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Oral Lichenoid Contact Lesions - Clinical Presentations and Dental Implications

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Abstract

Oral lichenoid contact lesions (OLCLs) typically present in direct topographic relationship with restorative materials. OLCLs present clinically similar to oral lichen planus (OLP) with features ranging from patches or plaque-like lesions to large, painful ulcerative/erosive lesions. However, lesions are distinct from OLP due to their asymmetric unilateral presentation and their relationship with dental restorative materials. Most lesions consist of white, linear, parallel, and non-elevated streaks that radiate from a central erythematous area adjacent to dental restorations. There is sufficient evidence for an increased risk of malignant transformation of OLCLs according to the latest classification of oral potentially malignant disorders. Therefore, recognition of mucosal changes related to lichenoid inflammation is paramount. Dental implications and clinical significance are highlighted by the potential for malignancy, necessitating careful management and consideration of alternative restorative materials. We present the clinical presentations of OLCLs and highlight dental implications relevant to the overall management of affected patients.

Keywords: Oral Lichenoid Lesions; Dental Materials; Patch Testing; Oral Potentially Malignant Disorders

Introduction

A lichenoid reaction is a response that occurs in the oral cavity with specific clinical and histopathological features. Oral lichenoid contact lesions (OLCLs) typically present in direct topographic relationship with restorative materials, most commonly amalgam. An amalgam restoration is commonly seen adjacent to a lichenoid lesion, suggesting that when the surface metals including silver, mercury, tin, copper, and zinc oxidize, they represent a tarnished metal surface which in turn impart a contact allergy [1]. Other dental materials reported in the literature such as resins and acrylate polymers may also induce lichenoid lesions [2]. There is sufficient evidence for an increased risk of malignant transformation of OLCLs according to the latest classification of oral potentially malignant disorders (OPMDs) [3]. Therefore, recognition of mucosal changes related to lichenoid inflammation is paramount. This review aims to illustrate the clinical presentations of OLCLs and highlight the dental implications relevant to the overall diagnosis and management.

Clinical Presentations

OLCLs present clinically similar to oral lichen planus (OLP) with features ranging from patches or plaque-like lesions to large, painful ulcerative/erosive lesions. However, lesions are distinct from OLP due to their asymmetric unilateral presentation and their relationship with dental restorative materials. Most lesions consist of white, linear, parallel and non-elevated streaks that radiate from a central erythematous area adjacent to dental restorations (Figures 1 and 2). The white streaks do not overlap or crisscross as in typical OLP and they cannot be scraped off. In some cases, lesions may present with less erythema (Figure 3), or as a predominantly white

plaque-type lesion (Figure 4). The differences in clinical presentations may be attributed to the duration of contact and type of material. Additionally, the proximity between the mucosal tissue involved with the offending material plays a role, for example, patients with thick, puffy, or prominent cheeks show a more severe lichenoid reaction. Amalgam is commonly involved in direct topographic relationship with OLCLs (Figures 5 to 7), however composite restorations are also seen to be a contributing factor for the development of these lesions (Figure 8). Patients with OLCLs may present with or without symptoms. Symptoms include burning sensation and discomfort while eating. Some may report spontaneous pain or soreness from the involved region, which may indicate active inflammatory response to the offending dental material.

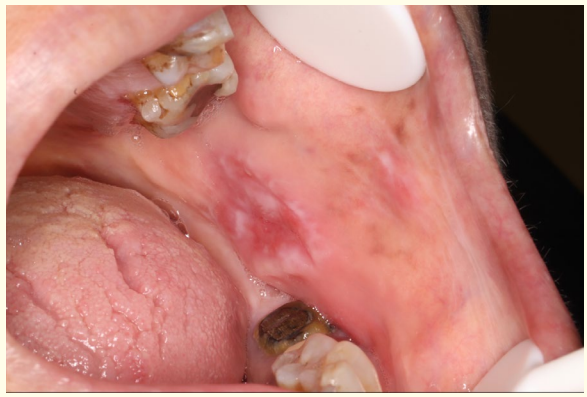
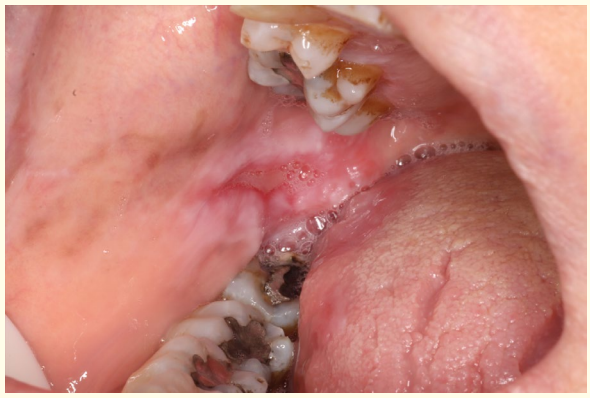


