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Intraoral Contact Lichenoid Reaction to Dental Amalgam

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Authors' contributions

This work was carried out in collaboration between all authors. Author SS wrote the first draft of the manuscript. Author NY managed the literature searches, with clinical case description. Author RB performed the restorative aspect and contributed about the same. Author KAB diagnosed the lesion with contribution on histological aspect. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Oral lichenoid reactions are histologically and clinically very similar to oral lichen planus, but differ, as in these cases the underlying cause is particularly identifiable. Various etiological factors for such lesion are dental restorative materials, drugs, food additives, oral hygiene products etc. Most common cause in oral cavity is contact sensitization to dental amalgam restorations which may result in clinical lesion as well as symptoms of burning sensation. Patch tests may identify such

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lesions but are not routinely recommended and hence diagnosis depends mainly on clinico-pathologic correlation. Histopathology has certain limitations due to overlap of findings with oral lichen planus. But with the advent of immuno-histochemical markers this pitfall can also be rectified. Treatment in case of dental material associated lesion involves simply removal/ replacement of the culprit material. We report a case of oral lichenoid reaction due to dental amalgam restoration which was successfully diagnosed and treated.

Keywords: Hypersensitivity; dental amalgam; oral lichenoid reaction; oral lichen planus.

1. INTRODUCTION

Oral lichenoid reactions (OLRs) or oral lichenoid lesions (OLLs) are the terms used to refer those lesions which histologically and clinically are similar to oral lichen planus (OLP), but differ, as in these cases the underlying cause is particularly identifiable [1]. Literature reports many causative factors for such lesions like dental materials, graft versus host disease, food additives, oral hygiene products, drugs, phytotherapy, oral cosmetics and beverages [2,3]. Dental materials used in the oral cavity which act as triggers are, silver amalgam, mercury, gold, nickel, copper, cobalt, palladium, chromium, epoxy resins and composites [4,5]. Among the drugs, most commonly implicated agents are non steroidal anti-inflammatory drugs (NSAIDs) and angiotensin converting enzyme inhibitors (ACEI) [1]. Substances in oral hygiene products (cinnamon acid, menthol, peppermint) and food products (tea, liquors, gum, candies with cinnamon flavor) may also cause oral lichenoid lesions [6]. Some of the predisposing factors are mechanical trauma (Koebner phenomenon), calculus, plaque, rough dental restorations, poorly fitting dental prostheses, tongue, lip, cheek biting and oral surgical procedures. The particularity of these lesions is that when these factors are removed the lesion heals.

The pathogenesis behind OLL due to dental restorative materials is development of delayed hypersensitivity reaction due to contact sensitization to the material. In oral cavity, chemical and electrochemical corrosion occurs with saliva acting as an electrolyte. The resulting galvanic currents and corrosion processes lead to the release of ions and their complexes from metals which may act as haptens and bind with host keratinocyte surface proteins in susceptible individuals resulting in a cell mediated response directed at basal keratinocytes resulting in formation of lesion [7,8]. These lesions are categorized as delayed type hypersensitivity reactions as prolonged intimate contact of the oral mucosa with dental material over a long period is required [9].

OLL are usually present on the buccal mucosa, tongue and lips where there is close topographic relationship between the restoration and the lesion hence these are termed cause-effect lesions [7]. Close topographic relation of OLL to dental amalgam is considered a good prognostic sign [3]. These lesions are clinically very similar to oral lichen planus lesions with mainly reticular, erosive and ulcerative components and characterized by the presence of white streaks i.e. Wickham striae. Other clinical forms described are papules, plaque and bullous types. A significant distinguishing factor with respect to OLP is their typical location (mostly in regions where oral mucosa comes in contact with dental materials), and the absence of bilaterality (unlike OLP) of the manifestations [10]. Symptoms vary from being asymptomatic to burning and pain especially on eating hot and spicy food. Investigations include patch testing and biopsy but the drawback is that histopathology is not very confirmatory for these lesions due to overlap with OLP lesions and patch testing is not routinely done or not agreed upon by patients.

