Oral Ulcerative Disease

TRAUMATIC ULCERS

Because of the constant motion of the masticatory mucosa over the teeth and the introduction of hard objects into the oral cavity, traumatic ulcers are frequent. Generally, traumatic ulcers are the consequence of mechanical injury resulting from inadvertent biting of the mucosa, from irritation caused by fractured amalgam restorations of prostheses, or from sharp objects introduced into the mouth. Ulcerations may also occur as a consequence of thermal or chemical burns. Symptomatically, the most outstanding feature of traumatic ulcers is pain. Discomfort usually follows 24 to 48 hour after insult to the tissue. The patient can often identify a specific event or object associated with the trauma although, in the case of faulty restorations, a specific event may be absent. The clinical appearance of traumatic ulcers is similar to that of aphthous ulcers. The ulcer is usually ovoid and has a yellowish-white necrotic center surrounded by a broad erythematous border. In the case of mechanical trauma, the lesion usually conforms in area and linearity to the source of the trauma. Hence, any ulceration that appears to be linear is usually the result of trauma.

The diagnosis of traumatic ulcer is based on the history or on the identification of a specific source of irritation. Once the source of irritation is eliminated, traumatic ulcers heal in approximately 10 to 14 days. Treatment of traumatic ulcers is aimed at maintaining cleanliness by frequent rinsing with saline or hydrogen peroxide in water, palliation with anesthetic ointments and elimination of the source of irritation.

APHTHOUS STomatitis

Recurrent aphthous ulcerations, frequently referred to as canker sores, are among the most common lesions of the mouth. Their recurrent pattern and associated discomfort make them extremely bothersome and, at times, debilitating to patients. Aphthous stomatitis may occur as occasional single ulcerations or may be manifested as a never-ending continuum of severe ulcerative lesions.

The prevalence of aphthous stomatitis depends largely on the population studied. Although an overall prevalence of 20% has been reported, individuals in middle and upper middle class economic groups appear to be most frequently affected. An incidence of aphthous stomatitis of 66% was noted among professional students, as compared with only 5% in an indigent population. There appears to be a slight predilection for females. Seasonal peaks have also been noted in winter and spring.

The etiology of aphthous stomatitis has been scrutinized for some time and as yet is not completely resolved. It seems that a specific cause for the condition is identifiable in about 30% of cases. A suspected infectious cause was originally attributed to herpes simplex virus (HSV). However, numerous attempts to isolate HSV from aphthous ulcerations have failed to demonstrate its presence. In addition, no anti-HSV antibodies could be demonstrated in patients with aphthous stomatitis. Furthermore, the relatively high frequency of HSV infection in patients of low economic status is incompatible with the demographic pattern of the disease.

The observation that exacerbations of aphthous stomatitis in women may correlate with the menstrual cycle has led to speculation that there may be a hormonal bases for the disease. However, the nearly equal distribution of aphthous stomatitis in men and women argues against this possibility.
Most likely, multiple factors can initiate aphthous stomatitis. A number of workers have suggested and emotional cause for the disease. The finding of a correlation between stress and severity of the disease, as well as its demographic distribution, supports a psychogenic cause. Animal studies have demonstrated aphthous stomatitis-like lesions following artificially induced stress.

A variety of other conditions have also been identified as causative of true aphthous or aphthous-like lesions. These include deficiencies of vitamin B12, folate, zinc, and iron, cyclic neutropenia, human immunodeficiency virus (HIV) infection, agranulocytoses, Crohn's disease, and food allergies.

The pathophysiologic basis of aphthous stomatitis seems to be clearer. Evidence now suggests an autoimmune mechanism for the tissue destruction noted in aphthous lesions. The histologic appearance of aphthous lesions is one of nonspecific ulceration preceded by a lymphocytic infiltrate. The latter is suggestive of a cell-mediated immune response.

In vitro measurements of cell-mediated immunity (CMI), using lymphocytes from patients with aphthous stomatitis, also support an autoimmune mechanism for the pathophysiology of the disease.

Lymphocyte proliferation (blastogenesis) in response to an antigen correlates with cell-mediated immunity. Lehner allowed peripheral blood lymphocytes from patients with active aphthous stomatitis to react with allogeneic fetal tissue homogenates and measured the amount of lymphocytic proliferation. Lymphocytes from patients with no history of aphthous stomatitis or patients in remission.