Figure 1



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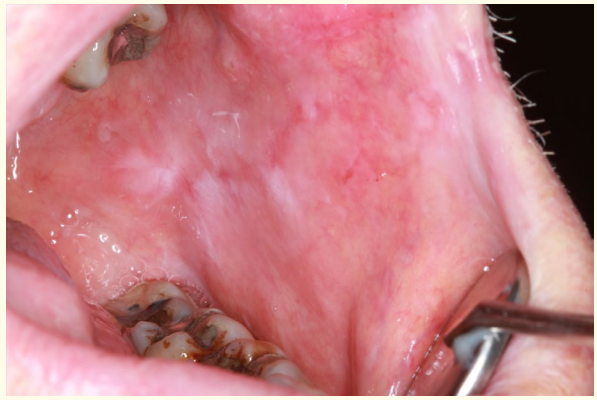

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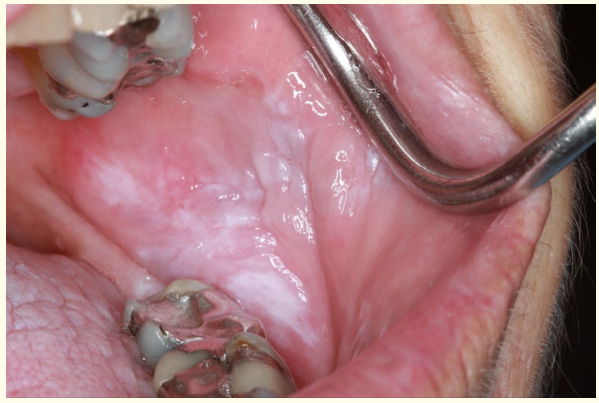

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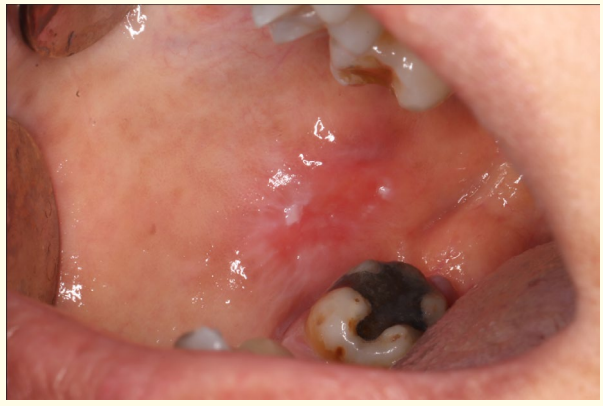
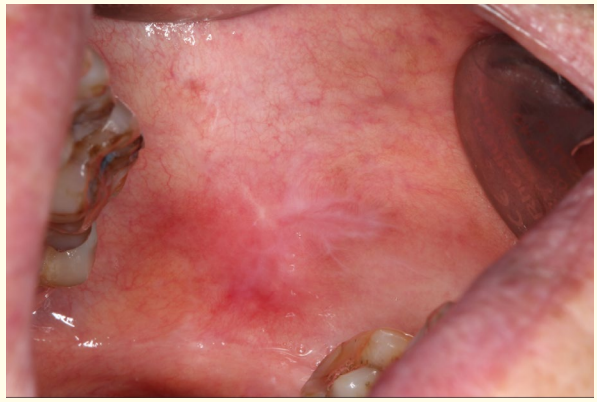


Figure 5



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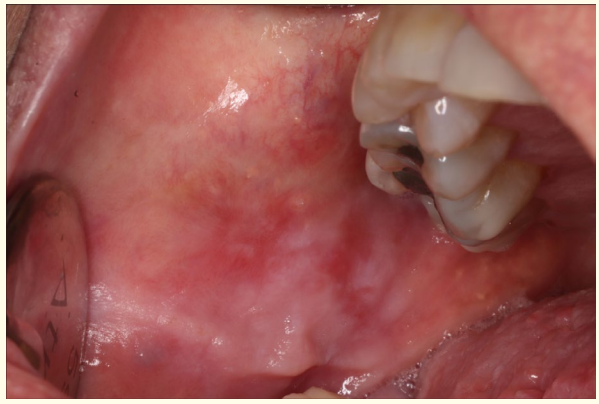

