Oral Complications of Systemic Bacterial and Fungal Infections

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KEYWORDS

• Bacterial infections • Fungal infections • Oral lesions • Ulcers

KEY POINTS

- Infections of the oral cavity can be primary and caused by pathogenic oral microflora or secondary and caused by dissemination of the infections from other systemic organs.
- Disseminated infections should be listed in the differential diagnoses of solitary deep mucosal ulcerative lesions.
- Ulcerative lesions caused by local or systemic infections may be clinically indistinguishable from oral malignancies and other immune-mediated lesions. Additional diagnostic modalities are required to confirm the diagnosis.
- Histopathology and microorganisms staining are helpful in revealing definitive diagnosis.
- The patient's immune status plays a critical role in infection dissemination and majorly affects treatment approaches.

Introduction

The oral cavity is colonized by a variety of microorganisms, beginning shortly after birth. The oral microenvironment provides appropriate nutrients, pH, and temperature for bacterial colonization. Consequently, synergetic and symbiotic polymicrobial communities live in harmony with the surrounding oral environment and form biofilms that adhere to soft and hard oral tissues. Any alterations in the oral microenvironment, by local or systemic factors, may shift this balance and cause proliferation of pathologic microorganisms at the expense of healthy oral microflora.

Dental caries is one of the most common oral diseases attributable to pathogenic microflora, such as streptococcal species, lactobacilli, actinomyces, and prevotellae. The symbiotic relationship between candida and other caries-associated bacteria might also have a role in caries development.[1] Gingivitis, periodontitis, and jaw osteomyelitis are also caused by the pathogenic bacterial oral microflora (mostly anaerobes). Systemic bacterial and fungal infections may also give rise to secondary oral manifestations, especially in individuals with compromised immunity.

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Bacterial infections

Tuberculosis

Tuberculosis (TB) is a systemic bacterial infection that seldom involves the oral cavity. Globally, TB is the second most common death-causing infectious disease after human immunodeficiency virus (HIV).[2] Southeast Asia, Western Pacific Regions, and Africa have the highest prevalence of TB.[2]

Causative pathogen

Mycobacterium tuberculosis is the causative pathogen.[2]

Clinical presentation

TB is known to affect men more than women, mostly adults. Lungs are typically the primary site of infection but other sites may be affected including skin, central nervous system, lymphatic system, kidneys, and gastrointestinal tract.[2,3]

The disease is transmitted by inhalation of contaminated air droplets of a patient with active disease. First-time exposure primarily involves the lungs and may cause nonspecific signs of inflammation, such as fever and pleural effusion (primary TB). This can result in formation of fibrocalcified nodules that are seen on chest radiograph at the primary site of infection (usually at the apex of the lung). Mycobacteria may remain dormant inside these nodules for many years (latent TB). Weakening of the patient's immunity may lead to disease reactivation and spread via vascular and lymphatic systems to other body sites (secondary TB).[3,4]

Oral manifestations

A total of 1% to 5% of affected patients has oral manifestations and 1.33% of the cases are associated with HIV infection.[5] Most oral manifestations of TB are secondary to the primary pulmonary infection. However, primary oral TB may occur from direct inoculation of the organisms in the oral mucosa, in which

younger patients are mostly affected.[3,5] Tongue is the most commonly affected site followed by buccal mucosa, lip, palate, and gingiva (Fig. 1). The lesions are mostly manifested as single chronic ulceration with irregular margins. However, multiple ulcers, swelling, nodular masses, and mandibular osteomyelitis have been reported.[5]

Histopathology
Histopathology shows caseating granulomatous lesion that consists of a mixture of histiocytes, multinucleated giant cells, and lymphocytes with central necrosis (Fig. 2).

Diagnosis
Diagnostic modalities may include tuberculin skin test, special mycobacterial stains of infected tissue (Ziehl-Neelsen stain) (see Fig. 2), culture of infected sputum, and molecular methods (polymerase chain reaction [PCR]).[5]

Differential diagnosis
Oral ulcerative lesion of TB can clinically mimic traumatic ulcer; deep fungal infections; and squamous cell carcinoma.

