

Amalgam Contact Hypersensitivity Lesion: An Unusual Presentation-Report Of A Rare Case

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Abstract

Amalgam or its components may cause Type IV hypersensitivity reactions on the oral mucosa. These amalgam contact hypersensitivity lesions (ACHL) present as white striae and plaques, erythematous, erosive, atrophic, or ulcerative lesions. Postinflammatory pigmentation in such lesions and pigmentation due to amalgam incorporation in the soft tissue have been reported in the literature. However, ACHL presenting primarily as a black pigmented lesion is extremely rare if not reported. The clinician should be aware of one such presentation of ACHL; we report a unique case of ACHL in a 30-year-old female with such a pigmented lesion in close contact with amalgam restorations. The lesion regressed considerably in a year after replacement of the restoration with posterior composites.

Keywords: Amalgam, Amalgam contact hypersensitivity lesion, Lichenoid reaction, Oral mucosa

Introduction

Eruptions in the oral cavity having an identifiable etiology that are clinically and histologically similar to oral lichen planus (OLP) are termed oral lichenoid lesions (OLL).^[1] Different terminologies have been used in literature such as contact allergies,^[2] OLL,^[3] contact lesions^[4] or oral lichenoid reactions (OLR).^[5] Pinkus in 1973,^[6] published the first microscopic description of these reactions. In 1982, Finne *et al.*^[7] proposed the term OLR to designate clinically indistinguishable lesions of OLP in which a specific etiological factor can be inferred and/or demonstrated and differentiate it from idiopathic OLP. In 1986, Lind *et al.*^[8] employed the term lichenoid reaction (LR) to refer to clinical lesions related with amalgam restorations. Ever since the concept has been proposed, these lesions have been described as a response to a wide variety of triggering factors and said to involve several clinical types [Table 1].^[9]

Contact allergic reactions due to hypersensitivity to dental materials in professionals and patients have been extensively studied.

Materials such as amalgam,^[10,11] polymethylmethacrylate^[10] and resin composites^[12] have long been identified as allergens in a dental setup. Amalgam is the most widely used dental restorative material. However, because of the continuous low level release of mercury, its safety and wide scale use have been questioned. Laine *et al.* in their immunological studies observed true allergy to mercury.^[3,13] Hypersensitivity to amalgam has been attributed to mercury in amalgam, rarely copper, palladium, silver, tin or zinc and their corrosive by products. The allergic response is either toxic/irritative or allergic in nature. These lesions are most often seen in direct topographic relation to the causative agent, which induces a sensitivity response resulting in immunologically mediated damage to the keratinocytes of the basal layer of an epithelium. It is Type IV/delayed hypersensitivity reaction involving cell mediated immunity primarily macrophages and T lymphocytes. These cells are sensitized to the antigen (haptens) thus triggering the cell mediated response which are directed against the basal keratinocytes.^[11] However the exact mechanism of how mercury or other metallic haptens released from dental materials are capable of triggering the immune response is not known. Bolewska *et al.*^[2] in their study have concluded that these products might give rise to lesions in patients with a higher sensitivity or susceptibility to develop a reaction. Rarely, an acute generalized or systemic reaction occurs in 2-24 h of restoration and resolves 10-14 days of its removal.^[14] Contact hypersensitivity lesions (ACHL) affects 1-2% of the population and adverse effects to dental amalgam is estimated in 1/million population.

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Case Report

A 30-year-old female patient reported with a complaint of a black patch in the right side of the mouth in the cheek region, which she noticed 4 months back. The area felt rough and was associated with mild burning sensation. The patient did not notice any change in the size of the lesion since then. Her medical history was noncontributory and she did not give any history of medication or allergy. Teeth #16 (maxillary right first molar) and #46 (mandibular right first molar) was restored with silver amalgam 2 years back. One month prior to this visit she had her amalgam restoration of 46 replaced with a temporary restoration. Examination of skin and nails did not reveal any abnormalities.

On intra-oral examination, a diffuse black patch was seen on the right buccal mucosa in relation 15, 16, 17, 45, 46, and 47 at the level of occlusion, which was interspersed with whitish areas. The lesion measured about 3 cm × 2 cm and the surrounding mucosa appeared normal. 16 had a Class II (mesio-occlusal) silver amalgam restoration and 46 had a Class II temporary restoration [Figure 1]. The lesion was non scrapable and non-tender. The left buccal mucosa appeared normal. The case was provisionally diagnosed as ACHL. Differential diagnosis of amalgam tattoo, healing phase of lichen planus (LP),

