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Complications, diagnosis, and treatment of odontogenic infections

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INTRODUCTION

Odontogenic infections, consisting primarily of dental caries and periodontal disease (gingivitis and periodontitis), are common and have local (eg, tooth loss) and, in some cases, systemic implications. In the United States, it is estimated that 25 percent of adults over the age of 60 have lost all their teeth (edentulism), approximately one-half from periodontal disease and one-half from dental caries [1,2].

In addition to producing pain and discomfort, odontogenic infections can extend beyond natural barriers and result in potentially life-threatening complications, such as infections of the deep fascial spaces of the head and neck. (See "Deep neck space infections in adults".)

Periodontal infection can also be associated with a number of systemic disorders. These include fever of unknown origin, bacteremic seeding of heart valves and prosthetic devices, preterm birth of low birth weight children, and an increased risk for coronary heart disease and cerebrovascular events.

The complications, diagnosis, and treatment of odontogenic infections will be reviewed here. The epidemiology, pathogenesis, and clinical manifestations of these infections are discussed separately. (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections" and "Overview of gingivitis and periodontitis in adults".)

COMPLICATIONS

Suppurative odontogenic infections may extend to potential fascial spaces in the orofacial area (orofacial space infections) or deep in the head and neck (peripharyngeal space infections). The latter complication is often life threatening. (See "Deep neck space infections in adults".)

In addition, odontogenic infections may spread contiguously to cause osteomyelitis of the jaw or hematogenously to produce systemic illness.

Orofacial space infections — Superficial orofacial space infections can involve the buccal, submental, masticator, canine, and infratemporal spaces. The location of the space infection can assist in recognizing the underlying infected tooth (table 1).

If unrecognized and untreated, these infections are potentially serious since they can spread contiguously into the deeper fascial spaces of the head and neck, such as the submandibular, lateral pharyngeal, and retropharyngeal spaces, or into the carotid sheath. (See "Deep neck space infections in adults".)

The spread of infection into the deeper fascial spaces of the head and neck is suggested by the finding of trismus (the inability to open the jaw). Trismus indicates pressure or infection of the muscles of mastication (the masseter and the pterygoids) or involvement of the motor branch of the trigeminal nerve. Such infections can spread intracranially to cause purulent meningitis or subdural empyema and caudally to result in aspiration, airway obstruction, or fatal necrotizing mediastinitis [3].

Buccal and submental spaces — Infections arising from mandibular or maxillary bicuspid and molar teeth tend to extend in a lateral or buccal direction. The relation of the root apices to the origins of the buccinator muscle determines whether infection will exit intraorally into the buccal vestibule or extraorally into the buccal space (figure 1). Infection of the buccal space is readily diagnosed because of marked cheek swelling with minimal trismus and systemic symptoms.

Involvement of a mandibular incisor can perforate below the mentalis muscle and present as a submental space infection. The chin appears grossly swollen and is firm and erythematous.

Masticator spaces — Masticator spaces consist of the masseteric, pterygoid, and temporal spaces, all of which are well differentiated but intercommunicate with each other as well as with the buccal, submandibular, and lateral pharyngeal spaces (figure 2). Infection of the masticator spaces arises most frequently from molar teeth, particularly the third molars

(wisdom teeth). The clinical hallmark of masticator space infection is trismus and pain in the area of the body or ramus of the mandible.

Swelling may not be a prominent finding, especially in the masseteric compartment, since the infection is beneath large muscle masses that can obscure or prevent clinically apparent swelling. When present, swelling tends to be brawny and indurated, suggesting the possibility of cervicofacial actinomycosis or mandibular osteomyelitis. (See "Cervicofacial actinomycosis".)

Infection of the deep temporal space usually originates from involvement of the posterior maxillary molar teeth. Little external swelling is observed early in the course; if present, it usually affects the preauricular region and an area over the zygomatic arch. As infection progresses, the cheek, eyelids, and whole side of the face may be involved. Infection may extend directly into the orbit via the inferior orbital fissure and produce proptosis, optic neuritis, and abducens nerve palsy.

