43] Most of the studies do not include such long-term follow-up.

OLP with dysplasia cases must be monitored at regular intervals to detect any early carcinogenesis, as the malignant transformation of OLP was reported in previous studies. Bandyopadhyay et al reported that the majority (92.31%) of cases were diagnosed with oral lichen planus (OLP) without dysplasia. Additionally, two patients (1.4%) who were previously diagnosed clinically and histopathologically with OLP subsequently developed oral squamous cell carcinoma.[30] For early detection, several markers could be used in the prediction of the malignant transformation. Abdallah A et al, proposed that survivin could predict malignant transformation and could be an early diagnostic and prognostic marker. Also, CD146 expression was observed in all OLP cases, which progressed to OSCC.[44] Additionally, miR-137 expression was lower in OSCC cell lines, and its inhibition led to a significant increase in cell proliferation, highlighting the tumor suppressor role of miR-137 in OSCC.[45-46]

Periods of longer follow-ups are necessary to fully understand the long-term malignant transformation risk in OLP patients, and this implements educational programs to increase awareness among patients and healthcare providers about the risks of OLP and the importance of regular monitoring to improve early detection and management.

Conclusion

The findings of the present study reveal that OLP is prevalent in adult females, commonly presenting with bilaterally affecting the buccal mucosa, accompanied by varying levels of oral discomfort. The lesions are generally reticular or erosive forms. Hyperkeratosis, along with basal cell degeneration and subepithelial bands of dense lymphocytes, are the key features noted. The study does not specify the duration of follow-up for the patients. Longer follow-up periods are, however necessary to fully understand the long-term malignant transformation risk in OLP patients. OLP with dysplasia highlights the need for vigilant monitoring and follow-up.

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Data availability: Available on request

Conflicts of Interest: None declared

Financial interests: The authors declare they have no financial interests.

Ethical approval: Prior informed consent was obtained from all the patients included in the study [Saveetha Dental College-Institutional Human Ethical Committee (SDC-IHEC) with approval number IHEC/SDC/OPATH-2105/24/148]