melanoplakia, and melanoma were considered. The silver amalgam restoration was replaced with a temporary restoration. The patient was reviewed after 2 months. There was no change in the size of the lesion and as the patient was worried about it, incisional biopsy was done under local anesthesia and the specimen was sent for histopathological examination. Hematoxyllin and Eosin (H and E) section showed hyperplastic parakeratinized stratified squamous epithelium with acanthosis, basilar hyperplasia and degenerative changes. Connective tissue showed dense inflammatory cell infiltrate predominantly lymphocytes and few plasma cells. Melanophages and melanin incontinence was also seen [Figures 2 and 3]. The histopathological picture was suggestive of LR. Based on the clinical and Histopathological picture, the case was diagnosed as ACHL. The temporary restoration in 16, 46 were replaced with posterior composites. The patient was reviewed periodically for a year and the lesion regressed considerably [Figure 4].

Discussion

Amalgam has always been one of the most widely used restorative materials for posterior teeth. Even today, with the advent of new synthetic non-metallic materials and novel time-saving procedures, silver amalgam is the most widely used and cost-effective dental material in restorative dentistry. Known for its high compressive strength and minimal technique sensitivity, amalgam for long has been used for posterior restorations and core build ups. Reports of hypersensitivity to amalgam are rare. The cause of such low incidence may be that saliva sweeps, dilutes and makes allergens disappear quickly, low mucosal keratinization which makes hapten combination more difficult; high vascularity of the oral mucosa, which eliminates the allergens from the area; and high resistance of the oral mucosa.^[15] High turnover rate of oral mucosal cells may also be a reason.

Amalgam contact hypersensitivity lesions are most often seen in area partially or completely in contact with amalgam. The lesions are most common on the buccal mucosa, lateral

Table 1: Clinical types of OLL^[9]

Clinical types of OLL

OLLC as a result of allergic contact-stomatitis which occurs in direct topographic relation with dental restorative materials, most commonly with amalgam

OLLD in which oral and/or skin lesions appear in temporal association with the ingestion of certain drugs

Oral lichenoid lesions in patients suffering from acute graft versus host disease

lesions that have a lichen planus like aspect, but lack one or more characteristic clinical aspects

OLL: Oral lichenoid lesions, OLLC: OLL related to contact, OLLD: OLL related to drugs



Figure 1: Intra-oral photograph showing diffuse black patch on the right buccal mucosa interspersed with whitish areas. Class II amalgam restoration seen in the right maxillary first molar

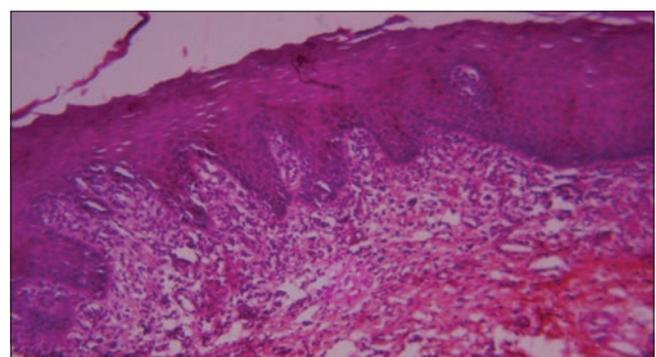


Figure 2: Photomicrograph (light microscopy, x10) showing parakeratinized epithelium with saw tooth rete ridges, basilar hyperplasia with degenerative changes and dense mixed inflammatory cell infiltrate in the connective tissue

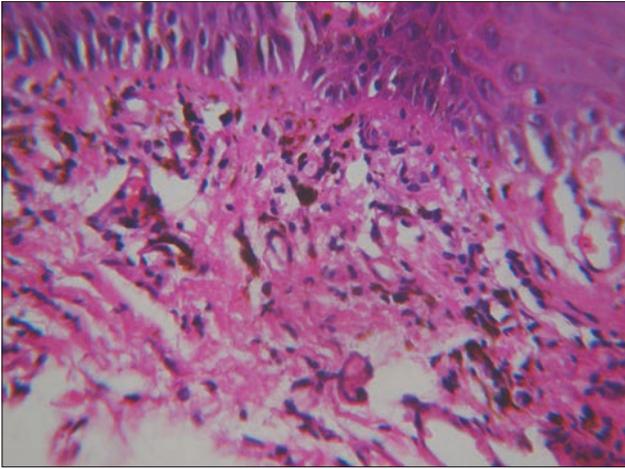


Figure3: Photomicrograph (light microscopy, $\times 40$) showing subepithelial melanin incontinence with inflammatory cells infiltrate

surface of the tongue and less common on the gingiva, lips and floor of the mouth. They present as white striae and plaques, erythematous, erosive, atrophic, or ulcerative lesions.^[16] Contact lesions presenting as an area of hyper pigmentation in the oral cavity is extremely rare and no such case has been reported in the literature. Hence, this makes the case unique and interesting. Such lesions have to be differentiated from oral postinflammatory pigmentation which also present as localized or generalized brown-black pigmentation.^[17] These lesions are associated with chronic inflammatory conditions such as LP, pemphigus and pemphigoid. However, a feature that differentiates these lesions is the presence of acute symptoms such as erythema, white plaques, burning sensation or desquamation, which precedes pigmentation. These features were not seen in our case.

