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Emergent periodontal conditions: An overview

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ABSTRACT

Dental emergency patients present often with periodontal conditions which they deem emergent. Most of these disease states are acute, although some are more associated with chronic ulcerative lesions, such as those seen in desquamative gingivitis cases. Pain or discomfort is the usual reason these patients seek treatment, in addition to swelling, infection, and/or bleeding/suppuration. Oral health care practitioners should have available to them a systematic approach to diagnosis and treatment of these conditions, leading to resolution and improved quality of life for their patients. This article describes common periodontal emergencies, their etiology, and the clinical management of these disorders.

Introduction

The definition of an emergency is often very different to a clinician than what a patient perceives as an urgent or pressing need as any condition impacting daily function or esthetics is often considered an emergency by the patient, even if it is not a "true" emergency from a medical standpoint. Most emergent periodontal conditions are acute lesions characterized by pain, discomfort, and/or infection [1]. They may present as localized or generalized lesions, with possible systemic manifestations. A few symptomatic oral pathoses are ulcerative lesions associated with chronic diseases. Acute periodontal conditions usually respond to treatment, provided the diagnosis is correct. If the therapy performed was clinically acceptable but the therapeutic response is not relatively prompt, then the clinician should re-evaluate the initial diagnosis and/or treatment [2].

A complete oral/periodontal examination, to include clinical and radiographic findings, is imperative in formulating an initial diagnosis. The first mandatory step in the exam process is a thorough review of the patient's medical history, to include a listing of all current medications [3]. Many oral ulcerative lesions are associated with drug intake, especially some commonly used nonsteroidal anti-inflammatory drugs (NSAIDs) [4]. A comprehensive appraisal of the medical and dental histories should always be completed as part of the examination process prior to making a diagnosis.

The following conditions (not including traumatic/chemical/thermal injuries or severe dentinal hypersensitivity) affecting the periodontium are those that this paper will address as potential reasons for patients to seek care on an emergency basis:

1. Gingival/Periodontal abscess (differentiate from periapical abscess and combined perio-endo lesions)
2. Pericoronal abscess (pericoronitis or PCOR)
3. Necrotizing periodontal diseases (NG, NP)
4. Herpetic/viral infection ulcerations (differentiate from aphthae)
5. Aphthae ulcerations (differentiate from viral ulcerations)
6. Desquamative gingivitis (differentiate from other erythematous/ulcerative lesions)

Overview

Gingival abscess/periodontal abscess/periapical abscess

The gingival abscess is a localized pus-containing infection confined to the marginal gingiva or interdental papillae (Fig. 1) [5]. The periodontal abscess is also a localized suppurative infection, not confined to the marginal tissues, but involving the adjacent periodontium. Abscesses of the periodontium have been reported to be commonplace dental emergencies in multiple studies,[1,6–9] with Herrera et al. finding the periodontal abscess to be the 3rd most frequent dental emergency [7]. The periodontal abscess has also been found to have a high prevalence among oral emergencies in a variety of different practice settings [10–13]. Both the gingival and periodontal abscess may exhibit a shiny erythematous swelling, purulent exudate, and pain, but there will be an increased probing depth associated with possible radiographic evidence of bone loss in the periodontal abscess (Fig. 2). Tooth mobility is also a common finding, especially in periodontal abscesses. Distinguishing periodontal abscesses from periapical abscesses (Fig. 3) is also essential

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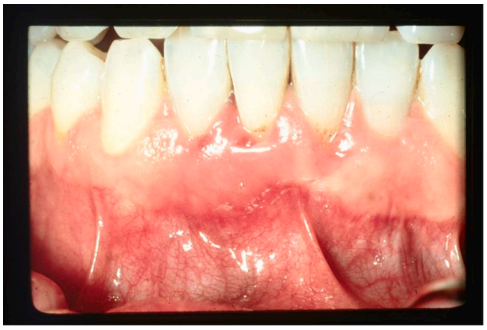


Fig. 1. Gingival abscess localized to the mandibular incisor, characterized by erythema and swelling.

as the primary etiology is different (i.e., periodontal pathogens for periodontal abscesses versus pulpal pathogens for periapical/endodontic abscesses). In a study of 168 adult patients needing hospital care for severe odontogenic infection, Furuholm et al. discovered that apical periodontitis (apical abscess) was the most common dental abscess requiring hospitalization (67 % of cases) [14]. Treatment for an abscess is drainage/irrigation, with or without local/systemic chemotherapy as needed. Extraction is also an option if the tooth is deemed hopeless. Although many studies have shown that an abscess is the primary reason teeth are extracted during periodontal maintenance therapy, McLeod et al. in a retrospective study showed that 55 % of abscessed teeth could be treated and retained for many years (5 to 29 years) [15].

