CASE REPORT
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ORAL LICHENOID REACTION TO ORAL SQUAMOUS CELL CARCINOMA: A CASE REPORT

Oral Medicine & Radiology

Dr. Anuradha Yadav* Associate Professor Oral Medicine And Radiology I.T.S Dental College, Ghaziabad *Corresponding Author

Dr. Parul Tikoo Senior Lecturer Department Of Oral Medicine And Radiology I.T.S Dental College, Ghaziabad Up – 201206

Dr. Smriti C. A Post-graduate Student, Department Of Oral Medicine And Radiology I.T.S Dental College, Ghaziabad Up – 201206

Dr. Nandini Goswami Post-graduate Student, Department Of Oral Medicine And Radiology I.T.S Dental College, Ghaziabad Up – 201206

Dr. Kajal Malhotra Senior Lecturer Department Of Oral Medicine And Radiology Faculty Of Dental Sciences SGT University, Gurgaon, Haryana

ABSTRACT

A family of oral lesions known as lichenoid reaction shares clinical and histological similarities with oral lichen planus. Lichenoid frequently have a recognized, identifiable initiating factor, in contrast to Lichen planus, which is idiopathic. Unlike lichen planus, this lesion typically exhibits a cause-and-effect link. Triggers for this disease might range from medications to dental materials. In response to a metal ceramic prosthesis, we report a typical case of lichenoid reaction on the maxillary alveolus that, a year later, transformed into cancer.

KEYWORDS

PFM, Metal prosthesis, Oral lichenoid reaction

INTRODUCTION:

A collection of skin conditions collectively referred to as lichen are distinguished by their eruptive skin lesions and are named after the lichen-like algae that grows on rocks. Skin and mucosa are affected by the comparatively common autoimmune illness known as oral lichen planus (OLP). Oral Lichenoid reaction (OLR), which is triggered by external causes that may be topical or systemic, is a comparable clinical and histological lesion.[1]

A number of triggering factors such as restorative materials, graft versus host reaction and broad group of drugs are known to cause Lichenoid reaction [2,3]. Drug interactions resulting in oral lesions clinically similar to OLP were first mentioned in the literature in 1971 by Almeyda and Levantine [4].

With the exception of a substantial ulcerative component and mostly erythematous erosive lesions, the clinical signs of oral lichenoid lesions are identical to those of oral lichen planus. All of these illnesses have white streaks known as Wickham striae, which resemble lichen planus. However, one crucial feature that distinguishes OLP is their unusual stance, specifically the absence of bilaterality in their presentations. There isn't a single clinical trait that can be used to differentiate drug-induced lichenoid reactions from lichenoid reactions caused by other factors. The sole indication in this instance is a history of recent or continuous drug substance exposure.

As a result, a differential diagnosis must be established, considering cases associated to the use of dental materials, where the lesions are found in contact with or very close to the restorative materials, and confirming that the patient has no antecedents of graft-versus-host disease [5].

OLR presents with similar histopathologic features of OLP like hyperkeratosis, Superficial band of infiltrate in the lamina propriae, areas of basal cell degeneration and saw tooth retepegs [6]. Certain variations like presence of a substantial number of plasma cell in the infiltrate along with some acute inflammatory cells like eosinophils and neutrophils differentiate OLR. The number of mast cells are comparatively lesser in Lichenoid reaction than in Lichen planus [7]. Moreover, although both OLP and OLR have been assumed to be potentially malignant, it has been suggested that the one which has the premalignant nature is OLR [8,9,10]. Some of Recent studies have shown rate of malignant transformation of OLR even more than OLP [11].

The purpose of this paper is to present a rare instance of squamous cell carcinoma (SCC), which occurred in a 57-year-old female patient, and that established within an oral lichenoid lesion.

Case report

A 57-year-old female patient presented with a complaint of swelling and ulceration on left side of upper jaw since 1 year. Patient's past medical history was non-contributory. However past dental history revealed that patient had multiple fixed metal ceramic prosthesis about 2 ½ years back followed by which patient developed greyish -white line like lesions along with erythema adjacent to the prosthesis region [Fig.1a and b]. Patient also gave history of pain along with burning sensation in the lesions for which she took symptomatic treatment from general dentist. This resulted in non-resolution of the lesion. Following this, approximately 1 $^{1/2}$ year later she developed ulceration in maxillary left posterior alveolus region for which she consulted private practitioner where scaling and curettage was done. As the ulceration progressively increased in size hence the patient visited us. Any history of deleterious habit was ruled out. On intra oral examination there was Ulcero-proliferative growth on left maxillary alveolus extending antero-posteriorly from 22 -27 region and involving entire gingival region with obliteration of buccal vestibule [Fig.2]. On palpation the lesion was tender with firm and indurated borders. Also on the palatal half left side – White wrinkled plaque like lesion was present which was non-scrapable and non-palpable in the vicinity of same region. However there was no regional lymph node involvement. Bony involvement was ruled out with the help of Orthopantomogram [Fig. 3]. Hence, we arrived at a provisional diagnosis of Peripheral giant cell granuloma with differential of Verrucous Hyperplasia. Following this the patient was then sent for incisional biopsy which revealed it to be moderately differentiated squamous cell carcinoma. Finally, the patient was then managed by Institute's cancer department.

