CASE REPORT

Amalgam-induced oral lichenoid lesion: Case report
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Abstract

Dental amalgam has served as an excellent and versatile restorative material for many years. However, some patients with amalgam restorations may develop hypersensitivity to one of compound of amalgam. Clinically, it presents as an oral lichenoid reaction affecting oral mucosa in direct contact with amalgam restorations. Replacing the amalgam restoration with resin composite lead in almost cases to complete healing of the lesion. We report a case of complete clinical healing of amalgam-induced oral lichenoid lesion after replacing amalgam restoration with resin composite. Unfortunately, delayed relapse was noticed 18 months later on follow-up.

Keywords: lichenoid Eruptions, dental Amalgam, mouth diseases, skin tests.

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Introduction

Dental amalgam has been used as a material for dental restoration for more than one century due to its clinical success and longevity. (1) Nevertheless, the constituents of amalgam may be slowly released over long period of time, leading sometimes to adverse reaction. (2) Most commonly, it presents as an oral lichenoid lesion (OLL) affecting oral mucosa, representing a delayed type IV cell mediated immune response to mercury or one of the other constituents of the dental amalgam. (3) The term of oral lichenoid lesion was proposed by Finne et al in 1982 and is currently used to define lesions with histological features indistinguishable from those found in oral lichen planus (OLP). (4) The clinical features associated with OLL may vary considerably, from white linear plaques, either associated with erythema and erosions or not, to homogenous white plaques. In additions, more than one form can be present concurrently. (5) Symptomatologically, these lesions can range from a subjective discomfort to severe pain. (5) Besides, OLL do not migrate, and they involve only the oral mucosa directly in contact with dental amalgam restorations which is a differential diagnosis from the true lichen planus. (5) The removal of such restorations by replacing amalgam with composite resin has been recommended. It offers a simple, esthetic and conservative approach for treatment of OLL. (3) In the present paper, we report a case of an amalgam-induced OLL with an early complete clinical healing three weeks after replacement of old amalgam restoration with composite resin. Unfortunately, we noticed a relapse on follow-up after 18 months.

Case Report

A 26 years old female was referred to our department with a chief complaint of burning sensation in her
mouth with gradual increasing of the symptoms. Her medical history was unremarkable unless a pollen allergy. The intraoral examination revealed a unilateral reticular white lesion around an erosive erythematous area located on the right cheek mucosa. The lesion was proximate to an amalgam restoration in the first right mandibular molar (figure1). The occlusobuccal amalgam was correctly made with no signs of hiatus or corrosion and the tooth responded normally to the cold vitality test (figure2). Meticulous exploration of the injured zone showed it’s projection on the amalgam restoration, making direct contact on mandibular rest position. Otherwise, the patient presented an overall good oral health condition.

Based on the clinical appearance and on the proximity of amalgam restoration, the diagnosis of an OLL was made and the replacement of the amalgam restoration by a composite resin was planned. Inferior alveolar nerve block was performed using 3% mepivacaine (Medicaine, Médis, Tunisia) and the removal of amalgam was done using transmetal bur 25 mm (dentsply maillefer, York, PA, USA). Then the cavity was filled with glass ionomer (PR Glass Ionomer, Pierre Rolland, Mérignac, France) as a temporary restoration. Clinical exam after three weeks showed complete clinical healing of lesion (figure3). Furthermore, the patient was no longer complaining about the burning sensation. Finally, open sandwich technique restauration was placed (Nextcomp ,Meta Biomed, Chungbuk, Korea) (figure 4).

To confirm our diagnosis a skin patch test (Finn Chamber®, Epitest Ltd Oy, Tuusula, Finland) was performed revealing a strong positive reaction to amalgam (figure 5).
After one year, clinical exam showed no sign of recurrence with clinically normal aspect of the buccal mucosa. But six months later the patient developed a slight raised lesion with reticular pattern exactly on the same location (figure 6). The patient didn’t report any symptomatology. This was consistent with partial relapse of the OLL. The patient is still under regular clinical follow-up twice a year.

Discussion

Oral lichenoid lesions are chronic inflammatory conditions that involve oral mucosa which are similar to OLP and that do not gather the entire typical clinical and pathological criteria. In fact, discrimination between OLP and OLL has always been a major challenge for both clinicians and pathologists (Table 1).

**Clinical criteria**

- Presence of bilateral, more or less symmetrical lesions
- Presence of a lacelike network of slightly raised gray-white lines (reticular pattern)
- Erosive, atrophic, bullous and plaque-type lesions are only accepted as a subtype in the absence of reticular lesions elsewhere in the oral mucosa

In all other lesions that resemble OLP but not complete the aforementioned criteria the term ‘clinically compatible with’ should be used.