A role for CMI in aphthous stomatitis is also suggested by the ability of lymphocytes from patients with aphthous stomatitis to mediate the cytolysis of oral mucosal cells. Dolby found that lymphocytes from patients with active aphthous lesions were able to lyse gingival target cells. This work has been confirmed by Togers and colleagues, who found that, whereas lymphocytes from patients who were free of disease did not effectively lyse oral epithelial target cells, lymphocytes from patients with those of Lefner, suggest that an immune mechanism is probably active in the initiation of the destructive phase of aphthous stomatitis.

Clinically, aphthous stomatitis is characterized by the recurrence of oral ulcerations. The ulcer stage is usually preceded by a 2- to 3-day period in which the patient notes a vague feeling of discomfort. The only clinical finding at this time may be an area of erythema. This is soon followed by the appearance of a deep ulcerative lesion. The lesion is usually ovoid, has some depth, a yellowish-white necrotic base, and a surrounding zone of erythema in a ring. Rarely do lesions exceed 1 cm in diameter. Most commonly, aphthous lesions are found on the buccal, labial, or alveolar mucosa or on the ventral surface of the tongue. It is uncommon for aphthous stomatitis to involve heavily keratinized areas such as the hard palate or attached gingiva. The major symptom associated with aphthous stomatitis is pain of such severity that eating and speaking patterns may be significantly altered. Because it is relatively common for patients to develop edematous enlargement of the tissue surrounding the ulcer, trauma to the lesion may occur inadvertently. Generally, lesions last a maximum of 7 to 14 days, although they are clinically most painful during the early phases of the disease.

The diagnosis of aphthous stomatitis is based on the clinical appearance and course of the lesion and recurrent history. It is usually difficult to establish a specific stressful incident related to the appearance of lesions, although there may be some correlation with the menstrual cycle.
Aphthous lesions must be differentiated from traumatic ulcers, acute herpetic gingivitis, allergy, and erythema multiforme as well as ulcerations caused by systemic disease or conditions described above.

The differentiation between aphthous lesions and thruma is based on the history and the relationship of the lesion to a source of irritation. Essentially, there is no clinical or histologic difference between aphthous and traumatic ulcerations, although any linearity in ulceration suggests a traumatic cause. Acute HSV infection may present as ulcerative lesions after vesicles have ruptured. This is a concurrent phenomenon, usually involving a number of areas in crop-like fashion. Patients with HSV infection may have experienced constitutional symptoms. These lesions tend to occur in a younger, nonsocioeconomically restricted age group with simultaneous gingival involvement. Allergic lesions tend to be more diffuse than aphthous lesions and although erythematous are usually nonulcerative. Erythema multiforme may present with some ulceration, especially after rupture of bullae. However, these lesions are nonrecurrent and are more diffuse than aphthous lesions. It may be difficult to differentiate ulcerations caused by systemic diseases from aphthous stomatitis. The major difference lies in the lack of recurrence and delayed healing time. Any ulcer that fails to heal in 14 days should undergo biopsy.

Although aphthous ulcers heal spontaneously 10 to 14 days after onset, they are extremely painful. The goal of therapy should be to reduce inflammation, minimize pain and discomfort in affected areas, and speed healing. Currently, no medications meet all three goals. A wide range of treatments have been suggested, including antibiotics, immunomodulators, antimicrobial mouthrinses, and dietary supplements. However, topical steroids remain the mainstay for the treatment of aphthous stomatitis in which a specific etiology has not been identified. These are dispensed either as an ointment (triamcinolone acetonide in Orabase) or as a cream (triamcinolone cream). It has been suggested that the ointment form is not as effective as the cream, because the corticosteroid may be inhibited by the bulk of the ointment. If the cream is used, Orabase ointment can be placed over it to help maintain it in position. IN prescribing triamcinolone, it is important to inform the patient to dry the ulcer before application and then to use the drug tid or qid. Some systemic diseases, such as hypertension, preclude the use of corticosteroids. Corticosteroids may also be administered by injection of small amounts of the drug (0.1ml) into the lesion. The disadvantages of this form of treatment are its reliance on professional application and the limitation in dosage.