Canine and infratemporal spaces — Involvement of the maxillary incisors and canines may result in a canine space infection, which manifests as dramatic swelling of the upper lip, canine fossa, and frequently the periorbital tissues. Pain is usually moderate, and systemic signs are minimal. Occasionally, direct extension of infection into the adjoining antrum leads to purulent maxillary sinusitis.

The infratemporal space is bounded medially by the lateral plate of the pterygoid process and the pharynx, posteriorly by the parotid gland, anteriorly by the maxilla, and superiorly by the roof of the infratemporal fossa, adjacent to which is the inferior orbital fissure (figure 2).

Primary infections of the infratemporal fossa usually originate from involvement of the posterior maxillary molar teeth, particularly the third molar [4]. Injection of local anesthetic into this area for a dental restoration may predispose to infection.

Clinically, marked trismus and pain are present but very little swelling is observed early in the course. Late manifestations are similar to those of temporal space infections, including extension into the orbit through the inferior orbital fissure. In addition, if the infection extends internally, it can involve an area close to the lateral pharyngeal wall, resulting in dysphagia.

Osteomyelitis of the jaw — Odontogenic infections can spread contiguously to cause osteomyelitis of the jaw. The mandible is much more susceptible to osteomyelitis than the maxilla, primarily because the cortical plates of the former are thin and vascular supply to the medullary tissues is relatively poor. Despite this, osteomyelitis secondary to odontogenic infection is relatively uncommon. When it does occur, there is usually a predisposing condition, such as compound fracture, irradiation, diabetes mellitus, or steroid therapy [5].

With initiation of infection, the intramedullary pressure markedly increases, further compromising blood supply and leading to bone necrosis. Pus travels through the Haversian and perforating canals, accumulates beneath the periosteum, and elevates it from the cortex. If pus continues to accumulate, the periosteum is eventually penetrated, and mucosal or cutaneous abscesses and fistulae can develop. Areas at greatest risk of perforation in the mandible are the lingual aspect in the region of molar teeth and anteriorly on the buccal aspect [3].

As the inflammatory process becomes more chronic, granulation tissue is formed. Spicules of necrotic and nonviable bone may become either totally isolated (sequestrum) or encased in a sheath of new bone (involucrum).

Severe mandibular pain is a common symptom of jaw osteomyelitis and can be accompanied by anesthesia or hypoesthesia on the affected side. In protracted cases, mandibular trismus may develop.

A clinical variant of osteomyelitis of the jaw is Garre chronic sclerosing osteomyelitis or proliferative periostitis [6]. This entity usually occurs in children and young adults following a periapical infection of the mandibular first molar. It is a nonsuppurative form of osteomyelitis characterized by a localized, hard, nontender swelling over the mandible. On radiographic imaging, the newly formed periosteal bone appears as layers outside the cortex, giving a characteristic "onion skin" appearance [7]. Actinomycosis and radiation necrosis are two common causes of this form of osteomyelitis of the jaw [5,8].

A related clinical entity, characterized by exposed necrotic bone in the maxillofacial region, is osteonecrosis of the jaw associated with long-term administration of bisphosphonates for the treatment of osteoporosis or cancer. The prevalence of this condition appears to have escalated, particularly among older female patients [9]. This is discussed in detail separately. (See "Risks of therapy with bone antiresorptive agents in patients with advanced malignancy", section on 'Osteonecrosis of the jaw'.)

Hematogenous dissemination — In addition to contiguous spread, odontogenic infections can disseminate hematogenously to seed native or prosthetic heart valves, joints, or other devices [10]. Although bacteremia can occur following almost all types of dental manipulations, including flossing, scaling, even tooth brushing and chewing hard candy, such episodes are usually transient and inconsequential in healthy individuals [11]. In contrast, the bacteremia in patients with dental caries and periodontal disease tends to be more frequent and sustained [12] and is a potentially important cause of infective endocarditis in older patients, who may be

more susceptible due to age-related degenerative or calcified valvular heart disease [13]. (See "Native valve endocarditis: Epidemiology, risk factors, and microbiology".)