2. CASE REPORT

A 38 year old male patient reported with complaint of burning sensation in right cheek and right side of tongue on eating hot and spicy food since one year. History for any associated skin lesions and drug intake was negative. He had consulted physicians and had already been prescribed antifungal ointment (Orasep OT) use for one week, chlorhexidine mouthwash for two weeks and topical application of steroid triamcinolone acetonide (kenacort) (0.1%) for two weeks. He gave history of decrease in burning sensation on using kenacort ointment but return of symptoms once the ointment was discontinued. Intra-orally right buccal mucosa revealed a linear, non scrapable, white lesion measuring approximately 2.5x1 cm in size at the level of occlusal plane in relation with teeth 25 and 26. At the periphery of the lesion radiating

white striae were present (Fig. 1). A non scrapable white patch conforming to scallops of lateral border was present on right side of tongue. Teeth 14 and 26 were missing and 16 had dental amalgam restoration which was done 6 years back. Provisional diagnosis of oral lichenoid lesion of right buccal mucosa (reticular type) and right lateral border of tongue (plaque type), and differential diagnosis of oral lichen planus was considered in consultation with an oral pathologist. Incisional biopsy of area under low power revealed parakeratinised atrophic stratified squamous epithelium and connective tissue (CT) showing juxta-epithelial band of inflammatory cells (Fig. 2). Further high-power view showed cellular infiltrate in lamina propria chiefly of lymphocytes, plasma cells and few histiocytes (Fig. 3). All the histopathological features were mimicking to oral lichen planus. Patch testing for dental amalgam and mercury was advised to which the patient did not agree. Hence, to rule out the allergic response immunohistochemical (IHC) staining for langerhans cells which act as chemical mediators for inflammatory response was done. CD1a marker for langerhan cells showed immunopositive staining in suprabasal, spinous layer and lamina propria region (Fig. 4). Final diagnosis of 'oral lichenoid lesion due to contact sensitivity to dental amalgam restoration in relation with teeth 26' was considered and the restoration was replaced with composite material after obtaining patient's consent. Tooth was isolated with a rubber dam during the removal of amalgam. A new bur was used to remove the restoration using airotor hand piece with copious water outlet and high volume suction was simultaneously used to evacuate the working area. Filtek Z250XT (3M ESPE), a nanohybrid composite was used along with Adper Easy Bond (3M ESPE), a self-etch adhesive for replacing the amlgam restoration. Within 5 days burning sensation disappeared along with 100 % healing of lesions of buccal mucosa and tongue (Fig. 5).

3. DISCUSSION

OLLs can be caused by dental amalgam and main causative factor identified is mercury vapors. Release of mercury vapor has been reported during insertion, condensation and carving of amalgam restoration. Similarly mercury as vapor or as salt dissolved in saliva can be found during eating and chewing in patients who have amalgam restorations in oral cavity. Studies have shown that the amount released directly correlates to the amount of amalgams present and their total surface area. The daily absorbed dose of mercury from amalgam for the average individual is 1.2 µg by inhaled mercury and 1.5 µg by ingested mercury. Other corrosive products in amalgam are copper and tin [11,12]. Our patient had one silver amalgam restoration in relation with tooth 16 which was done 6 years back.

There are studies assessing the contact hypersensitivity of OLL to various dental materials particularly amalgam and some have used patch testing to confirm the diagnosis. A study conducted on 25 patients with the same aim found that 15 (60%) patients showed sensitization to one or more allergens, and greatest frequency of positive reactions was particularly to mercury and amalgam They also concluded that contact sensitization was involved in the pathogenesis of lichenoid manifestations in the oral cavity [13]. Isaac van der Waal described four types of oral lichenoid lesions (OLLs) as:

1) Amalgamrestoration, topographically associated OLL
2) Drug related lichenoid lesions
3) Lichenoid lesions in chronic graft versus host disease (cGVHD)
4) OLL, unclassified (e.g. erythematous changes limited to the gingiva without signs of "classic" OLP elsewhere in the oral cavity, or lesions that have a lichen planus like aspect but that lack one or more characteristic clinical features, such as bilateral presentation) [14].

