Many different disease processes may manifest as oral ulcerations. Accordingly, dental professionals are often the first (and sometimes only) professionals to identify these lesions. Recognition, diagnosis and management for these patients is crucial not only to their oral health, but to their overall health in general.

Etiology and diagnosis must be determined to effectively render treatment. Causative agents may be reactive, autoimmune, neoplastic or infectious. Is the ulcer due to a patient biting his or her tongue, or is it because he or she has a serious undiagnosed autoimmune disease? This brief article will touch on common causes of oral ulcerations in an attempt to promote early diagnosis and intervention when needed.

**Traumatic ulcerations**

Traumatic ulcers are the most common cause for intraoral ulcerations and are seen in all age groups. Causes include biting of soft tissues (both accidental and parafunctional), fractured cusps or restorations, and hot or sharp food items.

Common locations for traumatic ulcers include lateral border of the tongue, buccal mucosa and labial mucosa. Diagnoses can usually be elicited by utilizing an accurate medical history and clinical examination (in the case of a sharp cusp or restoration).

Management of these lesions is aimed at removing the etiology and re-evaluating for resolution. In the case of a suspected bite-induced traumatic ulcer where no other etiology can be found, reevaluation at two weeks is appropriate.

Oral ulcerations that persist for more than two weeks without significant improvement or resolution require incisional scalpel biopsy to rule out malignancy (squamous cell carcinoma being the most common).

Incisional biopsies of ulcerated mucosa should include adjacent normal-appearing tissue, as some disease processes (see below) occur at the epithelial-connective tissue interface. Topical corticosteroids may be used to hasten healing and decrease pain.

**Herpesviridae**

There are eight known viruses in the herpesviridae family that affect humans. These include simplex 1 (oral) and 2 (genital), Varicella zoster, Epstein-Barr, cytomegalovirus, 6 (roseolovirus) and 7, and HHV-8, which is associated with Kaposi’s sarcoma. This article will focus on herpes simplex virus 1 (HHV-1) which is the most common of the viruses that cause oral and perioral ulcerations.

Herpes infections consist of two main phases. It should be noted that not everyone who contracts the disease displays clinical symptoms. The primary phase is most commonly seen in young children. Inoculation is usually by contact with a family member or at school.

This is followed by an incubation of up to 10 days followed by fever, malaise, pharyngitis, and cervical lymphadenopathy. Intraoral manifestations include gingivostomatitis and diffuse oral ulcerations that may occur on both attached and unattached mucosa. Desquamation of gingiva and inflammation of interdental
papilla may resemble acute necrotizing ulcerative gingivitis, but there is no actual loss of papilla.

Diagnosis is usually based on clinical presentation and history. A cytologic smear may be performed for diagnosis but should not delay treatment. Treatment goals include supportive therapy and palliative care. Antipyretics (avoid aspirin in children) are indicated as necessary, as is the need to maintain adequate hydration. Often, patients have decreased oral intake due to pain and young children may require hospitalization for intravenous fluids. Viscous lidocaine may be used in older patients but should be avoided in younger patients, who may swallow it.

Lesions typically resolve spontaneously in 10 days to 14 days in the healthy patient, but may persist in the immunocompromised patient and require systemic antiviral medications. The secondary (recurrent) phase of herpes infection typically occurs on the lip (herpes labialis) or intraorally on attached mucosa (gingiva or palate) and can be caused by stress or trauma, or be idiopathic in nature. These lesions often exhibit a prodromal stage or tingling sensation in the area before an outbreak occurs. This is followed by the appearance of crops of vesicles that subsequently rupture and coalesce into an ulcer, which crusts over and usually heals within 10 days to 14 days.

Intraorally, recurrent herpetic outbreaks may be precipitated by anesthetic injections or local trauma from scaling procedures.

Treatment is often palliative after the appearance of vesicles, as antivirals are usually only effective in the prodromal stage. Patients who experience recurrent outbreaks and can recognize prodrome may benefit from taking antivirals during this stage to attempt to abort an outbreak.

Aphthous ulcers

Aphthous ulcers (commonly referred to as canker sores) occur at all ages and represent one of the more common etiologies of oral ulcerations. Although the exact cause of the ulceration is not known, it is widely believed that aphthae represent an immunologic reaction. Unlike herpetic ulcers (and the herpetiform variant of aphthous mentioned later), aphthous lesions occur exclusively on unattached mucosa (tongue, labial and buccal mucosa, and soft palate).

The most frequently seen variation is dubbed aphthous minor, and consists of an ulcer (or fibrin-covered ulcer) less than one centimeter in diameter and surrounded by an erythematosus halo, and is exquisitely painful. Minor aphthae spontaneously resolve in seven days to 14 days. Major aphthous ulcers are similar in appearance to minor aphthous but can range in size from 1cm to 3cm and may take up to six weeks to heal.

Recalcitrant lesions may require systemic corticosteroids (i.e., prednisone) or other immunomodulatory agents (tetracycline/niacinamide). It should be noted that aphthous-like lesions are associated with many systemic disorders, including Behçet’s disease (ocular and genital involvement), Crohn’s disease, and disorders of the gastrointestinal tract, and may require referral to their physician for further evaluation.