Hyperpigmentation is commonly associated with contact lesions in the skin. According to Rycroft *et al.*^[18] such pigmentation associated with contact dermatitis is classified into three: (1) Hyperpigmentation due to incontinentia pigmenti histologica (IPH), (2) hyperpigmentation due to increase in melanin in the basal cells of the epidermis (basal melanosis), (3) hyperpigmentation due to slight hemorrhage around the vessels of the upper dermis resulting in accumulation of hemosiderin.^[18] Rycroft *et al.*^[18] have stated that when the grade of contact dermatitis is more severe or its duration is longer, secondary hyperpigmentation following dermatitis is more prominent. Manifestation of dermatitis such as erythema, vesiculation, papules or scaling rarely occur in IPH and such patients may complain of only pigmentation, though the disease is a result of contact dermatitis.^[18] The same analogy was seen in our case where the patient's complaint was only pigmentation, which was not associated with any other features of contact hypersensitivity such as erythema, burning sensation, desquamation, etc. This phenomenon of IPH explains the unique presentation seen in our case.

The diagnosis of ACHL is based on criteria suggested by Al-Hashimi *et al.*:^[19] (1) Clinical presentation



Figure4: Intra-oral photograph taken after 1 year showing regression of the lesion except for a small area of residual pigmentation

(2) histological results (3) patch test (4) results of replacing suspected material. The lesions are always in sites, which are in close contact to the amalgam restoration and are asymmetrically distributed. The case reported had a pigmented lesion in buccal mucosa in the region of 16 and 46 and was unilateral. Histopathologically, these lesions have many similarities to LP. van der Meij *et al.*,^[20] and Thornhill *et al.*^[11] have proposed certain histological criteria to differentiate OLL and LP. Our case showed basilar hyperplasia with desquamative changes, melanin incontinence (melanin pigment in the upper part of the connective tissue), lymphocyte infiltrate and plasma cells in the connective tissue. Patch test has been used to detect patient's hypersensitivity toward dental restorative material.^[1] However, Issa *et al.*^[21] have opined that patch test have limited benefit as a predictor of such reactions. Diagnosis of our case was based on the clinical presentation, histopathology and resolution of the lesion after replacement of the restoration.

In our case report, the term LR, ACHL, IPH have been used. The clinical manifestations of all these conditions are similar and all the three are associated with known allergic agents. These terminologies have been interchangeably used in the literature. Hyperpigmentation caused by IPH has been often termed LR due to the similar histopathological features to LP. ACHL is a form of LR specific to contact of the oral mucosa to amalgam. Hence in our case, we have used the term ACHL as the diagnosis.

Replacement of the restorative materials that are in direct contact with the lesion and are suspected of playing a causal role is the most accepted management approach for ACHL. Various clinical studies have found that replacement of amalgam restoration with hypoallergic ones such as composite and gold resolves these lesions within days or weeks.^[2-19] In a study by Thornhill *et al.*^[11] they found that 71.4% of cases had complete resolution in 3-12 months, 21.4%-8-27 months, 3.6% had little improvement after 15 months. The recovery range oscillates between 37.5^[5] and 100%.^[22] Recovery of

lesions is most noticeable when there is direct contact between the lesion and restoration and least when there is no contact. It has also been found that lesions heal when they are not in contact with the restorative material as well. This could be due to parafunctional aspects which may connect lesions and the amalgam fillings.^[5] The amalgam restorations in 16 and 46 were replaced with posterior composites and the patient was followed at regular intervals with the lesion having regressed considerably in about a year.

Conclusion

Available scientific evidence does not justify the discontinuation of the use of amalgam, nor does it recommend the removal and replacement of satisfactory amalgam fillings with other materials. Local allergic reactions are rare, when such lesions do occur, the clinician must be aware of the various clinical presentation including as an area of pigmentation. Diagnosis is made by the presence of an offending restorative material in close contact with the lesion. A wait and watch approach after replacement of the allergic restorative material would suffice. Biopsy and patch test may not be required always.

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How to cite this article: ????

Source of Support: Nil. Conflict of Interest: None declared.