Investigation such as patch testing is useful to identify patients with suspected hypersensitivity reactions to amalgam or mercury. This is done by using commercially available kits which are placed on the skin of the back or forearm and held in place for 48 hours with hypoallergenic adhesive tape. It is generally accepted that 5% amalgam and 1% ammoniated mercury are suitable for screening. The test results are generally read at 48 and 72 hours but evidence has shown that late readings at 10-14 days can diagnose previously missed positive reactions. A skin reaction with erythema and eczema reaction is considered positive. Routine use of patch testing for all patients with lichen planus like lesions is not recommended due to the possibility of false positive and false negative results [13]. Our patient refused to undergo patch testing probably due to the fear of unknown.

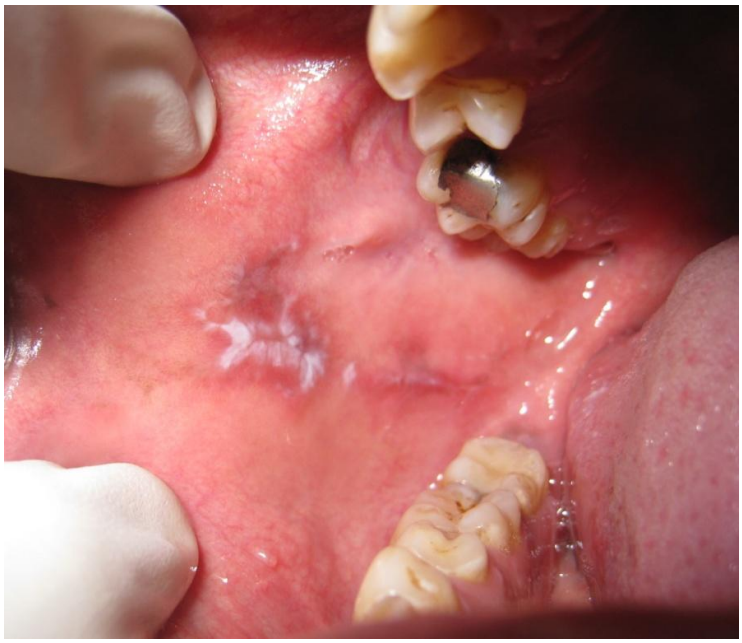

Fig. 1. Linear, white lesion, at the level of occlusal plane in relation with teeth 26 (restored with dental amalgam) with white striae at the periphery

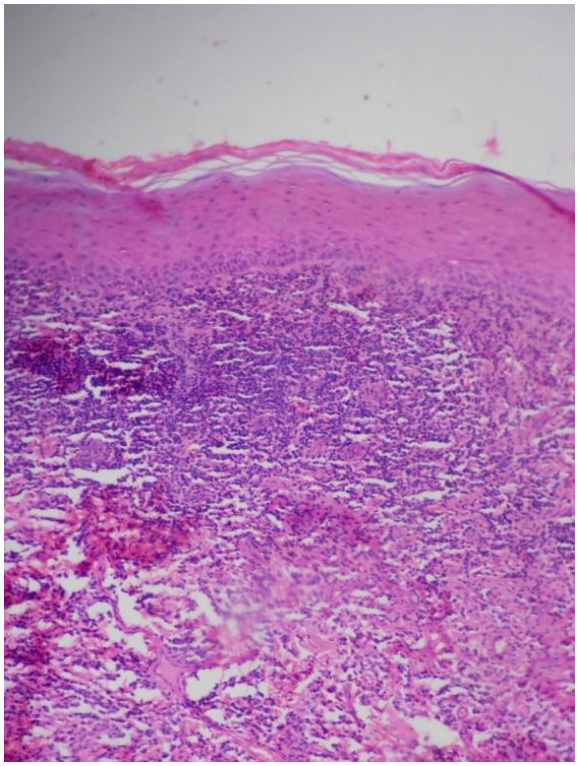

Fig. 2. Photomicrograph of H&E showing stratified squamous epithelium and connective tissue showing Juxta-epithelial band of inflammatory cells