DISCUSSION:

The OLR is a frequent condition, with prevalence of about 2.4% in general population [12]. These lesions occur generally in oral mucosa of adults, mostly in women with average age of 53 years old [13,14]. Similar finding was noted in our report as our patient was 57 year old female.

The diagnosis of OLRs is based on their clinical characteristics and the histological findings [15,16]. However, the sensitivity and specificity of histological diagnosis are very low [17]. In addition, no definitive molecular diagnostic markers have been established to date [18]. Van

der Meij et al. have proposed a modification of the diagnostic criteria of the World Health Organization for oral lichen planus and lichenoid reactions (Table 1) [19]. The diagnostic criteria put forward by Al-Hashimi et al suggests patch test apart from clinical and histopathological presentation that can assist in determining what alternative material can be used. Also, on removal or coverage of causative material, there should be resolution of the lesion [20].

According to Lygre et al the principal cause of OLR was associated with adverse reaction to dental materials, being amalgam fillings as responsible for 84% of the cases. The OLR associated with amalgam restorations can be observed in about 2% of population [21]. Furthermore, there is proof that metallic elements taken from dental cast restorations, including nickel, might alter the expression of several immune factors and, as a result, could be involved in the development of a number of intra-oral (and systemic?) pathological disorders.[22]. In our case also OLR was associated with nickel in the form of metal ceramic prosthesis.

In the pathogenesis of OLR cell mediated immunity plays an important role which is in tune with Lichen planus. Lichen planus is an autoimmune disease where as in Lichenoid reaction there is alteration in immunological mechanism due to external agent. The preliminary step is the interaction of the antigen with the keratinocyte. The local mast cells are triggered by the modified basal keratinocyte. RANTES and other cytokines mediate this process. Mast cells in turn cause T cell activation, which leads release of a number of cytokines. This also causes expression of CD54 expression by keratinocyte and leads to Basement membrane damage [23].

OLR have been studied less extensively than OLP in the literature, however recently in a systematic review it was shown that malignant transformation rate of OLL was 2.43% vs 1.37% for OLP [24]. A possible explanation of this could be that chronic inflammation would play an important role in the possible carcinogenesis of this process, causing genetic damage and inducing tissular proliferation. An increase in the production of cytokines and growth factors may aid in the progression of oral cancer.

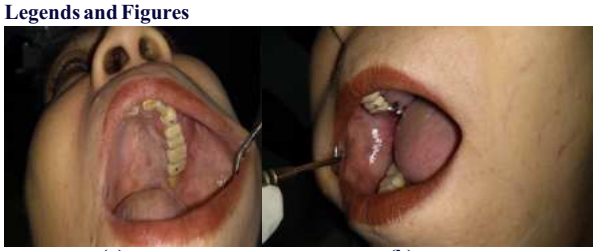
Chronic inflammation produces oxidative damage of the DNA by products derived from inflammatory induced enzymes, such as nitric oxide synthase (iNOS). Another inducible inflammation enzyme is cyclooxygenase-2 (COX-2) that acts inhibiting apoptosis of the keratinocytes and in so doing, facilitating carcinogenesis. In recent years, the process of malignant transformation of the lichenoid lesions has been related to a possible “field cancerization” phenomena, by which all associated events would predispose these patients to a greater risk of multiple and/or multifocal neoplastic malignancies in the oral cavity [25].

Therefore, it is essential to cautiously assess any symptoms or changes in the lesion's homogeneity at each visit, as these could be indicators of transformation. To rule out any malignant alterations, a subsequent biopsy should be performed if there are any changes in the clinical appearance.

CONCLUSION:

The potential risk of malignant transformation in OLR makes this entity of more clinical significance and thereby increases the need of proper understanding of its diagnosis and management. It is important, however, that every dentist and dental auxiliary be educated to detect early signs of oral cancer to ensure that these lesions are identified when the patients are seen for other purposes, such as routine examination and treatment. Also, It is mandatory to schedule meticulous long-term follow-ups, even in patients with asymptomatic or barely symptomatic oral lesions of lichen planus as well as oral lichenoid reactions to prevent these malignant transformations.

Legends and Figures



(a) (b)

Fig 1 a and b – PFM and metal prosthesis present in maxillary arch. White plaque non-scrapable lesion on left half of the palate adjacent to prosthesis.

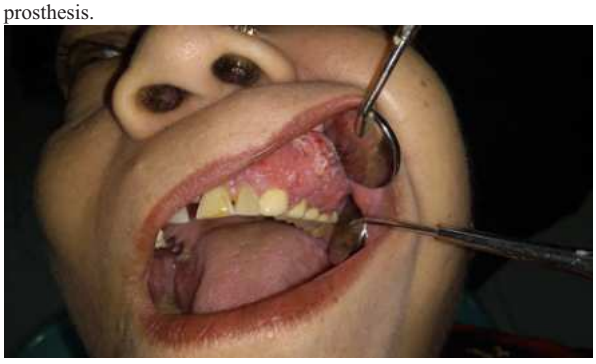


Fig 2 – An ulceroproliferative growth present on left side of maxilla extending from left lateral incisor to second molar region with evident buccal vestibule obliteration.

Fig 3 – Orthopantomogram shows no evidence of bone loss with respect to the lesion.

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