Dental sources of bacteremia in older adults are of increasing concern for those undergoing prosthetic heart valve implantation or prosthetic joint replacement. It has been recommended that routine dental assessment be performed in all patients undergoing valve surgery and that appropriate therapeutic interventions be initiated whenever possible before valve implantation [14]. Recommendations regarding the use of antimicrobial prophylaxis for the prevention of bacterial endocarditis in selected patients are presented separately. (See "Prevention of endocarditis: Antibiotic prophylaxis and other measures".)

The risk of prosthetic joint infection as a consequence of bacteremia following dental procedures has been controversial. This is also discussed in detail separately. (See "Prevention of prosthetic joint and other types of orthopedic hardware infection", section on 'Dental procedures'.)

Association with cardiovascular disease — An association between poor oral health and chronic periodontitis with coronary and cerebrovascular disease has been well established epidemiologically [15]. The relevant data and possible mechanisms are discussed separately. (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections", section on 'Association with cardiovascular risk'.)

DIAGNOSIS

Obtaining appropriate material for culture and processing it properly are important in the diagnosis of odontogenic infections. In addition, imaging techniques to assess the extent of involvement are essential.

Specimen collection and processing — One of the difficulties in defining etiologic agents for odontogenic infections is the presence of normal resident oral flora. For closed space infections, it is imperative that the normal oral flora be excluded during specimen collection in order to interpret culture results. Needle aspiration of loculated pus by an extraoral approach is desirable, and specimens should be transported immediately to the laboratory under anaerobic conditions.

However, contamination by the resident oral flora is inevitable for intraoral lesions. In this setting, direct microscopic examination of stained smears (eg, Gram stain for bacteria, calcofluor white stain for fungi) often provides more useful information than culture results from surface swabs. Tissue biopsies should be routinely examined for histopathologic evidence

of acute or chronic inflammation and infection. Specific microbial agents, including certain bacterial, mycobacterial, fungal and viral infections, can sometimes be detected by immunofluorescence or polymerase chain reaction.

Patients with chronic osteomyelitis often have soft tissue swelling and draining fistulae. Bone biopsies for histopathology and culture are often required for definitive diagnosis. Aspiration of soft tissue swelling for Gram stain and culture is not helpful. Cultures from sinus tracts can be obtained but might be misleading. Since there is communication between the sinus tract and external environment, isolated microorganisms could represent colonization of the sinus tract rather than the organism responsible for the infected bone. (See "Nonvertebral osteomyelitis in adults: Clinical manifestations and diagnosis".)

Imaging techniques — The choice of imaging technique varies with the clinical setting [16]:

- A panoramic or periapical radiograph may reveal the extent of advanced periodontitis or the presence of periapical abscess.

- Computed tomography (CT) is particularly sensitive for osseous structures and remains the imaging modality of choice for assessment of most odontogenic infections [16]. Cone-beam CT is a fairly novel technique that reduces the amount of radiation and has become an important tool in the diagnosis of head and neck pathology, including odontogenic infection [17,18]. Dental amalgam used in fillings may cause metallic streaking artifact on CT and obscure the region of interest. This problem can be minimized with multidetector CT imaging.

- For patients suspected of having deep fascial space infections of the head and neck, CT and magnetic resonance imaging (MRI) have become the preferred imaging modalities. In retropharyngeal infections, these can help determine if the infection is in the retropharyngeal space or the prevertebral space. The former suggests an odontogenic source, while the latter suggests involvement of the cervical spine. A lateral radiograph of the neck may demonstrate compression or deviation of the tracheal air column, or the presence of gas within necrotic soft tissues. (See "Deep neck space infections in adults", section on 'Clinical suspicion and urgent imaging'.)

- For patients with suspected osteomyelitis, the general approach to imaging is discussed in detail elsewhere. (See "Approach to imaging modalities in the setting of suspected nonvertebral osteomyelitis", section on 'Selecting an imaging modality'.)