Pericoronal abscess/pericoronitis (PCOR)

Soft tissue swelling and inflammation over a partially erupted tooth, usually the distal of a mandibular 3rd molar, is diagnosed as pericoronitis (PCOR). The operculum of soft tissue overlying the tooth which traps food debris and bacteria is often edematous and symptomatic, sometimes aggravated by traumatic occlusion from an opposing tooth directly impinging upon the operculum (Fig. 4). The most frequent type of partial impaction that may lead to PCOR was shown to be mesioangular by Shirzadeh et al. [16]. Pericoronitis is one of the most common emergent periodontal conditions observed [17]. Resolving the acute inflammatory phase of the infection should be the first priority, but always treat as aggressive as necessary because of the potential for spreading, leading to a space infection. Anatomic space infections developing into osteomyelitis of the mandible, albeit rare, have been reported [18]. Treatment should be focused on debridement, irrigation and drainage, and/or extraction as soon as possible if the tooth is not to be retained. If indicated, empirical systemic antibiotic therapy should be initiated. Despite the recent emphasis on antibiotic stewardship by the CDC and ADA, many practitioners still prescribe amoxicillin as the first drug of choice when conservative dental treatments are not readily available. Amoxicillin, when prescribed at a recommended dose, is proven to have a better efficacy against gram negative anaerobes with fewer gastrointestinal side effects. In a study of 184 bacterial strains in 64 patients with periodontal, periapical or pericoronal infections, Bresco-Salinas et al. found the antibiotic with the greatest sensitivity and lowest resistance was amoxicillin + clavulanate (Augmentin) [19]. For patient with a history of penicillin allergy without anaphylaxis, angioedema or hives, oral cephalexin at a dosage of 500 mg, 4 times a day for 3 −7 days is recommended. Since cephalexin is known to have a limited anaerobic coverage, an addition of metronidazole is often recommended should the response be delayed. For patients with a history of penicillin allergy with the above mentioned adverse symptoms, oral azithromycin at a starting dosage of 500 mg for 1 day followed by 250 mg for the next 4 days or clindamycin at a dosage of 300 mg four times a day for 3–7 days is recommended [20–21]. Clindamycin has a black box

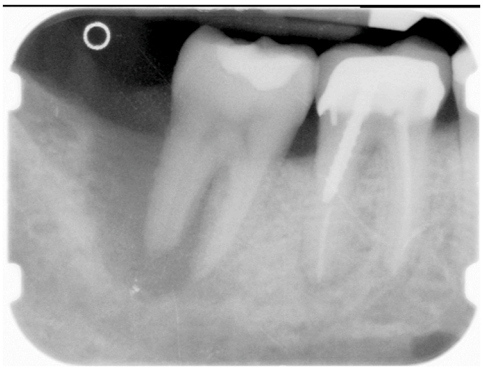


Fig. 3. Periapical radiograph presenting a combined endodontic-periodontal lesion on nonvital tooth #31 showing periapical and distal lateral radiolucency.

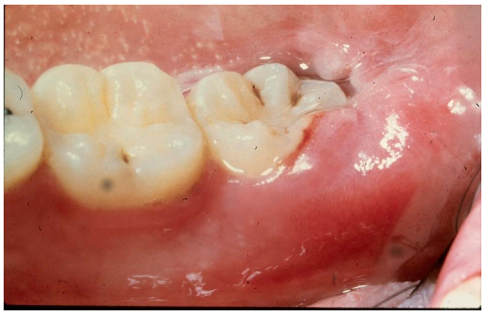


Fig. 4. Pericoronitis on partially erupted #32 presenting with inflamed operculum and peri-coronal tissue.

