



## SQUAMOUS CELL CARCINOMA ORIGINATING FROM EROSIIVE ORAL LICHEN PLANUS: A CASE REPORT AND REVIEW OF LITERATURE

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### ABSTRACT

Oral lichen planus (OLP) is a T- cell mediated mucocutaneous disease of autoimmune origin affecting stratified squamous epithelium in 0.5-2.6% population. Genetic predisposition, stress, anxiety, hepatitis C, immunocompromising status, dental materials, certain drugs have been found to cause lichen planus but with lack of evidence. It is associated with the increased risk of developing into oral squamous cell carcinoma (OSCC). Transformation rate of OLP to OSCC in some review ranged from 0.4 – 5.6% and in another from 0% to 12.5%. Considering its malignancy potential, it seems important to have a standardized diagnostic criteria, treatment, and clinical follow-up of patients with OLP so that the disease is diagnosed at an early stage and cured on time. Here we present a case of 50-year-old female who reported with erosive form of lichen planus in left buccal mucosa, later lost the follow up and reported with oral squamous cell carcinoma (OSCC) in the same region after 1.5 years, potentially originated from pre-existing OLP.

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## INTRODUCTION

Lichen planus (LP) is a chronic mucocutaneous disease affecting stratified squamous epithelium- skin, oral mucosa and genitalia, occurring in 0.5-2.6% of population.<sup>(1)</sup> Erasmus Wilson coined the term lichen planus (*lichen* means primitive plant like algae & fungi and *planus* means flat). LP was first described clinically in 1869 as a chronic mucocutaneous illness. In 1906 it was histologically described by Dubreuilh for the first time. Oral lichen planus (OLP) could either occur alone or accompany or precede cutaneous lesions. Etiology of OLP is not very well understood. Genetic predisposition, stress, anxiety, hepatitis C, immunocompromising status, dental materials, certain drugs have been found to cause lichen planus in literature but there is lack of evidence. OLP is characterised by hyperkeratosis with varying thickness of the epithelium, basal cell liquefaction degeneration with a dense infiltrate of lymphocytic cells adjacent to the basement membrane.

It mostly affects the middle age population with slight female predisposition. In 1910, Hallopeau reported 1<sup>st</sup> case of malignant transformation in pre-existing OLP. From that point, many such cases have been reported in the literature. Considering its malignancy potential, it seems important to have a standardized diagnostic criteria, treatment, and clinical follow-up of patients with OLP so that the disease is diagnosed at an early stage and cured on time. Here we present a case report of oral squamous cell carcinoma (OSCC), potentially originated from pre-existing OLP involving one of the most common site of malignant transformation, that is buccal mucosa.<sup>(2,3,4)</sup>

## CASE REPORT

A 50-year-old female reported to the department of Oral Medicine and Radiology with the complain of burning sensation and ulcer in the left cheek region for past 4-5 months. On examination there were red patches on the left buccal mucosa surrounded by the white radiating striae and white striae arranged in an interlacing manner were found on the right buccal mucosa [Figure 1A and 1B]. Patient was completely edentulous for past 10 years.

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Figure 1A: Intraoral view showing erosive lichen planus in left buccal mucosa

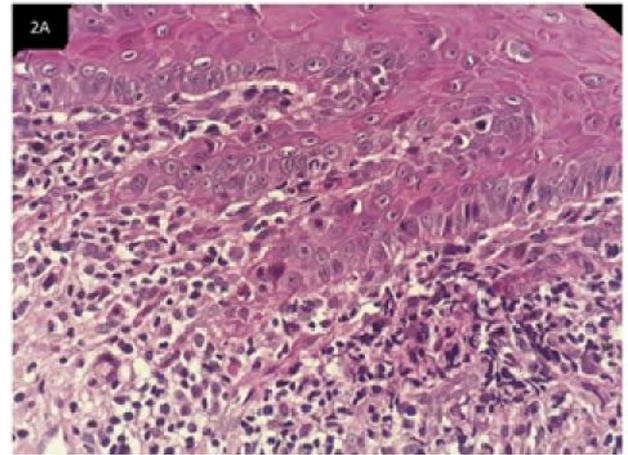


Figure 2A: Histopathology slide showing keratinocyte degeneration, saw tooth rete peg and inflammatory cells