References

1. González-Moles MÁ, Ramos-García P, Warnakulasuriya S. An appraisal of highest quality studies reporting malignant transformation of oral lichen planus based on a systematic review. Oral Dis. 2021 Nov;27(8):1908-1918. doi: 10.1111/odi.13741.
2. Vo PT, Choi SS, Park HR, Lee A, Jeong SH, Choi Y. Gene signatures associated with barrier dysfunction and infection in oral lichen planus identified by analysis of transcriptomic data. PLoS One. 2021 Sep 10;16(9):e0257356. doi: 10.1371/journal.pone.0257356.
3. Swathi KV, Maragathavalli G. Erosive oral lichen planus—A case report. Res J Pharm Tech. 2019;12(6):3007-3011. doi:10.5958/0974-360X.2019.00508.0.
4. Sugerman PB, Savage NW, Walsh LJ, Zhao ZZ, Zhou XJ, Khan A, Seymour GJ, Bigby M. The pathogenesis of oral lichen planus. Crit Rev Oral Biol Med. 2002;13(4):350-65. doi: 10.1177/154411130201300405.
5. Vičić M, Hlača N, Kaštelan M, Brajac I, Sotošek V, Prpić Massari L. Comprehensive Insight into Lichen Planus Immunopathogenesis. Int J Mol Sci. 2023 Feb 3;24(3):3038. doi: 10.3390/ijms24033038.
6. Deng X, Wang Y, Jiang L, Li J, Chen Q. Updates on immunological mechanistic insights and targeting of the oral lichen planus microenvironment. Front Immunol. 2023 Jan 9;13:1023213. doi: 10.3389/fimmu.2022.1023213.
7. Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal lichen planus: a comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. ScientificWorldJournal. 2014 Jan 30;2014:742826. doi: 10.1155/2014/742826.
8. Elenbaas, Andrea & Enciso, Reyes & Al-Eryani, Kamal. (2021). Oral Lichen Planus: A review of clinical features, etiologies, and treatments. Dentistry Review. 2. 100007. 10.1016/j.dentre.2021.100007
9. Yildirim B, Sengüven B, Demir C. Prevalence of herpes simplex, Epstein Barr and human papilloma viruses in oral lichen planus. Med Oral Patol Oral Cir Bucal. 2011 Mar 1;16(2):e170-4. doi: 10.4317/medoral.16.e170.
10. Lucchese A, Di Stasio D, Romano A, Fiori F, De Felice GP, Lajolo C, Serpico R, Cecchetti F, Petruzzi M. Correlation between Oral Lichen Planus and Viral Infections Other Than HCV: A Systematic Review. J Clin Med. 2022 Sep 19;11(18):5487. doi: 10.3390/jcm11185487.
11. Hasan S, Ahmed S, Kiran R, Panigrahi R, Thachil JM, Saeed S. Oral lichen planus and associated comorbidities: An approach to holistic health. J Family Med Prim Care. 2019 Nov 15;8(11):3504-3517. doi: 10.4103/jfmpc.jfmpc_749_19.
12. Santhosh T, Vadivel JK, Malaiappan S.Prevalence of erosive lichen planus: A retrospective study. Eur J Mol Clin Med. 2020;7(1):1547-1554.
13. De Porras-Carrique T, Ramos-García P, Aguilar-Diosdado M, Warnakulasuriya S, González-Moles MÁ. Autoimmune disorders in oral lichen planus: A systematic review and meta-analysis. Oral Dis. 2023 May;29(4):1382-1394. doi: 10.1111/odi.14127.
14. Nosratzehi T. Oral Lichen Planus: an Overview of Potential Risk Factors, Biomarkers and Treatments. Asian Pac J Cancer Prev. 2018 May 26;19(5):1161-1167. doi: 10.22034/APJCP.2018.19.5.1161.
15. Mithani SK, Mydlarz WK, Grumbine FL, Smith IM, Califano JA. Molecular genetics of premalignant oral lesions. Oral Dis. 2007 Mar;13(2):126-33. doi: 10.1111/j.1601-0825.2006.01349.x.
16. Luis-Montoya P, Yamamoto-Furusho JK, Vega-Memije E, Rodríguez-Carreón A, Ruiz-Morales JA, Vargas-Alarcón G, Domínguez-Soto L, Granados J. HLA-DRB1*0101 is associated with the genetic susceptibility to develop lichen planus in the Mexican Mestizo population. Arch Dermatol Res. 2007 Oct;299(8):405-7. doi: 10.1007/s00403-007-0769-2.
17. Idrees M, Farah CS , Khurram SA, Firth N, Soluk-Tekkesin M, Kujan O. Observer agreement in the diagnosis of oral lichen planus using the proposed criteria of the American Academy of Oral and Maxillofacial Pathology. J Oral Pathol Med. 2021; 50: 520–527. https://doi.org/10.1111/jop.13170
18. Rotaru DI, Sofineti D, Bolboacă SD, Bulboacă AE. Diagnostic Criteria of Oral Lichen Planus: A Narrative Review. Acta Clin Croat. 2020 Sep;59(3):513-522. doi: 10.20471/acc.2020.59.03.16.
19. Vibhute NA, Jagtap SV, Patil SV. Velscope guided oral cancer screening: A ray of hope in early oral cancer diagnosis. J Oral Maxillofac Pathol. 2021 Sep-Dec;25(3):548-549. doi: 10.4103/jomfp.JOMFP_315_20.
20. Al-Jamaei AA, Subramanyam RV, Helder MN, Forouzanfar T, Ruslin M, Meij EV, et al. Significance of immunohistochemistry biomarkers in prediction of malignant transformation of oral lichen planus: A systematic review. Med Oral Patol Oral Cir Bucal. 2022 Sep 1;27(5):e480-e488. doi: 10.4317/medoral.25491.
21. Haripriya R, Vadivel JK. Usage of Topical Therapies in the Management of Oral Lichen Planus. J Evid Based Med Healthc 2022;9(06):30.
22. Vaishnavi Devi B, Santhosh Kumar MP. Treatment modalities in the management of oral lichen planus—An institutional experience. J Res Med Dent Sci. 2022;10(8):184-195