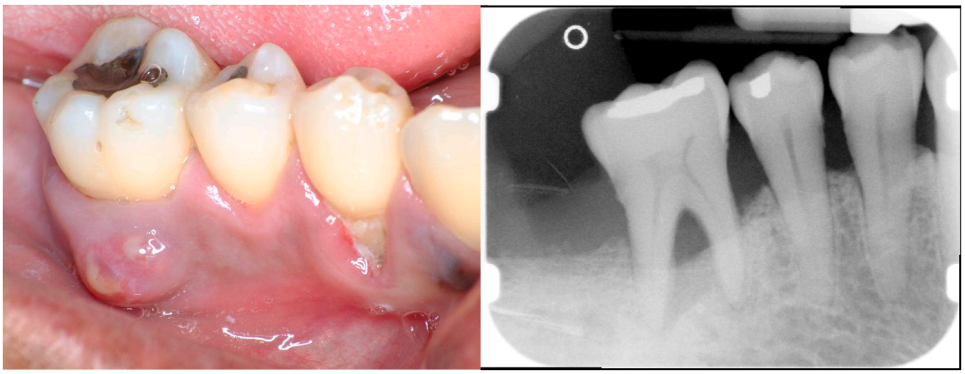


Fig. 2. Periodontal abscess associated with tooth #31 presenting with swelling, purulent exudate and loss of supporting structure.

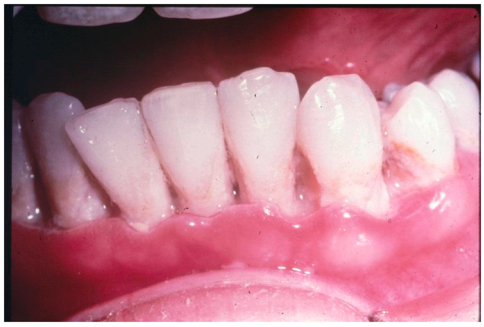


Fig. 5. Necrotizing periodontal disease affecting the mandibular anterior region, presenting ulcerated interdental papillae and pseudomembrane formation.

warning for C. difficile infection, however azithromycin has higher resistance rates. Hence, patients should be advised to closely monitor the symptoms and seek immediate care should worsening occur. Herrera et al. compared amoxicillin/clavulanate to azithromycin in the short-term systemic treatment of acute periodontal abscesses and discovered equal effectiveness for both antibiotic regimens [21–22].

Necrotizing periodontal diseases (NPD)

Necrotizing periodontal diseases consist of acute oral infections characterized by the extent of involvement in the periodontium; and which can be very severe dependent upon the stage of the disease. Some authors have noted that these necrotizing oral infections (necrotizing diseases or NDs) are the same disease process distinguished only by their stage of progression [23]. NPDs, mainly consisting of necrotizing gingivitis (NG) and necrotizing periodontitis (NP), were previously referred to as NUG (necrotizing ulcerative gingivitis) and NUP (necrotizing ulcerative periodontitis). The accepted current terminology omits the word "ulcerative" since ulceration is considered to be a secondary sloughing of necrotic tissue [1]. NG, confined to the gingiva, may progress to NP, which involves attachment/bone loss. Severe untreated NP uncommonly may advance into orofacial tissues beyond the periodontium, leading to necrotizing stomatitis (NS) or even noma, a serious gangrenous infection with a high frequency of mortality and disability [24]. According to the World Health Organization (WHO), noma consists of five stages, with the first stage being NG [25]. Feller et al. proclaim that NG, NP and NS are not stages of noma, but are instead distinctive oral NDs that may rarely develop into noma [26]. Primary ("classic triad") clinical signs and symptoms of NG are pain, bleeding, and interdental papillary necrosis ("punched out" appearance of papilla). Anterior gingiva is a common infected site, with the mandibular anterior the most frequently affected [9]. (Fig. 5). Secondary conditions which may be observed are mouth odor, bad taste, fever, lymphadenopathy, and/or malaise. Predisposing factors may be stress, decreased resistance to infection from systemic illness, poor oral hygiene, malnutrition, and smoking. It has been shown that >90 % of NG patients are heavy smokers [27–28]. A 2017 study of NG in the British Armed Forces revealed that although NG was rare, the two strongest risk factors for diagnosed NG were smoking and poor oral hygiene [29]. The etiology of NPDs are fusospirochetal organisms that respond well to a pure anti-anaerobic antibiotic such as metronidazole [30]. In addition to systemic antibiotic therapy, mechanical debridement and antiseptic rinses should be used to treat NPD patients [5,31–33].