Lichenoid lesions

Lichenoid lesions include both reticular and erosive lichen planus as well as lichen mucositis and drug reactions. Lichen planus is autoimmune in origin and typically affects middle-aged females.

Patients may have oral lesions, skin lesions, or both. Clinical presentation of the reticular form resembles white lace

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A less-common form called herpetiform aphthous can occur on the palate and mimic recurrent intraoral herpetic. Treatment is palliative and also aimed at reducing overall time to resolution. Topical corticosteroids are an excellent choice for minor, herpetiform, and some major forms of aphthous. Some practitioners use cautery in either the form of laser or silver nitrate. All methods are acceptable, as lesions typically resolve without any treatment at all. (Wickham’s striae) and is commonly found on the buccal mucosa and tongue. These are typically asymptomatic but may become symptomatic if ulceration occurs.

Erosive lichen planus commonly presents as desquamative gingivitis of the anterior maxilla and mandible. Unlike the reticular form, the erosive form is commonly symptomatic and may mimic other disease processes (see below). Incisional biopsy is indicated to confirm diagnosis. Other
Lichenoid lesions (lichenoid mucositis) may be the result of administration of any of a number of different classes of drugs, including ACE inhibitors and NSAIDS. Contact mucositis may also appear lichenoid and may be the result of restorative materials, metals, or parafunctional habits. Treatment of lichenoid and contact-induced lesions involves determination of the offending agent and subsequent removal. Asymptomatic lichen planus often requires no management other than routine follow-up, as there is debate on potential for malignant transformation. Symptomatic lesions usually respond well to topical agents, but systemic medicaments may be necessary.

**Benign mucous membrane pemphigoid (BMMP)/pemphigus vulgaris (PV)/erythema multiforme (EM)**

These disease entities will be briefly discussed because of their clinical presentation and association with systemic manifestations. BMMP and PV are both autoimmune vesiculobullous diseases. They often present intraorally with desquamative gingivitis and multiple painful ulcerations (especially in areas susceptible to trauma). Clinically they can both demonstrate sloughing of gingival mucosa with lateral pressure (Nikolsky’s sign).

Diagnosis is made by biopsy and both H&E and immunohistochemical methods. The etiologies of BMMP and PV include circulating autoantibodies to different adhesion proteins with resultant detachment of epithelial cells from the underlying connective tissue or from adjacent epithelial cells.

Patients diagnosed with BMMP require ophthalmological evaluation, as they are prone to conjunctival adhesions (symblepharon) that may result in blindness. Pemphigus patients require treatment by specialists in dermatology and vesiculobullous diseases, as they have multiple organ systems affected.

Oral lesions may be managed symptomatically with topical steroids, but these patients are usually placed on high-dose immunosuppressive therapy, which may result in concomitant intraoral opportunistic infections such as candidiasis.

**Erythema multiforme** represents a spectrum of disease that ranges from EM minor (crusting of lips, oral ulcerations, desquamative gingivitis) to EM major and to toxic epidermal necrolysis (sometimes referred to as Stevens-Johnson syndrome). Targetoid lesions on the skin are pathognomonic for EM, though not always.

**QUIZ Oral Ulcerations**

1. Treatment of primary herpetic gingivostomatitis is aimed at:
   A. Complete elimination of the herpes virus from the body
   B. Preventing recurrence
   C. Palliative care, including proper hydration
   D. Periodontal procedures to address loss of interdental papilla

2. A patient with an oral ulcer persisting for more than two weeks should:
   A. Follow-up in another two weeks for reevaluation
   B. Have an incisional biopsy with scalpel
   C. Undergo pharmacological intervention
   D. Be informed that it is most likely traumatic and it should heal by itself

3. Which of the following does not typically present with desquamative gingivitis?
   A. Benign mucous membrane pemphigoid
   B. Pemphigus vulgaris
   C. Aphthous minor
   D. Lichen planus

4. The causative agent of erythema multiforme major is usually:
   A. Drug-related
   B. Bacterial
   C. Idiopathic
   D. Viral

5. Important factors in diagnosis and management of the oral ulcer include:
   A. Adequate history
   B. Comprehensive physical exam
   C. Appropriate referrals as indicated
   D. All of the above

present. EM minor (usually idiopathic or secondary to herpetic outbreak) may be treated with burst steroid therapy. EM major (usually adverse drug reaction) and toxic epidermal necrolysis require hospitalization and intensive care.

Other causes

Malignant neoplasms may present as ulcerations at any point in time. The most common malignant neoplasm in the oral cavity is squamous-cell carcinoma. Any non-healing ulcer (more than two weeks) requires incisional scalpel biopsy for diagnosis and appropriate referral for management.

Infectious agents (other than the aforementioned herpes viruses) may also result in oral ulcerations. These run the spectrum from candidiasis to tuberculosis to syphilis. An accurate and detailed history and physical is essential to determining cause. Candidiasis can be managed by the dental professional. Other infectious diseases require referral to an infectious-disease specialist.

Summary

There are many causes of oral ulcerations. Some are easy to diagnose. Some are not. Proper investigative techniques, including history, physical and biopsy (when indicated), allow for accurate diagnosis, management and referrals as indicated so that patients are treated appropriately and expeditiously.

The accompanying quiz will help you gauge your knowledge on this topic.

Questions for the author? Comment on this article at Dentaltown.com/magazine.aspx.

Author Bio

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