Figure 1B: Intraoral view showing reticular lichen planus on the right buccal mucosa

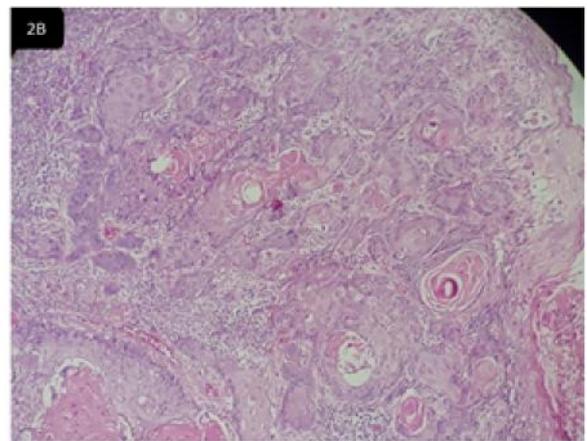


Figure 2B. Histopathology slide showing intense chronic inflammatory response around dysplastic cell in connective tissue with abundant dilated blood vessels. Keratin pearl formation seen in dysplastic sheets and islands



Figure 1C: Intraoral view showing cauliflower like growth on the right buccal mucosa

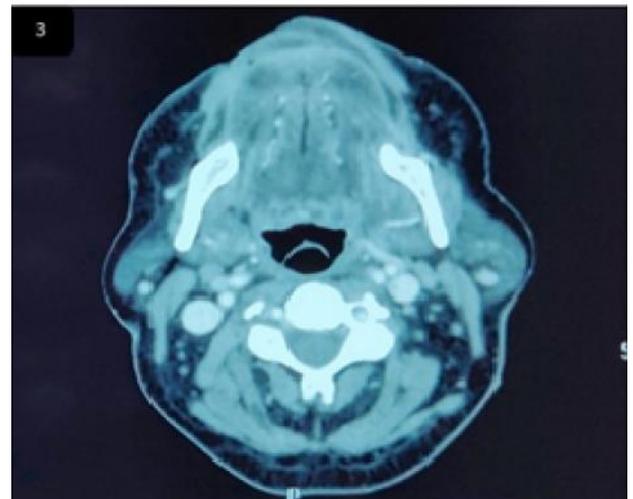


Figure 3. Axial view of CECT shows mild asymmetric differentially enhancing soft tissue attenuating lesion measuring approximately 18 x 6mm lesion in left buccal mucosa with obliteration of gingivobuccal sulcus

Her medical history was not significant. Patient was not having any deleterious habit of smoking or chewing tobacco. Biopsy specimen of lesion from the left buccal mucosa revealed histologic features consistent with lichen planus [Figure 2A].

Treatment was started with an antioxidant (capsule Oxitard) twice a day, an immunomodulator (tablet Dicaris) once a day three days a week for six weeks and topical application of corticosteroid (ointment Kenocort) three times a day.

After three weeks patient reported with mild relief but the patient failed to follow up for treatment for next 1.5 years. After 1.5 years, the patient reported with a cauliflower like growth of approximately size of 3cm x 3.5cm on the left buccal mucosa which was tender and firm on palpation [Figure 1c]. The indurated margins of the growth were surrounded by Wickham's striae. On the right buccal mucosa there were white radiating lines without any erythema or ulceration. CECT revealed mild asymmetric differentially enhancing soft tissue attenuating lesion measuring approximately 18 x 6mm lesion in left buccal mucosa with obliteration of gingivobuccal sulcus. There was no irregularity of the mandibular cortex [Figure 3]. Incisional biopsy of the growth of left buccal mucosa revealed well differentiated squamous cell carcinoma [Figure 2B].