  We do not typically use bone scan for patients with suspected bony complications of odontogenic infections. One study reported that bone scan had a 7 percent specificity and

62 percent positive predictive value for the diagnosis of mandibular osteomyelitis [19]. In addition, any dental work or trauma can make interpretation of bone scan impossible, as bony turnover can still cause false-positive technetium uptake for more than two years. Technetium bone scanning may be helpful for the diagnosis of early acute osteomyelitis if metallic scatter from dental fillings makes CT difficult to interpret. However, in chronic osteomyelitis, the technetium scans may or may not be positive, and gallium- or indium-labeled scans are often negative.

THERAPEUTIC CONSIDERATIONS

Meticulous attention to oral hygiene is the most important strategy for effective control of supragingival and subgingival plaque that, in turn, is essential for both caries prevention and the treatment of periodontitis. Individuals with physical or mental limitations who cannot adequately perform oral hygiene by themselves should receive daily oral hygiene by care providers. More frequent visits to dentists and use of electric toothbrushes should also be considered in these patients. Regular check-ups and prompt restorative care by dental professionals should be actively promoted.

With the recognition of the microbial specificity of odontogenic infections, topical antiseptics and systemic antibiotics have played an increasingly important role in the control and treatment of both dental caries and periodontal disease (table 2).

The need for dental extractions has been reduced considerably by the availability of improved dental restorative materials, such as bonding and fluoride-releasing agents, as well as improved restorative care [20].

Dental caries — Caries management with restorative therapy (eg, fillings) is the preferred therapeutic approach in many countries [20]. However, restorative therapy must be combined with preventive measures, since restorations have relatively short durability and new caries may form at the margins of restorations if the causes of the disease persist [1,20]. Caries prevention is discussed below. (See 'Prevention' below.)

Pulpitis — Pulpitis, inflammation of the dental pulp, occurs when progression of dental caries exposes the dental pulp, leading to infection (figure 3). The early and dominant symptom of acute pulpitis is a severe toothache that can be elicited by thermal changes, especially cold drinks. (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections", section on 'Pulpitis and periapical abscess'.)

Pulpitis can be classified as reversible or irreversible. Reversible pulpitis occurs when caries encroach on the pulp and is associated with mild inflammation of the pulp. Irreversible pulpitis refers to ongoing inflammation within the pulp chamber with rapid buildup of pressure, occlusion of blood vessels at the apical foramen, ischemia, and necrosis of the pulp tissue.

Irreversible pulpitis is characterized by acute and intense pain and is one of the most frequent reasons that patients seek emergency dental care. Apart from removal of the tooth, the customary approach to relieving the pain of irreversible pulpitis is by drilling into the tooth, removing the inflamed pulp (nerve), and cleaning the root canal. A minority of dentists begins with a trial of antibiotics and analgesics, although there is no proof of benefit from this approach.

A 2013 literature review found only one randomized controlled trial that compared treatment with systemic antibiotics to placebo in irreversible pulpitis [21]. In this trial, 40 patients were treated with analgesics and randomly assigned to either penicillin or placebo [22]. No operative endodontic treatment was performed. Although the study was limited by small sample size, no significant difference in pain intensity or use of analgesics was noted between the two groups. Photoactivated disinfection has been evaluated as an aid to mechanical irrigation in eradicating bacterial counts within the root canal during endodontic treatment [23]. It was highly effective in eliminating endodontic pathogens from the root canal [24,25]. It remains to be determined whether this approach provides incremental benefit compared to conventional endodontic treatment in symptom relief or disease progression.

Necrotizing periodontal disease — Acute necrotizing ulcerative gingivitis, also known as Vincent angina or trench mouth, should be treated with systemic antimicrobials, such as metronidazole, amoxicillin-clavulanate, or clindamycin (table 2) [26,27]. Acute necrotizing ulcerative gingivitis and periodontitis are discussed in detail elsewhere. (See "Overview of gingivitis and periodontitis in adults", section on 'Necrotizing periodontal disease'.)

Plaque-associated acute gingivitis — Acute simple gingivitis rarely requires systemic antimicrobial therapy [27,28]. Chlorhexidine 0.12% oral rinse can be used until symptoms resolve.