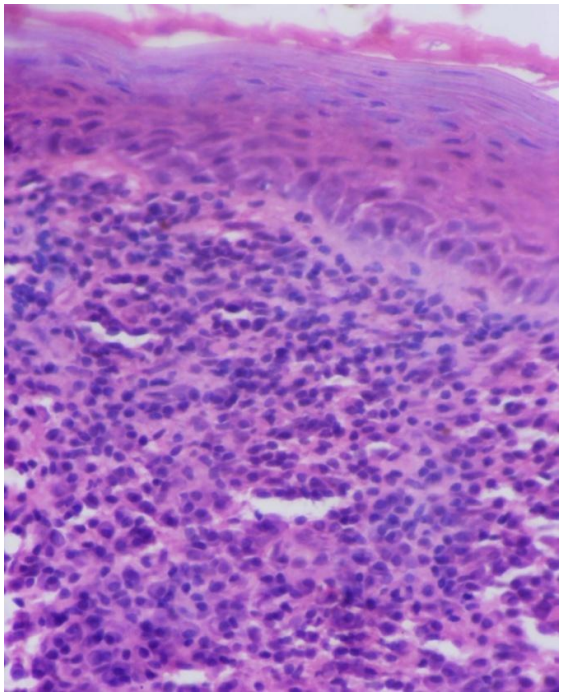

Fig. 3. Photomicrograph of H&E in high power cellular infilterate in lamina propria chiefly of lymphocytes & plasma cells

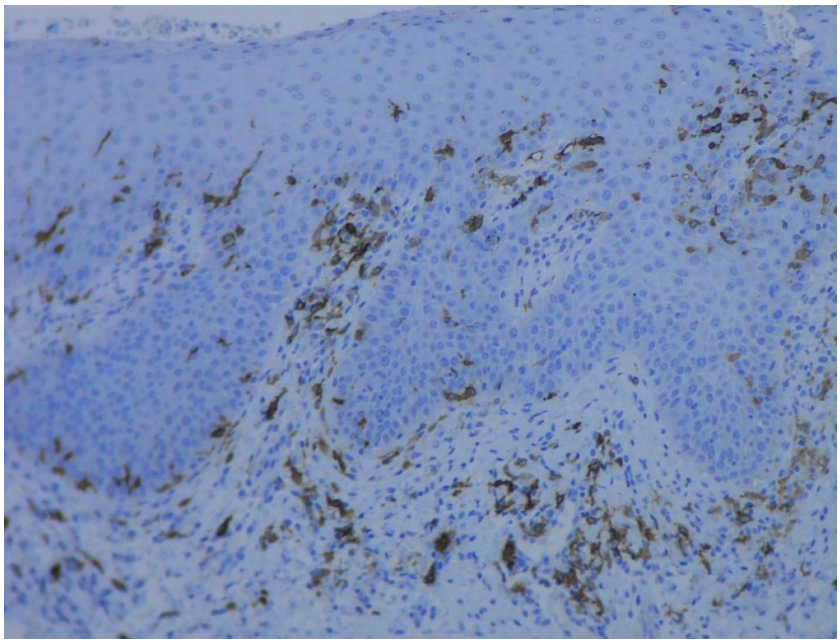

Fig. 4. CD1a Immunopositive staining in suprabasal, spinous layer and lamina propria region