Figure 7

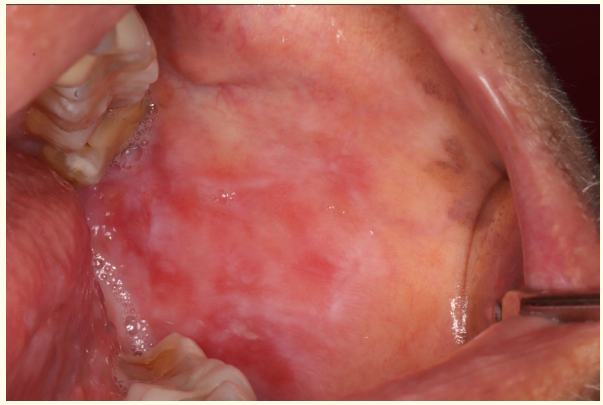

Figure 8

Dental Implications and Clinical Significance

OLCLs were classified as a subcategory of oral lichenoid lesions (OLLs) at the 2006 World Workshop of Oral Medicine IV [4]. Clinically, OLCLs are usually unilateral, asymmetrical, and most likely affect the buccal mucosa and ventral tongue [5]. Oral potentially malignant disorders (OPMDs) are a group of conditions that may precede the development of oral squamous cell carcinoma (OSCC). According to the latest WHO classification of OPMDs, OLCLs are considered to have a potential for malignancy following the emergence of new evidence after 2017 [6].

OLCLs and OLP are indistinguishable histopathologically. OLCLs due to close contact with dental materials is considered a type IV hypersensitivity reaction. A dermatologic patch test is suggested when cases of OLCLs are detected [2]. The rationale behind patch testing is to distinguish between OLCLs and OLP as they differ with regards to management protocols. The association between dental restorations and OLCLs is well documented in the literature [7]. Metals (Palladium, Nickel, Gold, Cobalt, Copper, Platinum), resins (Bis-GMA) and acrylate polymers are examples of materials with the capability of inducing OLCLs. In these cases, they are present in a direct topographic relationship with the dental material [2].

As previously noted, utilizing patch testing is a valuable approach when OLCLs are suspected, and there may be a need to consider replacing dental restorations. Existing evidence supports the replacement of dental restorations associated with OLCLs, irrespective of the patch testing outcome [8]. It's worth noting that resolution and improvement of OLCLs have been observed following the removal of dental material, even in cases where patch testing yields negative results [9]. Recent studies demonstrated that OLCLs results from cell mediated hypersensitivity to dental materials in patients sensitized from long exposure [10]. The improvement of OLCLs following the removal of the dental material has been described as complete or partial improvement. In one study OLCLs related to amalgam restorations resulted in 93% improvement following the removal of amalgam [8]. In another recent study, 23 of 24 patients (95.8%) with OLCLs showed a complete or partial improvement after the removal of dental metal [5].

OLLs and OLP malignant transformation is a controversial topic in research due to the number of reported cases in the literature. The dilemma arises when these cases are diagnosed histopathologically (true OLP vs OLL) [10]. Clinically, OLP and OLL should be clearly segregated. The rate of malignant transformation of OLP and OLL was found to be similar, with OLCLs affecting the tongue due to amalgam restoration most likely to transform to OSCC [11].

In a recent meta-analysis, the malignant transformation of OLL was found to be higher than OLP (2.5% vs 1.1 %) [12]. Another systematic review also noted the higher transformation rate of OLL compared to OLP (3.2% vs 1.09%) with the tongue as the most common site affected [13]. The higher malignant transformation of OLL including OLCLs warrants the replacement of dental restorations responsible for the development of these lesions. In cases where complete improvement is not achieved and only partial improvement is noted, regular observation and follow-up is advised.