Systemic antibiotics are usually indicated for patients with rapidly advancing disease, severe pain, or immunocompromising condition. Possible regimens include penicillin plus metronidazole, amoxicillin-clavulanate, or clindamycin (table 2).

Rarely, patients with intense gingivostomatitis may warrant intravenous antibiotics if they cannot tolerate oral intake.

We continue antibiotics until oral lesions have healed and pain has subsided, generally for five to seven days.

Mechanical debridement (scaling and root planing of subgingival biofilm and calculus) by a dental professional and augmented oral hygiene are also warranted. (See "Overview of gingivitis and periodontitis in adults", section on 'Treatment of plaque-associated gingivitis and periodontitis' and "Overview of gingivitis and periodontitis in adults", section on 'Necrotizing periodontal disease'.)

Plaque-associated periodontitis — Due to microbial specificity in various forms of periodontitis, certain types of severe periodontitis are amenable to topical or systemic antimicrobials in conjunction with mechanical debridement (scaling and root planing) (table 2) [29]. This approach has often obviated the need for radical surgical resection of periodontal tissues.

- **Periodontitis in children or severe periodontitis in adults** − For juvenile periodontitis [30-32] and severe adult periodontitis (Stage III or Stage IV, as indicated by the 2017 World Workshop Classification of Periodontal and Peri-implant Diseases and Conditions, based on severity and complexity of management [33]), we use amoxicillin plus metronidazole or amoxicillin-clavulanate. For patients who are penicillin allergic but who can take cephalosporins, we use an oral cephalosporin plus metronidazole. For patients who cannot take cephalosporins, other alternatives include clindamycin, azithromycin, clarithromycin, and doxycycline [34]. Clindamycin has good activity against gram-positive anaerobes but poor activity against most gram-negative anaerobes; the other antibiotics generally have poor activity against anaerobes (see "Anaerobic bacterial infections", section on 'Dental infections'). Doxycycline should be used with caution in children under eight years of age due to a small risk of tooth staining, especially with prolonged (≥21 days) courses [34] (see "Tetracyclines", section on 'Young children'). A 2015 panel of experts convened by the American Dental Association Council on Scientific Affairs recommended consideration of a three to nine-month course of oral low-dose (20 mg twice daily) doxycycline as an adjunct for scaling and root planning moderate to severe chronic periodontitis, although the evidence supporting this practice is sparse [35]. In a randomized clinical trial of patients with severe chronic periodontitis, the combination of metronidazole (250 mg or 400 mg orally three times daily) plus amoxicillin (500 mg orally three times daily) for 14 days was superior to scaling and root planing alone after one year of follow-up [36]. An earlier trial of 94 patients with advanced periodontitis had demonstrated that metronidazole or doxycycline for one to two weeks in conjunction with rigorous mechanical debridement of the root surfaces reduced the need for radical

surgery by 81 percent compared with placebo [37]. However, metronidazole alone does not provide coverage against some relevant organisms (eg*, Aggregatibacter actinomycetemcomitans), and* tetracycline resistance among periodontal pathogens has been increasingly recognized [38].

- **Nonsevere periodontitis in adults** − For nonsevere adult periodontitis (Stage I or Stage II, as indicated by the 2017 World Workshop Classification of Periodontal and Peri-implant Diseases and Conditions, based on severity and complexity of management [33]), topical antibiotic formulations that release controlled amounts of the antibiotic beneath the gum are used in conjunction with scaling and root planing to reduce pocket depth. Such adjunctive local antibiotics have been shown to reduce pocket depth or degree of periodontal detachment [28]. Effective agents include 2% minocycline hydrochloride microspheres (Arestin), 10% doxycycline hyclate extended-release liquid (Atridox), chlorhexidine periodontal chips, and 25% metronidazole gel (Elyzol); 25% metronidazole gel is not available in the United States and Canada [28,39,40].

The routine use of systemic antimicrobials to prevent postoperative infections following oral and/or periodontal surgery in a healthy host remains controversial [39]. In a blinded, placebo-controlled trial of 118 patients undergoing third molar extractions, a single dose of intravenous (IV) penicillin was shown to significantly reduce the incidence of surgical site infection (8.5 versus 0 percent) [41].