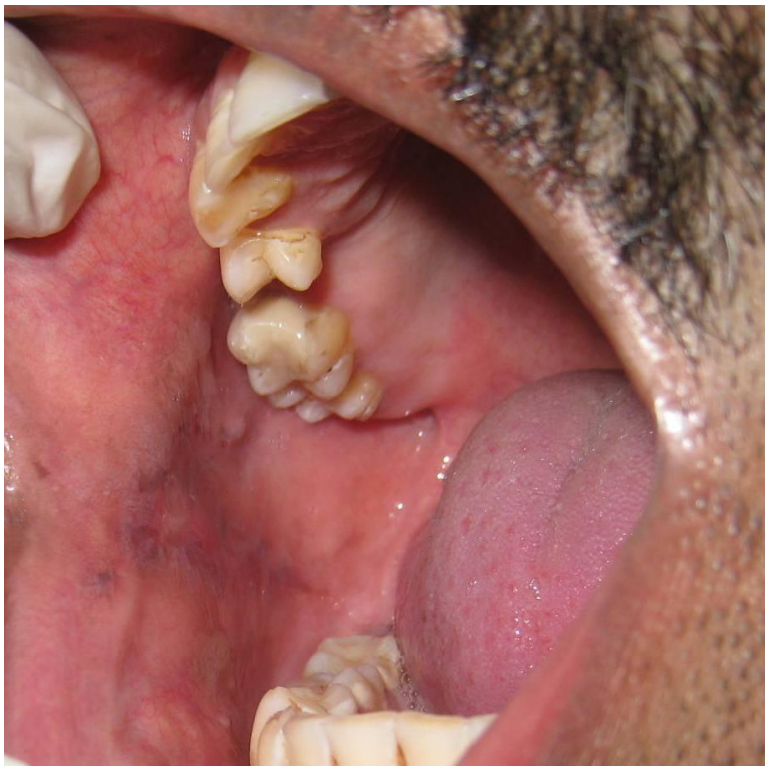

Fig. 5. Post treatment clinical picture of right buccal mucosa

Modified World Health Organization (WHO) criteria for the diagnosis of oral lichenoid reactions (OLRs), oral lichenoid lesions (OLLs) and oral lichen planus (OLP) has been proposed by Van de Meij et al. Histopathological changes are similar to oral lichen planus but it has been suggested that a mixed cell subepithelial infiltrate and a deeper diffuse distribution in lamina propria can help to distinguish a lichenoid lesion from OLP [15]. Histopathology of incisional biopsy of our patient revealed parakeratinised atrophic stratified squamous epithelium and connective tissue (CT) showing juxta-epithelial band of inflammatory cells. CT showed cellular infiltrate in lamina propria chiefly of lymphocytes, plasma cells and few histiocytes. Subepithelial infiltrate and a deeper diffuse distribution of inflammatory component in lamina propria were also seen in our case. Differential diagnosis of OLLs includes OLP but OLP is a more widespread condition involving many anatomical sites in the oral cavity (usually bilateral) and also with concurrent skin lesions, whereas OLLs are sharply demarcated, are unilateral and have a clear anatomical relationship to the dental restorations, hence commonly seen on the buccal mucosa and tongue.

The management requires identification of the triggering factor, and the elimination of exposure to it. In the case of drug induced OLRs, evaluation of the risk / benefit ratio of suspending the medication is required. After stoppage or replacement of causal medication the lesions may take several months in improving. In case of lesions related to dental materials, reversal of lesions following removal/replacement of contact allergen is reported [3]. Some authors found a good response to replacement of dental amalgam restorations in patients with positive patch test reactions [4,16,17,18]. Dunsche A [19] described regression in of lichenoid changes after amalgam substitution in 97% of 134 patients, independent of results of patch testing. Premalignant potential of these lesions has been reported [2,20]. In one study 10 out of 11 patients with intraoral carcinoma, patch tests revealed hypersensitivity to gold, mercury, silver and copper [21].

4. CONCLUSION

With an increase in awareness of mercury toxicity people who seek preventive dental care are on a rise. There is already an established

data that amalgam is capable of causing lichenoid reaction. A careful clinical evaluation and its co relation to existence of lichenoid reaction adjacent to a huge amalgam restoration is the key to successful treatment plan and its execution. Immunohistochemical marker's introduction takes care of any pitfalls in diagnosing such cases.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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