## DISCUSSION

Oral lichen planus may be asymptomatic or associated with burning or painful sensation or dry mouth. The erosive form tends to be the most painful one. It may undergo spontaneous remission and exacerbations phases. WHO recognizes OLP as oral potentially malignant disorder, but the rate of malignant transformation still not clear. Transformation rate of OLP to OSCC in some review ranged from 0.4 – 5.6% and in another from 0% to 12.5%.<sup>(4,5)</sup> This huge range is because of the different diagnostic criteria used by different studies. After publication of first critical review in 1978, authors concluded that strict diagnostic criteria had to be followed to definitively accept the malignant transformation of OLP patients. With new diagnostic criteria, Krutchkoff et al. found that only 15 out of 223 cases reported in the literature would have been due to malignant transformation in OLP and drew the conclusion that inherent potential of OLP progressing to cancer lacks the evidence, but they accepted that patients with OLP have a slightly higher predisposition to develop carcinomas compared to non OLP patients.<sup>(5,7)</sup> In 1978, WHO gave diagnostic criteria of OLP which included the clinical and histopathological features which was later modified by van der Meiji and van der Waal in 2003 where they included the term OLL and differentiated OLL from OLP clinically and histopathologically.<sup>(6,8)</sup> They also included "absence of dysplasia" in histopathological diagnostic criteria. Our case presented in the report followed the strict diagnostic criteria of OLP.

Van der Meij et al followed strict diagnostic criteria and found that none of the OLP case transformed to malignancy but 4 cases of OLL developed to SCC. In literature also, it is found that the oral lichenoid lesions (OLL) (2.43, 3.2% & 2.5%) has higher malignant transformation rate than OLP (1.47, 1.09% & 0.9%).<sup>(2,3,4)</sup> Certain sites have chances of higher predilection of malignancy, such as the tongue, followed by the buccal mucosa.<sup>(2,3,4)</sup> Case report included case from the 2nd most common site having more predilection of developing the OSCC. Due to overlapping features of OLP and OLL and also the dysplastic lesions with "lichenoid" inflammatory infiltrate, makes prediction of the malignant potential of OLP challenging. There are certain theories behind the malignant transformation of OLP to OSCC. Widely-accepted one is the production of nitric oxide (NO) by inflammatory cells within the lesion, which later reacts with O<sub>2</sub> to induce the formation of 8-dihydro-2 $\epsilon$ -doxyguanosine (8-oxodG).<sup>(9,10)</sup> which further cause G-T nucleotide transversion, promoting

carcinogenesis.<sup>(11)</sup> Also, activity of cyclo-oxygenase II enzyme within the infiltrating inflammatory cells, increasing the release of inflammatory cytokines and malondialdehyde, a carcinogenic metabolite that is known to cause DNA damage.<sup>(12)</sup> Some studies reported high proliferation rate of the basal epithelial cells within OLP lesions which was found to be higher in patients who later developed OSCC than those who did not.<sup>(13-16)</sup> OSCC may present like an ulceroproliferative lesion with indurated margins or hyperkeratotic, exophytic mass or as a sub mucosal, slightly indurated red lesion with an intact epithelium. Therefore, it is difficult to distinguish dysplastic areas from the ulcerative and hyperkeratotic areas of OLP. Hence, earlier identification of dysplastic changes would increase the chances of a successful intervention to reduce the chances of development of an oral cancer from pre-existing OLP. Differentiation of Lichenoid dysplasia and OLL from the OLP need to be done on the basis of clinical and histologic findings critically. Therefore, thorough regular oral examinations are recommended in OLP patients to prevent at the earliest the event of malignant transformation.

## CONCLUSION

Risk factors which trigger or aggravate the OLP need to be reduced to prevent the risk of developing the OSCC. Also, the risk of malignant transformation in OLP patients may be reduced by the removal of carcinogens, elimination of irritating factors, treatment of lesions and intake of a healthy diet including fresh fruit and vegetables. Regular follow up with rebiopsy of the patient is a must in cases of erosive or atrophic form of OLP to keep a check on the malignant changes.