**Histopathological criteria**

- Presence of a well-defined band-like zone of cellular infiltration that is confined to the superficial part of the connective tissue, consisting mainly of lymphocytes
- Signs of ‘liquefaction degeneration’ in the basal cell layer
- Absence of epithelial dysplasia

When the histopathological features are less obvious, the term ‘histopathologically compatible with’ should be used.

**Final diagnosis OLP or OLL**

To achieve a final diagnosis clinical as well as histopathological criteria should be included.

A diagnosis of OLP requires fulfillment of both clinical and histopathological criteria OLL. The term OLL will be used under the following conditions:

1. Clinically typical of OLP but histopathologically only ‘compatible with’ OLP,
2. Histopathologically typical of OLP but clinically only ‘compatible with’ OLP,
3. Clinically ‘compatible with’ OLP and histopathologically ‘compatible with’ OLP

Table 1: proposal for a set of modified WHO diagnostic criteria of oral lichen planus and oral lichenoid lesions (vander meij 2003)
The clinical appearance of OLL may mimics the OLP features and be presented as slightly raised white lines, erosion, erythema or plaque-like lesion. In addition, more than one form can be present concurrently. (5) However, the erosive form of OLL was reported more frequently than reticular forms (8). The amalgam-induced OLL are mainly located in direct contact with amalgam restoration which explains that the majority of OLL were reported to be on the buccal mucosa and on the lateral border of tongue, less frequently on the gingiva, the lips, the palate and the floor of the mouth. Furthermore the histopathological criteria established to differentiate OLP from OLL (table 1). Martin H and al. noticed the presence of a deep inflammatory infiltrate, focal perivascular infiltrate, plasma cell or eosinophil in amalgam-induced OLL compared with classic OLP. Moreover, Reddy and al. showed significant increase of degranulated mast cell, eosinophil densities and number of capillaries in oral lichenoid reaction compared to oral lichen planus. (6) Although the direct immunofluorescence is almost unspecific in OLL and OLP some differences have been reported. In fact, Anekal and al. found a fibrinogen deposit at the basement membrane zone in both OLP and OLL, but the fluorescence was less intense in OLL. Fibrinogen deposition in the OLP was discontinuous and it occurred in area paralleling liquefaction degeneration. However, in OLL fibrinogen deposition was along the basement membrane zone with more homogenous linear fluorescence or bandlike ragged (9). These histochemical features may help the oral pathologist to distinguish this two conditions. (10)

The mercury hypersensitivity is rare and affects only 3.2% of the population. (4) Despite the accepted biocompatibility of amalgam, some of its compounds can promote adverse reactions such as OLL. Often the allergen is mercury but occasionally the response could be generated by one of other components of amalgam alloy such as copper, tin or zinc.

The pathogenesis of lichenoid reaction seems to be complex, and is still not fully understood. OLL might be assumed to a hypersensitivity reaction in which an excessive manifestation of the immune response to an antigen leads to tissue damage. In fact, the biodegradation of amalgam may release metallic ion that penetrates epithelium and forms complexes with protein. This antigenic material would be processed either by intra epithelial Langerhans’s cells or by macrophages within the lamina propria. (2) Then, basal keratinocytes become the target of cell-mediated auto-immune damage.

At present, a diagnosis of amalgam-induced OLL depends on the following statements (11):

1) The close topographic relationship between the lesion and the amalgam restaration mostly unilateral. (12)
2) Histological features suggestive of OLP/OLL
3) A positive result of the patch test to mercury.
4) Resolution of the lesion following to the removal of the suspected causative amalgam restoration.

Many studies have demonstrated that the replacement of amalgam filling with other materials has resulted in either complete or partial healing of OLL varying from one to 24 months. (5) It is currently well accepted that the combination of positive patch test and a strong physical contact between the lesion and amalgam restoration are the best predictors of clinical healing following amalgam replacement. Although, many OLL not in strict contact with amalgam have been demonstrated to benefit from amalgam removal. (10) Moreover, some studies demonstrate no difference in the healing rate between positive or negative patch test, whereas others reported a significant value of patch testing as a predictive indicator. (10)

There is some literature evidence that clinical healing after amalgam removal does not necessarily means complete disappearance of histological sign related to the OLL. (10) This may explain how in our case the relapse was occurred after complete clinical healing of the OLL. Thus, we recommend a periodic follow-up of the patient even complete disappearance of the lesion.

**Conclusion**

The dental clinician should evoke the diagnosis of OLL in front of any lesion of the oral mucosa next to amalgam restoration. Often, the topographic location and the unilaterality of lesion are sufficient criteria to
establish the diagnosis. However, the patch test to amalgam, although it is not always positive, could help to establish the diagnosis. The replacement of the amalgam with a composite resin restoration is most often accompanied by a complete or partial healing of the lesion.

**References**


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