Treatment
Treatment includes 6-month regimen of pyrazinamide, isoniazid, rifampin, and ethambutol as the first-line treatment of TB. Patients with multiple drug-resistant TB, mainly to isoniazid and rifampicin, require longer treatment regimen for up to 20 months.[2]

Bacterial salivary gland infection

Sialadenitis is inflammation of salivary glands, which can be induced by several etiologic factors including infectious agents (bacterial and viral), radiation, and autoimmune disorders (eg, Sjögren syndrome). Bacterial sialadenitis may be facilitated by hyposalivation. Decreased salivary flow may impede the ability of salivary glands to flush out microorganisms and debris and lead to retrograde spread of the bacterial pathogens from the oral microflora to salivary gland ducts and parenchyma. Less commonly, systemic bacteremia in immunocompromised patients may cause secondary bacterial sialadenitis.[6–8]

Causative pathogens
Acute bacterial sialadenitis
Gram-positive *Staphylococcus aureus* is the most common causative organism in acute bacterial parotitis. However, other bacterial microorganisms, such as other gram-positive cocci (*Streptococcus pyogenes* and *Streptococcus viridans*), gram-negative bacilli (*Prevotella* and *Porphyromonas* species), and gram-negative bacilli (*Escherichia coli*) are also implicated.[6,9]

Chronic bacterial sialadenitis
This is caused by mixed bacterial community. *S viridans* is the most common followed by *S aureus* and *Streptococcus pneumoniae*.[9]

Clinical presentation
Acute bacterial infection
Mucous content of submandibular and sublingual salivary glands acts as protective barrier against bacterial infection. Paucity of mucous secretion renders the parotid glands more susceptible to infection. Acute bacterial parotitis is most commonly seen in elderly and immunocompromised patients. However, neonates and hospitalized patients are also susceptible to this infection. Acute bacterial parotitis is strongly linked to salivary ducts obstruction by sialolith, foreign body, or tumor. Poor oral hygiene, Sjögren syndrome, or drug-induced hyposalivation may also increase the risk of acquiring the acute infection. Clinical manifestations include sudden onset of tender swelling of the auricular area with occasional pus discharge when massaging the infected gland.[8,9]

Chronic bacterial infection
It is characterized by recurrent episodes of painful swelling of salivary glands and chronic purulent discharge (Fig. 3). Both parotid and submandibular glands can be affected. Chronic hyposalivation combined with ascending infection from the oral cavity to the gland ducts/parenchyma is the most common scenario. Congenital malformation of the glands ducts or acini can also impact salivary flow and may be associated with chronic recurrent juvenile parotitis.[9]

Diagnosis
Diagnostic modalities may include bacterial culture, sialography, and biopsy to rule out salivary gland tumors.[9]

Histopathology
Acute bacterial infection
Neutrophils accumulation within salivary gland tissue.

Chronic bacterial infection
Scattered infiltration of lymphocytes and plasma cells with occasional acinar atrophy, dilated ducts and fibrosis are reported.[3]

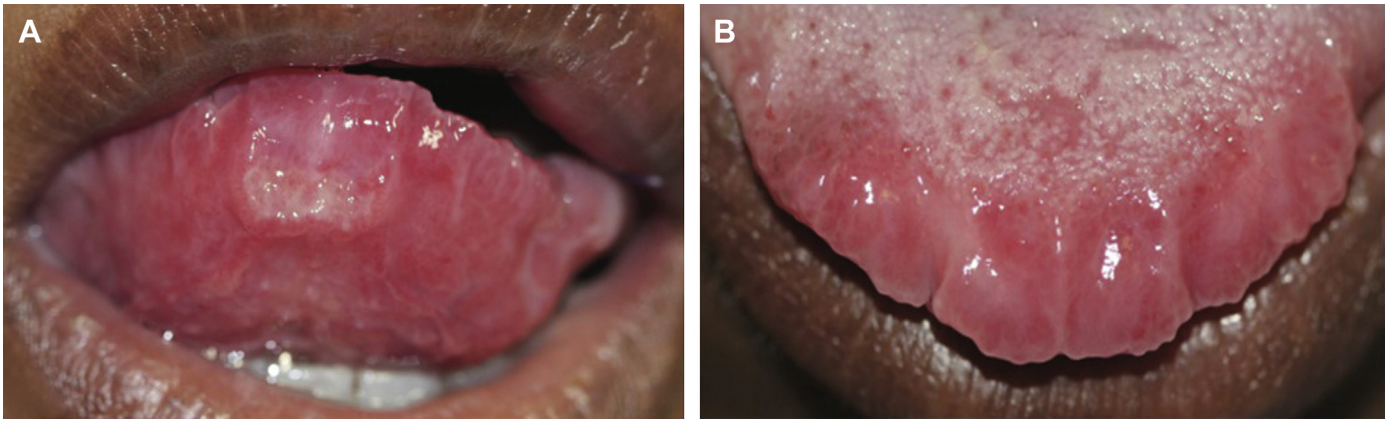

Fig. 1 Primary oral TB. (*A*) Ulcerative lesion on the tongue tip with a surrounded erythematous zone. (*B*) Lobulated erythematous appearance of anterior third of the tongue caused by granulomatous inflammation in the lip submucosa. (*Reproduced from* Khammissa RA, Wood NH, Meyerov R, et al. Primary oral tuberculosis as an indicator of HIV infection. Patholog Res Int 2010;2011:893295.)

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