Herpetic/viral infection (ulcerations)

The most common oral/perioral viral infection in adults is due to herpes simplex virus (HSV). HSV infection can be primary, as usually seen in children or young adults, or recurrent. Primary herpetic infection

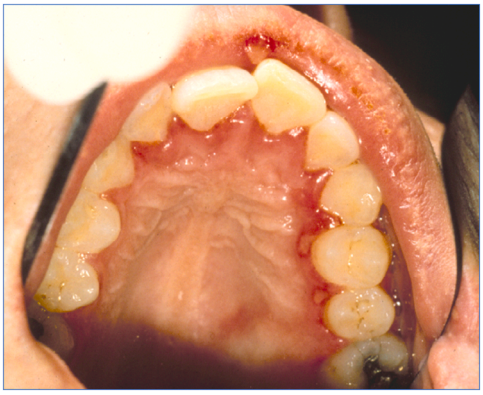


Fig. 6. Multiple sites of palatal ulcerations of viral (herpetic) origin affecting the keratinized mucosa.

can be characterized by pain and ulceration in oral mucosa, a feeling of malaise, swollen lymph glands and/or fever, especially in adults who are exposed to the virus for the first time later in life. Most people are exposed at some time early in childhood to HSV, where the clinical symptoms are not near as severe or may be even subclinical and asymptomatic [34]. Nahmias revealed that >85 % of adults show laboratory confirmed verification of previous exposure to HSV [35]. Conversely, recurrent HSV infection is distinguished by localized vesicles that coalesce to form ulcers (Fig. 6) mostly on keratinized mucosal surfaces (as opposed to aphthous ulcerations which occur mainly on nonkeratinized mucosa) [36–37]. The exception to this common clinical rule is when treating immunocompromised patients; then, the ulcerations may be on keratinized and/or nonkeratinized oral surfaces versus ulcerations usually found on immunocompetent individuals [38]. In Eisen's 1998 study on intraoral HSV infection in 52 patients with normal immunity, he found that most ulcerated lesions (47 of 52) were on only keratinized surfaces with just 5 patients exhibiting persistent ulcers on nonkeratinized mucosa [39]. Recurrent HSV infection is commonly seen on gingiva, palate and/or lip (herpes labialis). Treatment is aimed at controlling discomfort and shortening disease episodes with antiviral medication. Mouthrinses proven to help alleviate pain include 0.12 % chlorhexidine or a compounded rinse of anesthetic, antihistamine and aluminum hydroxide/magnesium hydroxide (1–2–3 Mouthwash: viscous lidocaine, diphenhydramine, Maalox®). Oral systemic antivirals shown to be effective include acyclovir, valacyclovir and famciclovir. Multiple studies have revealed the efficacy of early prodromal episodic treatment with valacyclovir,[40–42] which has greater bioavailability than acyclovir. Spruance et al in a randomized controlled clinical study of over 700 patients found that single dose famciclovir showed even greater benefit to valacyclovir in treating recurrent herpes labialis [43]. The authors postulated that the increased benefit may be from a longer duration of intracellular concentration of the famciclovir metabolite penciclovir (famciclovir converted to penciclovir in liver, excreted mostly unchanged by kidneys), as revealed by Earnshaw et al. [44]. Topical antivirals like acyclovir ointment, penciclovir cream, and docosanol cream have all showed promise for extraoral herpes labialis. Lastly, we must mention the anecdotal practice by some patients who have experienced persistent outbreaks of recurrent HSV infection, the daily intake of supplemental L-lysine for HSV prophylaxis. Many patients believe there is a positive effect of lysine when on a low arginine diet (chocolate, nuts, pumpkin seeds) for HSV prevention, though most studies produced mixed results. A fairly recent (2017) systematic review of this literature by Mailoo and Rampes revealed that L-lysine supplementation for prophylaxis or HSV treatment with doses of <1 *g*/day without low arginine diets was ineffective [45]. However, doses greater than 3 *g*/day may possibly reduce recurrence rates and improve self-reported symptoms. Doctors who have patients that self-medicate with lysine should warn of the dangers of too high lysine levels, i.e.