Discussion

The mechanism of lichenoid inflammation in OLCLs is still debated. However, the most widely accepted hypothesis is that OLCLs are a result of a type IV (delayed) hypersensitivity reaction. This type of reaction is a cell-mediated, as opposed to antibody mediated, immune response in which T- lymphocytes are activated due to the detection of an antigen [14, 15]. The most implicated dental material causing OLCLs is amalgam. Several studies have demonstrated that amalgam constitutes, mainly mercury but also other constitutes, can induce a type IV (delayed) hypersensitivity reaction [16, 17]. In the oral cavity, corrosion of amalgam through processes such as electrochemical reactions and mechanical wear of the restoration form mastication results in the release of mercury salts and other corrosion by-products [18, 19]. Subsequently, these corrosion by-products can accumulate in the oral mucosa and initiate an immune-mediated response directed at the basal keratinocytes causing localized damage to the mucosa [14, 15]. It is thought that, through the dissolution in saliva, the sensitizing constitutes could spread beyond the contact zone allowing lesions to develop [20].

Allergic reactions to other dental materials and alloys have been reported, though to a significantly lesser degree. For example, Zinc oxide eugenol has been implicated as a sensitizing agent which can induce a type IV hypersensitivity reaction [7]; Behzad et al. [21] describes a case where patient presented with a histologically confirmed lichenoid lesion on the gingiva associated with a bridge cemented using eugenol cement. Similarly, a case report describes the resolution of a lesion after the replacement of a eugenol temporary restoration with glass ionomer [7]. Moreover, resin monomers (i.e., HEMA, Bis-GMA, EGDMA methacrylate) present in composites and some lab made dental appliances have been reported to cause OLCLs [7, 22-25]. However, free-monomer rates in composite after light curing are scant which could explain the low incidence of OLCLs associated with resin dental material [15].

It has also been proposed that another mechanism of OLCLs is due to toxic reactions [14, 26, 15]. Unlike type IV hypersensitivity reactions, toxic reactions are not cell mediated allergic reaction and patient with a chronic toxic reaction to a dental material would not test positive to a dental material patch test [14]. The localized inflammation arises from prolonged direct contact to a toxic substance and as such, the lesion is usually confined to the area in direct contact with the restoration [15, 26]. Though little is known about amalgam induced toxic reactions, it has been suggested it is more prevalent with high zinc amalgams [14].

Treatment of a confirmed amalgam induced OLCLs is replacement of the offending restoration with an alternative. As with any dental intervention, replacement of an amalgam restoration has its advantages and drawbacks. Studies have shown that replacement of amalgam in patients with OLCLs can in result in complete clinical recovery and termination of symptoms [28, 20, 27] with success rates reported to be 48 to 95% [15]. However, some patients with an OLCLs did not experience improvement after undergoing amalgam restoration replacement [28, 20].

OLCLs are classified as potentially malignant; thus, treating the lichenoid lesions, even if they are asymptomatic, would benefit patients affected by acting as a prophylactic measure that would, theoretically, prevent the development to OSCC [11]. However, it must be noted that clinically healed lesions do not necessarily correlate to histological healing [20], so routine oral cancer screening should be carried out at every dental visit.

Little research has been conducted to compare treatment outcomes of different amalgam replacements. In OLCLs, amalgam is often replaced with composite restorations [29] which can induce OLCL [25]. When metal-ceramic crowns have been used as amalgam replacements, the involved OLCLs did not regress [28]. One study examined the applicability of feldspathic ceramics as an alternative restorative material; 94% of the participants showed complete or partial regression [29]. However, similar healing rates were reported in other studies using other alternatives [25, 28]. As of yet, no single alternative restorative material is universally recommended as a replacement of amalgam restorations in OLCLs.

Conclusion

In summary, this comprehensive exploration of OLCLs underscores their clinical presentations, dental implications, and associated risks of malignant transformation. Recognizing mucosal changes related to lichenoid inflammation is crucial, given the established link between OLCLs and OPMDs. The clinical presentations, often asymmetrical and in proximity to dental restorative materials, emphasize the importance of distinguishing OLCLs from OLP. Dental implications and clinical significance are highlighted by the potential for malignancy, necessitating careful management and consideration of alternative restorative materials. The discussion delves into the debated mechanisms of lichenoid inflammation, emphasizing the role of type IV hypersensitivity reactions, particularly with amalgam restorations. Replacement of offending dental materials is advocated, with various alternatives explored. The progression and biological characteristics of numerous OPMDs are not well understood, emphasizing the need for additional research. There is a consensus that further exploration of these disorders is necessary [3]. Overall, this paper contributes valuable insights for the diagnosis and management of OLCLs, shedding light on the complexities and considerations in the realm of OPMDs management.

Conflicts of interest

All authors declare that they have no conflicts of interest.

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