Duration of antibiotic therapy for periodontitis is generally seven to 14 days. For patients with mild periodontitis whose symptoms have resolved, seven days of topical antibiotic therapy is usually sufficient. For severe periodontitis, we generally continue antibiotics for 14 days or until clinical resolution [42].

Suppurative odontogenic infections

Drainage — The most important therapeutic modality for pyogenic odontogenic infections is drainage and removal of necrotic tissue. Needle aspiration by the extraoral route can be particularly helpful both for microbiologic sampling and for evacuation of pus. In a study of 15 patients with oral-maxillofacial abscesses who underwent needle aspiration of abscess, all patients reported a reduction in pain and none required further surgical incision and drainage [43]. The need for definitive restoration or extraction of the infected tooth, the primary source of infection, is usually readily apparent. Deep periodontal scaling and endodontic treatments with root filling is required in most instances.

Effective surgical management requires a thorough understanding of the most likely anatomic routes of spread. The neighboring potential fascial spaces should be carefully and

systematically surveyed. The optimum timing for incision and drainage is equally important. Premature incision into an area of cellulitis in an ill-conceived search for pus can disrupt the normal physiologic barrier and cause extension of infection.

Antibiotic therapy — Antibiotic therapy can halt the local spread of infection and prevent hematogenous dissemination.

Indications — Antimicrobial agents are generally indicated if fever and regional lymphadenopathy are present, or when infection has perforated the bony cortex and spread into surrounding soft tissue. Severely immunocompromised patients are particularly at risk for spreading orofacial infections, and empiric broad spectrum antimicrobial therapy in these patients is warranted [3,44].

Initial empiric therapy — For patients with pyogenic odontogenic infections, we favor parenteral therapy initially. However, for adult patients with mild infections and without comorbidities or signs of sepsis, it is reasonable to give an oral regimen while arranging for the patient to see a dentist or oral surgeon. Oral regimens are discussed elsewhere. (See 'Oral step-down therapy and duration of therapy' below.)

The choice of specific antibiotics for the treatment of odontogenic infections is based more upon knowledge of the indigenous organisms that colonize the teeth, gums, and mucous membranes, as well as specific cariogenic and periodontopathic pathogens associated with clinical disease, rather than upon the results of culture and susceptibility testing [27,28]. The microbiology and, thus, the empiric regimens differ between immunocompetent and severely immunocompromised (eg, patients with leukemia and severe neutropenia after chemotherapy) (table 3):

- **Immunocompetent patients** − For most patients, ampicillin-sulbactam (3 g IV every six hours) is the treatment of choice. An alternative is penicillin G (2 to 4 million units IV every four to six hours) in combination with metronidazole (500 mg IV or orally every eight hours) (table 3). These regimens cover mouth flora streptococci and anaerobes, including those that produce beta-lactamase. Beta-lactamase production among oral anaerobes, particularly pigmented *Prevotella* spp and *Fusobacterium* spp, is increasingly recognized, and treatment failure with penicillin alone has been well documented [45]. Thus, penicillin monotherapy is no longer recommended. Although metronidazole is highly active against anaerobic gram-negative bacilli and spirochetes, it is only moderately active against anaerobic cocci and is not active against aerobes, including streptococci. As a result, it should not be used as a single agent in suppurative odontogenic infections.

In patients with a nonsevere penicillin allergy, we prefer second- or third-generation cephalosporin (eg, cefoxitin 2 g IV every six hours, cefotetan 2 g IV every 12 hours, or ceftriaxone 2 g IV every 24 hours) plus metronidazole (500 mg IV or orally every eight hours). For patients with severe penicillin allergies who cannot tolerate cephalosporins, options include metronidazole (500 mg IV or orally every eight hours) plus levofloxacin (750 mg IV or orally every 24 hours) or meropenem (1 g IV every eight hours). Clindamycin is not recommended for initial empiric therapy due to resistance among mouth flora streptococci, such as *Streptococcus anginosus* (*milleri*) group (20 to 30 percent), and among anaerobes, such as *Prevotella* spp and *Porphyromonas* spp (31 to 38 percent) [46,47]. Macrolides (eg, erythromycin, clarithromycin, azithromycin) and tetracyclines (eg, doxycycline) are not recommended because of increasing resistance among some strains of streptococci and their lack of optimal anaerobic activity [48,49].

