Oral ulceration with bone sequestration

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Abstract

Oral mucosal ulceration is a common manifestation of various disease processes. Identification of the aetiological factor(s) involved greatly facilitates the management of such conditions. This report describes oral ulceration of the mucosa overlying the lingual shelf and mylohyoid ridge of the mandible and, less commonly on tori and exostoses, in association with bone sequestration. Trauma, which involves the subjacent periosteum resulting in a focus of ischaemic bone necrosis, in conjunction with local anatomical and perhaps other systemic predisposing factors, forms the aetiopathogenesis for this particular type of focal ulcerative lesion.

Key words: Oral ulceration, sequestrum, mylohyoid ridge.

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INTRODUCTION

Oral ulcerative lesions are common clinical findings and, although often of similar clinical appearance, their aetiologies can range from immunological, traumatic and neoplastic to the oral manifestations of systemic and dermatologic disease.1 Oral ulceration is often painful and if not managed appropriately, the associated morbidity can cause considerable disruption to a patient's usual lifestyle. Identification of the aetiological factor(s), including any underlying related medical condition, is paramount in the management of mucosal ulceration.

Traumatic ulcers, including those that subsequently develop into aphthae, are commonly occurring ulcerative lesions of the oral mucosa. They usually result from local dental trauma, are limited to the superficial tissue levels, and heal spontaneously within seven to 10 days or following the removal of the identified irritant. In this paper we describe a case of oral ulceration involving the full thickness of the mucosa with bony sequestration on the lingual aspect of the mandible, in association with the mylohyoid ridge and lingual shelf.

Although the condition is not common in general dental practice, there has been an apparent increase in the number of cases managed by the authors, along with recent reports describing similar presentations in the literature.2-5

In light of this, and the difficulties associated with the diagnosis and management of the condition, we report the presentations encountered in our clinical setting.

Clinical presentation

This article reports a particular type of focal ulcerative lesion on the lingual aspect of the horizontal ramus of the mandible, in association with the lingual shelf and mylohyoid ridge. Patients presenting with this form of ulceration are often referred by their general practitioner for treatment of a non-healing and generally extremely painful ulcer. The typical ulcer is 5-8mm in diameter, and presents as a surface defect with exposed underlying bone (Fig 1). The ulcer appears to have a glistening surface membrane, but close examination reveals a hard bony base that is generally non-vital and non-responsive to surface touch. However, the surrounding soft tissue margins are often extremely sensitive and may show considerable oedema and erythema. A similar clinical presentation is also seen in patients with lingual or palatal tori or bony exostoses (Fig 2 and 3).

A conservative strategy in the management of such cases is usually indicated, and involves rinsing with a tetracycline hydrochloride mouthwash three times daily, followed by the application of a topical corticosteroid. The rinses are prepared by mixing the contents of one 250mg capsule in a half glass of water and rinsing vigorously for one to two minutes. A number of topical corticosteroids are suitable, including 0.05 per cent betamethasone preparations and 0.1 per cent triamcinolone acetonide. This provides some anti-bacterial activity but the chemical débridement of the site is the main aim of the rinses, and allows the close application of the anti-inflammatory to minimize the soft tissue response.
causing the local pain. Both medications are used thrice daily, with the steroid being applied approximately 10 minutes after the rinse in order to allow the appropriate activity. Similar local tissue cleansing can be achieved with chlorhexidine or saline mouth rinses but this does not have anti-inflammatory activity and so may not provide any degree of improvement in local pain which is often reported as severe. It is outside the scope of this paper but clinicians should be familiar with both the indications and contraindications for the use of both medications as well as adverse reactions and hypersensitivity.

Patients are reviewed weekly for two to four weeks, until the bony sequestrum is mobile or can be manipulated. At this stage the bony fragment, typically measuring 2-5mm, can be removed. Once the sequestrum is removed, the ulcer resolves rapidly and the soft tissue defect will re-epithelialize within a few days.

Another management strategy is the surgical removal of the sequestrum to accelerate the healing process. This is done in situations of extreme and constant pain, by means of a small incision above the mylohyoid ridge and on the alveolus to ensure the lingual nerve is not placed at risk. The bony sequestrum can then be removed following local conservative dissection, and any spicules smoothed using a bone file. The flap is sutured back into position and healing usually occurs within a few days. If direct access to the sequestrum is possible, it can be grasped and removed without the need for surgical intervention and the inherent potential for complications. Local anaesthesia used for any of these procedures may be either local field block or a regional block in the mandible.Clinicians should be aware that injudicious use of local field anaesthesia with vasoconstriction and further periosteal damage could extend the clinical course.

Histopathological examination of the recovered specimen reveals fragments of non-vital irregular bone with lacunae on the inner and lateral surfaces indicating peripheral resorption. Attached soft tissue fragments are usually minimal but surface bacterial colonization particularly on the outer surface, with or without a small amount of suppurative material, is a common finding. There are no other remarkable features to suggest the histopathological features represent anything other than an uncomplicated spontaneous sequestrum.

In occasional patients there are several episodes of sequestration at the one site and this is inevitably associated with severe local pain over an extended period. However, the likelihood of this occurring is difficult to predict as the loss of one sequestrum is generally followed by ulcer healing but with the persistence or subsequent development of further pain.

**DISCUSSION**

A sequestrum is a fragment of non-vital bone separated from adjacent sound bone as a result of focal bone necrosis and subsequent isolation and resorption of the site margins. Sequestration is usually associated with a disruption to the blood supply of bone as a response to various local or systemic factors. These might include infection, diabetes, leukaemia, radiation, Paget’s disease of bone, fibrous dysplasia, malignant bone change, malnutrition, or heavy metal poisoning.