23. Scully C, Eisen D, Carrozzo M. Management of oral lichen planus. Am J Clin Dermatol. 2000 Sep-Oct;1(5):287-306. doi: 10.2165/00128071-200001050-00004.
24. Soh WK, Cheah KF, Veettil SK, Pandiar D, Nimbalkar S, Gopinath D. Photobiomodulation Therapy in the Management of Oral Lichen Planus: A Systematic Review and Meta-Analysis. Eur J Dent. 2024 May 14. doi: 10.1055/s-0044-1782213.
25. Kumar S L, Naik Z, Panwar A, M S, Keluskar V, Kumar RS. Comparative evaluation of the efficacy of Nigella sativa (75% v/v) cream and clobetasol propionate (0.05% w/w) gel in oral lichen planus-a double-blinded randomized control trial. Oral Maxillofac Surg. 2024 Mar;28(1):225-234. doi: 10.1007/s10006-022-01130-6.
26. Yuwanati M, Gondivkar S, Sarode SC, Gadbail A, Sarode GS, Patil S, Mhaske S. Impact of Oral Lichen Planus on Oral Health-Related Quality of Life: A Systematic Review and Meta-Analysis. Clin Pract. 2021 May 7;11(2):272-286. doi: 10.3390/clinpract11020040.
27. Sushanthi LC, Ramani P, Ramasubramanian A, Gheena S, Krishnan RP. Serum Cortisol Levels in Lichen Planus: A Systematic Review with Meta-Analysis. Indian J Dermatol. 2021 Nov-Dec;66(6):654-659. doi: 10.4103/ijd.ijd_95_21.
28. Vijayan AK, Muthukrishnan A, Nair AM, Baby JJ. Demographics, Clinicopathological Profile of Oral Lichen Planus of South Kerala Population: A Cross-Sectional Study. Cureus. 2022 Sep 28;14(9):e29688. doi: 10.7759/cureus.29688.
29. Shen ZY, Liu W, Zhu LK, Feng JQ, Tang GY, Zhou ZT. A retrospective clinicopathological study on oral lichen planus and malignant transformation: analysis of 518 cases. Med Oral Patol Oral Cir Bucal. 2012 Nov 1;17(6):e943-7. doi: 10.4317/medoral.17778.
30. Bandyopadhyay A, Behura SS, Nishat R, Dash KC, Bhuyan L, Ramachandra S. Clinicopathological Profile and Malignant Transformation in Oral Lichen Planus: A Retrospective Study. J Int Soc Prev Community Dent. 2017 May-Jun;7(3):116-124. doi: 10.4103/jispcd.JISPCD_103_17.
31. Atsushi Abe, Yu Ito, Hiroki Hayashi, Moeko Momokita, Shinichi Taniguchi, Atsushi Nakayama. Evaluation of the classification and clinical characteristics of oral lichen planus and its treatment effect in different clinical types: A retrospective study. Advances in Oral and Maxillofacial Surgery. 2022;8:100369. https://doi.org/10.1016/j.adoms.2022.100369.
32. Pelaez-Prestel HF, Sanchez-Trincado JL, Lafuente EM, Reche PA. Immune Tolerance in the Oral Mucosa. Int J Mol Sci. 2021 Nov 10;22(22):12149. doi: 10.3390/ijms222212149.
33. Vijayan AK, Muthukrishnan A. A Clinicohistopathological Characterization of Oral Lichen Planus: A Cross-Sectional Study. Cureus. 2022 Oct 21;14(10):e30568. doi: 10.7759/cureus.30568.
34. Munde AD, Karle RR, Wankhede PK, Shaikh SS, Kulkurni M. Demographic and clinical profile of oral lichen planus: A retrospective study. Contemp Clin Dent. 2013 Apr;4(2):181-5. doi: 10.4103/0976-237X.114873.