- **Immunocompromised patients** − For severely immunocompromised patients, it is prudent to additionally cover for facultative gram-negative bacilli (including *Pseudomonas* spp) with agents that have broad-spectrum activity against both aerobes and anaerobes. Appropriate regimens are listed in the table (table 3).

In addition, for patients with risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) infection, we typically add vancomycin (or other MRSA-active antibiotic) to the initial empiric regimen; if MRSA is not isolated on cultures, coverage for this organism can be discontinued. Risk factors for MRSA and antibiotics active against MRSA are discussed elsewhere. (See "Methicillin-resistant Staphylococcus aureus (MRSA) in adults: Epidemiology", section on 'Risk factors' and "Methicillin-resistant Staphylococcus aureus (MRSA) in adults: Treatment of bacteremia", section on 'Treatment regimens'.)

Oral step-down therapy and duration of therapy — For patients with odontogenic soft tissue infections, we continue IV therapy until there is evidence of clinical improvement, which usually occurs within three to five days following initiation of antibiotics and abscess drainage in those with severe infections and within a shorter period in those with mild to moderate infections. Some patients with mild infection may also receive oral therapy initially, pending dental or surgical evaluation.

The preferred oral regimen is amoxicillin-clavulanate (875 mg orally twice daily). For penicillin-allergic patients who can tolerate cephalosporins, we usually give a second- or third-generation oral cephalosporin (eg, cefuroxime 500 mg orally twice daily, cefdinir 300 mg orally twice daily, or cefpodoxime 400 mg orally twice daily) plus metronidazole. If both penicillins and cephalosporins cannot be tolerated, we prefer levofloxacin plus metronidazole. Clindamycin 300

mg to 450 mg orally three times daily is another option but should be avoided if clindamycin resistance is suspected (eg, based on local epidemiology) or documented.

Antibiotics should be continued until local inflammation has resolved completely, typically for a total of 7 to 14 days.

Osteomyelitis — Treatment of osteomyelitis of the jaw is complicated by the presence of teeth and persistent exposure to the oral environment. Antibiotic regimens should ideally be informed by culture of a bone biopsy specimen. When culture results are uninformative or unavailable, we use antibiotics with activity against streptococci and anaerobes. Appropriate antibiotic regimens include ampicillin-sulbactam, ceftriaxone plus metronidazole, or ertapenem. For immunocompromised patients who are at increased risk for *Pseudomonas aeruginosa*, appropriate antibiotic regimens include piperacillin-tazobactam, cefepime plus metronidazole, or meropenem (table 3). Oral options for jaw osteomyelitis include clindamycin or levofloxacin but should only be used when susceptibility has been confirmed by culture data. Antibiotic therapy needs to be prolonged, often for weeks to months.

The benefit of adjuvant therapy with hyperbaric oxygen has not been demonstrated [50,51]. (See "Hyperbaric oxygen therapy".)

Surgical management, including sequestrectomy, saucerization, decortication, and closed-wound suction irrigation, is occasionally necessary. Rarely, in advanced cases, the entire segment of the infected jaw has to be resected [8].

PREVENTION

The single most cost-effective measure for reducing dental caries is fluoridation of public water supplies [52,53]. Fluoride forms a complex with the apatite crystals in dentin by replacing the hydroxyl group, thereby lending strength to the entire structure. Fluoride also promotes remineralization of carious lesions and exerts a bacteriostatic effect.

In addition to fluoridated water, brushing two to three times daily with a fluoridated toothpaste (1000 ppm of fluoride, usually as sodium fluoride 1.1% or stannous fluoride 0.4%) effectively delivers fluoride to the tooth-plaque surface [54]. In high-risk individuals, additional fluoride therapy in the form of fluoride varnishes (22,600 ppm fluoride as 5% sodium fluoride, professionally applied three or four times a year) has been effective in caries prevention [20].