## REFERENCES

1. Shirasuna K. 2014. Oral lichen planus: Malignant potential and diagnosis. *Oral Science International.*, 11:1-7.
2. Giuliani M, Troiano G, Cordaro M, Corsalini M, Gioco G, Lo Muzio L. 2018. Rate of malignant transformation of oral lichen planus: A systematic review. *Oral Diseases.*
3. Aghbari S, Abushouk A, Attia A, Elmarazy A, Meshawry A, Ahmed M. et al. 2017. Malignant transformation of oral lichen planus and oral lichenoid lesions: A meta analysis of 20095 patient data. *Oral Oncology.* 68:92-102.
4. Fitzpatrick S, Hirsch S, Gordon S. 2014. The malignant transformation of oral lichen planus and oral lichenoid lesions: a systematic review. *Journal of American Dental Association.* 145(1): 45-56.
5. Gonzalez-Moles MA, Scully C, Gil-Montoya JA. 2008. Oral lichen planus: Controversies surrounding malignant transformation. *Oral Dis.*, 14:229-43.
6. van der Meij E, van der Waal I. 2003. Lack of clinicopathologic correlation in the diagnosis of oral lichen planus based on the presently available diagnostic criteria and suggestions for modifications. *Journal Oral Pathology and Medicine.* 2003;32(9):507-512.
7. Krutchkoff, D., Cutler, L., & Laskowski, S. 1978. Oral lichen planus: the evidence regarding potential malignant transformation. *Journal of Oral Pathology and Medicine.* 7(1):1-7.
8. WHO Collaborating Centre For Oral Precancerous Lesions. Definition of leukoplakia and related lesions: an aid to studies on oral precancer. *Oral Surg Oral Med Oral Pathol.* 1978;46(4):518-39

9. Chaiyarit P, Ma N, Hiraku Y, Pinlaor S, Yongvanit P, Jintakanon D, et al., 2005. Nitrate and oxidative DNA damage in oral lichen planus in relation to human oral carcinogenesis. *Cancer Sci.*, 96(9):553–9.
10. Yermilov V, Rubio J, Ohshima H. 1995. Formation of 8-nitroguanine in DNA treated with peroxynitrite in vitro and its rapid removal from DNA by depurination. *FEBS Lett.*, 376(3):207–10.
11. Shibutani S, Takeshita M, Grollman AP. 1991. Insertion of specific bases during DNA synthesis past the oxidation-damaged base 8-oxodG. *Nature.*, 349(6308): 431.
12. Mignogna MD, Fedele S, Russo LL, Muzio LL, Bucci E. 2004. Immune activation and chronic inflammation as the cause of malignancy in oral lichen planus: is there any evidence? *Oral Oncol.*, 40(2):120–30.
13. Maidhof R, Hornstein OP, Bauer G, Schönberger A, Bäurle G. 1981. Cell proliferation in lichen planus of the buccal mucosa. *Acta Dermato-venereologica.* 61:17.
14. Valente G, Pagano M, Carrozzo M, Carbone M, Bobba V, Palestro G. et al., 2001. Sequential immunohistochemical p53 expression in biopsies of oral lichen planus undergoing malignant evolution. *J Oral Pathol Med.*, 30(3):135–40.
15. González-Moles MA, Bascones-Ilundain C, Montoya JG, Ruiz-Avila I, DelgadoRodríguez M, Bascones-Martinez A. 2006. Cell cycle regulating mechanisms in oral lichen planus: molecular bases in epithelium predisposed to malignant transformation. *Arch Oral Biol.*, 51(12):1093–103.
16. Taniguchi Y, Nagao T, Maeda H, Kameyama Y, Warnakulasuriya K. 2002. Epithelial cell proliferation in oral lichen planus. *Cell Prolif.* 35:103–9.

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