Table 1
Characteristics of herpetic (viral) ulcerations versus aphthous (autoimmune) ulcerations.

	HERPETIC	APHTHAE
Site	Keratinized bound mucosa	Nonkeratinized movable mucosa
Features	Multiple vesicles rupture, coalesce and form ulcers with irregular borders. Preceded by prodromal symptoms	No vesicles. Start as single or multiple ulcerations bordered by erythematous halos. Typically no prodromal symptoms
Pain	Yes (> in herpes labialis)	Yes
Duration	7–14 days	5–10 days
Therapy	Topical or systemic antiviral medications	Topical steroids, systemic for major aphthae (eg. Sutton's disease)

possible increased cholesterol levels (cardiovascular) and/or increased incidence of gallstones (gall bladder).

Aphthous ulcerations/aphthous stomatitis (AS)

Aphthae (canker sores) are small, painful ulcers that have no prevesicular stage, nor do they usually have any prodromal symptoms like herpetic ulcerations. AS has been reported to be the most common oral mucosal disorder [46]. The exact etiology is unknown, although most evidence points to a cell-mediated immune reaction that could be initiated by multiple factors like stress, medications, diet, or hormonal changes [31,47]. Rees' findings from the Baylor Stomatology Center indicate that up to 85 % of patients develop aphthae before the age of 30, and suggests the possibility of a strong genetic predisposition [48]. As discussed in the previous section on HSV infection, despite some similarities with the clinical presentation (see Table 1), aphthous ulcerations do not usually occur on keratinized tissue [8]. Because they are usually on mucosa (nonkeratinized), patients who experience recurrent episodes of oral ulcerations (viral vs aphthae) can be clinically diagnosed mainly based on clinical presentation (Fig. 7). In fact, Cohen et al. reported that most patients who present with recurrent ulcerations probably have aphthae versus other causes such as HSV, trauma, and other etiology [49]. Therapy for aphthae is directed toward palliative care and possibly shortening the duration of ulcerations. Most patients respond well to topical steroids, and other treatments to include chemical cautery, laser ablation, and oral suspensions such as tetracycline/tetracycline derivatives. Low-dose doxycycline gel has shown significant improvement in most patients with healing by the third day [50]. Tetracyclines have a potent anti-inflammatory effect, especially due to inhibiting matrix metalloproteinases (MMPs) which promote tissue destruction [51]. A very recent meta-analysis review by Mashrah et al. looking at the efficacy of 20 different topical medications for treating AS, the only drug that showed a statistically significant reduction in healing time was topical doxycycline [52].

Desquamative gingivitis (DG)

Desquamative gingivitis is a descriptive clinical term usually involving autoimmune diseases and characterized by symptomatic oral mucosal changes such as erythema, desquamation and erosion, and vesiculation leading to ulcerated lesions. The most common disorders in this group seen intraorally are lichen planus, pemphigoid, and pemphigus [53]. Reticular is the most common form of lichen planus, usually seen on buccal mucosa, but the potentially painful erosive form is what is seen on gingiva as often as other mucosa. The "target" tissue for cicatricial pemphigoid is gingiva, while the most frequent extraoral site is ocular tissue. Pemphigus vulgaris (PV) patients often present with oral mucosal lesions, with or without cutaneous involvement, and are seen in the majority of patients with PV [54]. DG has been observed in 25 % of PV patients, while DG is the most common and often the only sign/symptom in pemphigoid patients [55]. Technically, although pemphigoid is an autoimmune bullous (blisters) disease, both