35. Youness A, Miquel CH, Guéry JC. Escape from X Chromosome Inactivation and the Female Predominance in Autoimmune Diseases. Int J Mol Sci. 2021 Jan 23;22(3):1114. doi: 10.3390/ijms22031114.
36. Lavanya N, Jayanthi P, Rao UK, Ranganathan K. Oral lichen planus: An update on pathogenesis and treatment. J Oral Maxillofac Pathol. 2011 May;15(2):127-32. doi: 10.4103/0973-029X.84474.
37. Paul M, Shetty DC. Analysis of the changes in the basal cell region of oral lichen planus: An ultrastructural study. J Oral Maxillofac Pathol. 2013 Jan;17(1):10-6. doi: 10.4103/0973-029X.110690.
38. Mignogna MD, Fedele S, Lo Russo L, Lo Muzio L, Bucci E. Immune activation and chronic inflammation as the cause of malignancy in oral lichen planus: is there any evidence ? Oral Oncol. 2004 Feb;40(2):120-30. doi: 10.1016/j.oraloncology.2003.08.001.
39. Saharkhiz, Arezoo & Aminzadeh Gohari, Atousa & Yahyaabady, Roya. (2022). A Histomorphology Comparison of Saw-Tooth Shaped Rete Ridge Between Oral and Cutaneous Lichen Planus. ENVIRO Dental Journal. 4. 20-24. 10.12944/EDJ.04.01.05.
40. Bansal D, Kamboj M, Anand R, Pandiar D, Narwal A, Sivakumar N, Devi A. Association of childhood vaccination with pediatric lichen planus: A systematic review. Int J Dermatol. 2023 Jan;62(1):22-31. doi: 10.1111/ijd.15974.
41. Lodi G, Scully C, Carrozzo M, Griffiths M, Sugerman PB, Thongprasom K. Current controversies in oral lichen planus: report of an international consensus meeting. Part 2. Clinical management and malignant transformation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005 Aug;100(2):164-78. doi: 10.1016/j.tripleo.2004.06.076.
42. Pimolbutr K, Lim WT, Leeson R, Hopper C, Kalavrezos N, Liew C, et al. Prognosis of oral epithelial dysplasia in individuals with and without oral lichen planus. Oral Dis. 2024 Mar;30(2):504-517. doi: 10.1111/odi.14503.
43. Gururaj N, Hasinidevi P, Janani V, Divynadaniel T. Diagnosis and management of oral lichen planus-Review. J Oral Maxillofac Pathol. 2021 Sep-

Dec;25(3):383-393. doi: 10.4103/jomfp.jomfp_386_21.
44. Abdallah EA, Baghdadi HM, Abdel Ghani SA. Role of Survivin and CD146 in the progression of Oral Lichen Planus to Oral Squamous Cell Carcinoma (Immunohistochemical study). Ain Shams Dental Journal. 2021 Sep 1;23(3):35-41.
45. Saber A, Tohamy S, Sholqamy M, Mohamed L. Down-regulation of MicroRNA-137 Improves Proliferation of Oral Squamous Cell Carcinoma by Up-regulation of NF-Y. Ain Shams Dental Journal, 2024; 34(2): 241-250. doi: 10.21608/asdj.2024.288126.1275
46. Denewer S, Elsabaa H. Effect of caffeine versus hydrogen peroxide on human skin fibroblast cell line cytotoxicity, cell cycle phases, and apoptosis. Ain Shams Dental Journal. 2024 Jun 1;34(2):152-8.