In a dental context, sequestrum formation in the jaws following routine dental extraction is occasionally
seen. These sequestra can either be left to spontaneously exfoliate, or can be surgically removed if pain or discomfort is experienced. The so-called eruption sequestrum presents itself overlying the crown of an erupting tooth, and is usually self-limiting.

This report describes the presentation of oral ulceration associated with bone sequestration on the lingual mandible at the level of the mylohyoid ridge and at other sites involving tori and exostoses. Proper diagnosis is important in the management of all ulcerative lesions, and is paramount in this instance, since treatment depends on the successful identification and removal of the bony sequestrum. This can occur either by active physiological resorption and assisted or spontaneous loss of the fragment or by formal surgical removal.

There are several factors that can be ascribed to the formation of this type of bone devitalization. It is most likely that a traumatic irritant, such as food trauma or tooth brushing, causes a disruption to the local blood supply in the area by creating a breach in the mucosal soft tissues and including the subjacent periosteum. The underlying bone is left unprotected and ischaemic necrosis ensues. The identified sites are more susceptible to this form of trauma because of the prominence of the mylohyoid ridge relative to the flat lingual aspect of the mandible, and the thin lining mucosa overlying this bony protuberance compared with the thick and resilient masticatory mucosa found on the adjacent gingiva. Palatine tissues are masticatory mucosa but over tori and exostoses it is a common clinical observation that the tissues appear stretched and atrophic. In this situation they would be predisposed to rather more severe soft tissue effects given the same degree of trauma occurring elsewhere in the mouth.

The normal alignment of the dentition and state of occlusion of the posterior molar teeth and the lingual inclination of the mandibular molars protects this area from irritation during mastication. In patients with missing posterior molars, or in the case of restorations with incorrect contours, food particles are not appropriately reflected away from the oral soft tissues, thus increasing the risk of continuous low grade trauma to the area. This is compounded by the accumulation of debris in the posterior part of the oral cavity as a result of the comparative lack of cleansing capability of the relatively non-mobile posterior portion of the tongue. This, along with microbial contamination could predispose the exposed bone to secondary infection.

A recent suggestion has placed the ulcerations within the category of aphthous lesions. From current knowledge of aphthous ulcerative disease it seems highly likely that those patients who suffer from recurrent aphthae will develop an aphthous lesion subsequent to the original traumatic lesion. Whether this would encourage the formation of a sequestrum is difficult to determine and perhaps could only be answered by a clinical analysis of cases in those who do and do not suffer from aphthae. Experience suggests that the local tissue reaction in aphthae would be significantly more severe and this may explain the variation in presentation between patients. It also introduces the complexity of an immunologically mediated condition as distinct from a comparatively simple traumatic event.

The role of trauma and ulceration in the formation of sequestra in this area can be further explored by studying the role of the periosteal microvasculature of the mandible which supplies the outer cortex.

The contributory role of systemic factors may further increase the risk of sequestration. Any of the systemic factors listed earlier could contribute to a loss of integrity of the periosteal and peripheral cortical circulation. This would heighten the chance of local trauma resulting in ischaemic necrosis of the outer cortex, with resultant tissue damage including both a soft and hard tissue component.

A variety of modalities exist for the treatment of oral ulceration. This usually takes the form of antibacterial mouth rinses and anti-inflammatory creams or ointments used either separately or in combination with the prescription of these agents being empirical rather than following careful consideration of the pathological processes and the desired end-point. The main aim of treatment with the current type of ulcerative lesion is to provide supportive treatment to the healing lesion in as non-invasive a manner as possible. This is achievable by chemical débridement of the site with a tetracycline hydrochloride mouth rinse and the suppression of the often marked local inflammatory activity with a topical corticosteroid. Bacterial overgrowth and secondary infection is not usually a consideration in the authors’ experience although the regional lymph nodes may be reactive. The above will usually achieve the patient’s major treatment aim of pain relief. The reduction in inflammatory activity and soft tissue responses achieve this aim although patients can certainly use any of the available proprietary antiseptic/anaesthetic preparations on an as-required basis.

The non-vital bone segment can be managed in a number of ways. However, in view of the traumatic aetiology of the lesion fostered by ischaemic necrosis of the dense cortical bone, with a possibly reduced vascular supply at the sites usually involved, the authors generally advise against active surgical intervention. If possible, the non-vital bone is left to sequestrate by the ingress of vital granulation tissue from the deep and lateral surfaces. Following either spontaneous or assisted mobilization, the fragment can then either spontaneously exfoliate or be carefully removed. Fragments that mobilize spontaneously are often lost unknowingly during normal oral functions. Assisted mobilization is also helpful with careful removal of the fragment. In either scenario, the patient’s pain will usually resolve rapidly following the loss of the sequestrum.
In those cases where surgical intervention is indicated the clinician is urged to ensure that whilst treatment should be conservative, the area should be cleared back to vital bleeding bone to minimize the opportunity for recurrence.

CONCLUSION

In summary, this report describes oral ulceration of the mucosa overlying the lingual shelf and mylohyoid ridge of the mandible, and less commonly on tori and exostoses, in association with bony sequestration. Trauma, in conjunction with local anatomical and in some cases, other systemic predisposing factors, and perhaps the superimposition of a pre-existing trait to develop aphthous ulceration following soft tissue trauma, allows the formation of a focus of ischaemic bone necrosis as a complication to the original ulcerative lesion.

REFERENCES


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