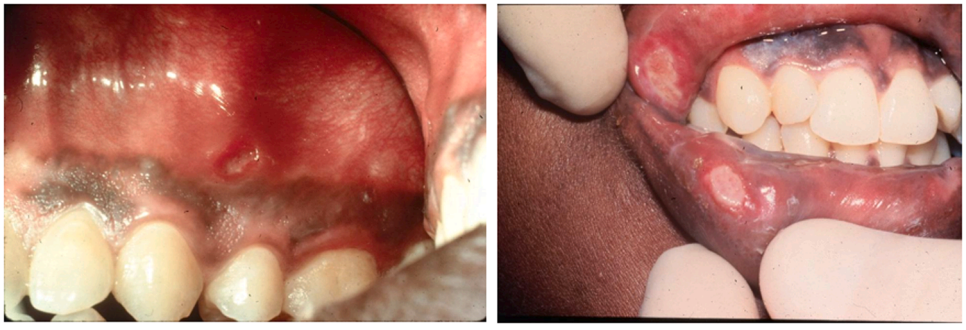
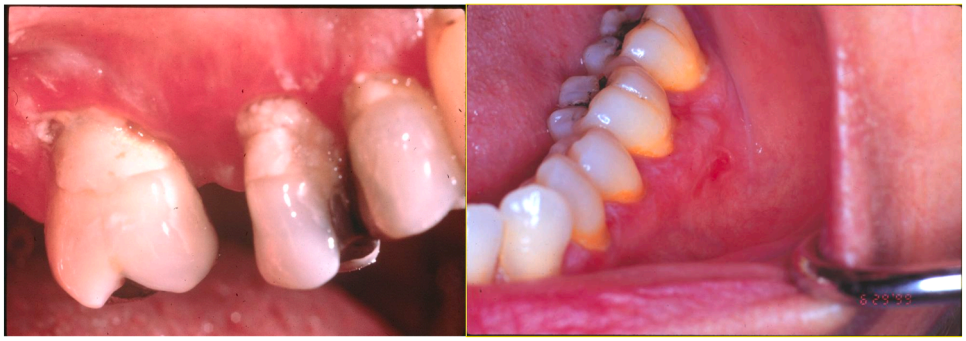


Fig. 7. Aphthous ulcerations on nonkeratinized mucosa featuring shallow ulcers with erythematous halo.



A B

Fig. 8. A. Erosive lichen planus on the right maxillary buccal region presenting with areas of erythema and ulcerations, B. Mucous membrane or cicatricial pemphigoid presenting with desquamation on the attached mucosa.

pemphigoid and lichen planus occurring on gingiva can look alike (Fig. 8) and be difficult to distinguish clinically without histology/immunofluorescence [56–57]. Treatment for these lesions usually include topical and/or steroids, dependent upon extent of involvement. Tacrolimus, a calcineurin inhibitor, has also shown in a recent trial to be very effective in treating patients with oral lichen planus and pemphigoid [58]. It is an immunosuppressant that blocks calcineurin, which is necessary for interleukin-2 production, which in turn will decrease proinflammatory cytokine production. In discussing lesions that may lead to oral ulcerations (recurrent AS, viral ulcers, autoimmune DG), if initial treatment options fail, one must also consider other rare immune-mediated diseases that can cause painful oral ulcerations like chronic ulcerataive stomatitis (CUS). CUS can resemble erosive LP, cicatricial pemphigoid, and pemphigus vulgaris, with the most affected intraoral sites being buccal mucosa, gingiva, and tongue [59]. Unlike other autoimmune diseases, to include aphthae, ulcers from CUS do not respond well to steroid therapy [60], but do get a good response from systemic hydroxychloroquine [61]. Recurrent AS ulcerations are usually smaller in size and heal rather quickly, while CUS is much more uncommon and the ulcerations are more persistent and do not heal easily.

Conclusions

As one can see, dental emergencies that involve the periodontium and surrounding tissues can be pervasive, but with very distinct and different etiologies. Obviously, to remedy the patient's chief complaint, we must perform a comprehensive examination in order to achieve an accurate diagnosis. Only with the correct cause of the disease can we then prescribe the appropriate treatment. An astute clinician will realize that many emergencies are localized conditions, but some may well have systemic involvement. When in doubt or lack of initial success, do not be reluctant to obtain specialty consultation(s). Do whatever is best for the patient's well-being.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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The authors report no conflict of interest.

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