

CASE REPORT

ORAL LICHENOID REACTIONS: A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Oral Lichenoid Reactions (OLR) are clinical and histological contemporaries of Oral Lichen Planus (OLP) often identical in manifestations. The benchmark of differentiation between the two groups is the association of the former with known inciting factors, which when identified and eliminated, often cause a regression of the lesion. This may not always be so and the differentiation then becomes more difficult. A detailed history helps in delineating the lichenoid reaction from lichen planus. The article intends to provide a case report on lichenoid reaction and provides a review into the clinical, histological features and the malignant potential of lichenoid reactions. (2017, Vol. 01; Issue 01: Page 37 - 41)

Keywords: Lichenoid reaction, Lichenoid lesion.

Abbreviations: Oral Lichenoid Reaction (OLR), Oral Lichen Planus (OLP), Oral Lichenoid Lesion (OLL), Graft Versus Host Disease (GVHD)

INTRODUCTION

The terms oral lichenoid reactions (OLRs) or oral lichenoid lesions (OLLs) refer to lesions histologically and clinically similar to oral lichen

planus (OLP), though with the particularity that in these cases the underlying cause is identifiable (1). Pinkus in 1973, published the first microscopic description of these reactions (2). In 1986 Lind employed

the term Lichenoid Reactions (LR) to refer to clinical lesions related with amalgam restorations (3). This mucosal reaction is seen commonly in tobacco products users also. A number of triggering factors such as restorative materials, graft versus host reaction and broad group of drugs are known to cause Lichenoid reaction (4, 5). Lichenoid reactions have been classified based on their etiology into four types by Van der Wall depending on the triggering factors- lichenoid reaction associated with amalgam restoration, drug related lichenoid reaction, lichenoid reaction associated with graft versus host reaction, lichenoid reaction unclassified which cannot be categorized into any of these (6).

CASE REPORT

A 30 years female patient reported to the department of Oral Medicine and Radiology with a chief complaint of burning sensation on having food in left posterior buccal mucosa past 4-5 month. The burning sensation has started gradually and used to occur on having spicy foods. Patient used to get relief on taking frequent sips of water. Past 1 month the burning sensation has progressively increased and is occurring on having any food. There was no history of any deleterious habit. The patient had undergone amalgam restoration in 36 approximately 8 months back. Patient has not changed any tooth powder or paste recently. Medical history did not reveal anything significant.

On clinical examination, a diffuse white keratotic area measuring 3cm x 2cm in dimension with numerous white striations and few erythematous areas is seen in left posterior buccal mucosa extending from 36

region to retromolar area and superiorly from upper to lower buccal vestibule. The surrounding mucosa appears normal. On palpation the white keratotic lesion is non tender and non scrapable. Considering the history and the intraoral examination, the provisional diagnosis of lichenoid contact reaction was made. The patient was advised routine blood investigations and after taking patient's consent incisional biopsy was done. Histopathological investigations revealed hyperparakeratinized hyperplastic epithelium with saw tooth rete ridges and juxta epithelial bands of chronic inflammatory cells chiefly lymphocytes. Basal cell degeneration and Max-Joseph spaces were also evident. After correlating the clinical and histopathological findings, a final diagnosis of lichenoid reaction was made.



Fig 1A: The profile picture



Fig 1B: The profile picture

The patient was sent to the department of conservative where the amalgam restoration was replaced by a temporary restoration. Patient was prescribed triamcinolone ace-tonide 0.1%. The lesion was healed completely after 2 weeks.



Fig 2: Intraoral picture showing white keratotic area with white striations close to amalgam resto- ration

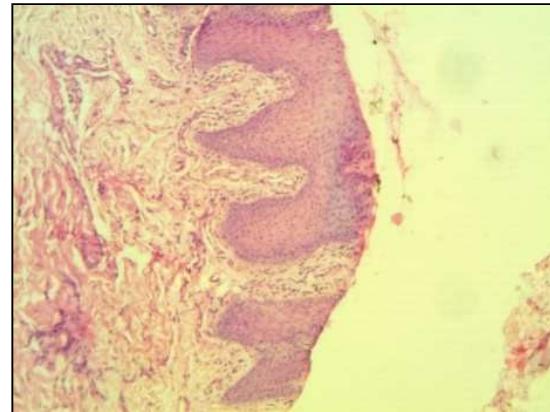


Fig 3. Photomicrograph (20X) showing hyperparakeratinized hyperplastic epithelium with saw tooth rete ridges and juxta epithelial bands of chronic inflammatory cells chiefly lymphocytes. Basal cell degeneration and Max-Joseph spaces were also evident.

DISCUSSION

Lichen planus (LP) is a common chronic mucocutaneous inflammatory disorder of unknown etiology which frequently affects the oral mucosa (7). It was first described in 1869 by Erasmus Wilson as "lichen planus," because the clinical appearance of these lesions is similar to lichens (8, 9). Lichenoid lesions are similar to lichen planus but with known etiology. The first microscopic description of lichenoid reactions was established by Pinkus in 1973 (8). The term oral lichenoid lesion (OLL) was proposed by Finne et al in 1982 (10). The oral lesions that are associated with drug intake, systemic disease (such as chronic liver disease), food or flavor allergies, hypertension and diabetes mellitus are considered as OLL (11). Both the OLP and OLL have the overlapping clinical and histopathological features. However, unlike OLP, OLL resolves after discontinuation of the causative agent (12). Distinguishing these

OLL from one another is also mandatory as some of the OLL, such as graft versus host disease (GVHD) and amalgam associated lichenoid reaction, have a high propensity for malignancy (6).

OLP can often be separated from OLLs to dental materials, which are most often detected on the buccal mucosa and the lateral borders of the tongue. OLP, on the otherhand, usually displays a more general involvement. Oral lichenoid drug eruptions have the same clinical and histopathologic characteristics as OLP. The patient's disease history may give some indication as to which drug is involved, but OLP may not start when the drug was first introduced. Oral GVHD has the same clinical appearance as OLP, but the lesion is usually more generalized (13).

Clinically and histologically it is not possible to distinguish OLP from OLL. OLLs share common clinical and histological features (14). Despite the reported differences between idiopathic LP and lichenoid drug eruptions (LDE), the WHO "gold standard" criteria for LP does not distinguish between the two conditions. OLL are distinguished from OLP by two factors: (1) the association with the administration of a drug, contact with a metal or food stuff or systemic disease and (2) their resolution when the offending agent is eliminated (12, 15). However, the differentiation is not always straight forward.

Treatment includes removal of the causative and offending factors. Patch test may be done wherever applicable and feasible. Topical corticosteroids may be prescribed. Biopsy is advisable. Patient should be under follow up every 3 months for the 1st year after treatment and

then biannually for the next 2 years (16).

CONCLUSION:

The diagnosis of OLL relies on important aspects, such as the clinical appearance of the lesions and the association with adjacent amalgam restorations or history of drugs etc. Both lesions can be very similar, being difficult to distinguish them clinically and histopathologically. However, OLLs are observed in intimate contact with the amalgam restorations, being more localized than OLPs. Although OLL-related conditions present low prevalence in the oral mucosa, they can cause significant discomfort for the patient. Therefore, dentists should be aware of their occurrence, diagnosis and treatment. However, the differentiation is not always straight forward.

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