The key for the prevention and control of dental caries and advanced periodontitis is the active promotion of oral hygiene. The components of such a regimen include:

- Regular brushing with a fluoridated toothpaste and dental flossing after each meal
- Dietary counseling to reduce the ingestion of sugar-rich foods or beverages
- Use of topical fluorides and oral antimicrobial rinses, such as chlorhexidine for high-risk patients
- Modification of risk factors, such as smoking cessation
- Overcoming the reluctance for regular visits to dental professionals

It is also important to dispel the common misconception by physicians and patients that progressive dental caries, gingival disease, and loss of teeth is an inevitable and irreversible part of aging. The current lack of motivation of both patients and healthcare providers for regular and rigorous preventative dental care must be reversed.

A variety of other measures have been effective for caries prevention. None of these measures is routinely applied in clinical practice, but they are useful in selected patients with rampant caries [55].

- Xylitol gum, a nonfermentable five-carbon sugar, as a sugar substitute between meals [56].

- Oral antimicrobial rinses with 0.12% chlorhexidine help to control dental plaque bacteria but have no proven effect on dental caries [55,57]. On the other hand, a meta-analysis found that application of 0.12% chlorhexidine varnish during fixed orthodontic treatment was effective in reducing caries incidence [58]. Chlorhexidine has a bitter taste, stains the enamel and tongue, and prolonged application can promote the emergence of resistant microorganisms.

- Among topical antibiotics, only vancomycin has been shown to reduce dental caries with some degree of success in humans [55,59].

- Oral probiotics may be effective for caries prevention and treatment of periodontal disease in certain high-risk populations [60-62]. More investigations are necessary to define their role in oral health and disease.

Vaccines based upon various immunogens derived from *Streptococcus mutans*, the principal bacterial agent associated with dental caries, have been explored [63]. However, the prospect for an effective and safe vaccine remains remote and unlikely to be available for clinical application in the near future. The need for and appropriate frequency of routine dental scaling and polishing in patients at low risk for periodontal disease is uncertain [64].

ANTIMICROBIAL PROPHYLAXIS

Recommendations regarding the use of antimicrobial prophylaxis to prevent bacterial endocarditis and prosthetic joint infections in selected patients undergoing dental procedures are presented separately. (See "Prevention of endocarditis: Antibiotic prophylaxis and other measures" and "Prevention of prosthetic joint and other types of orthopedic hardware infection", section on 'Dental procedures'.)

SUMMARY AND RECOMMENDATIONS

- **Definition** − Odontogenic infections consist primarily of dental caries and periodontal disease (gingivitis and periodontitis), are common and have local (eg, tooth loss) and, in some cases, systemic implications. (See 'Introduction' above.)
- **Complications** – Odontogenic infections can have local (eg, tooth loss) and systemic implications. Suppurative odontogenic infections may extend to potential fascial spaces in the orofacial area (orofacial space infections) or deep in the head and neck (peripharyngeal space infections) and can also result in osteomyelitis of the jaw or hematogenous dissemination, which may in turn cause endocarditis or prosthetic joint infections. (See 'Complications' above.)
- **Specimen collection and processing** − For closed space infections, it is imperative that the normal oral flora be excluded during specimen collection in order to interpret culture results. Needle aspiration of loculated pus by an extraoral approach is desirable, and specimens should be transported immediately to the laboratory under anaerobic conditions. (See 'Specimen collection and processing' above.)
- **Preferred imaging modality** − The choice of imaging technique varies with the clinical setting. Computed tomography (CT) is particularly sensitive for osseous structures and remains the imaging modality of choice for assessment of most odontogenic infections. (See 'Imaging techniques' above.)
- **Management**
  - **Oral hygiene** − Meticulous attention to oral hygiene is essential for both caries prevention and the treatment of periodontitis. (See 'Therapeutic considerations' above.)
  - **Pulpitis** − The customary approach to relieving the pain of irreversible pulpitis is by drilling into the tooth, removing the inflamed pulp (nerve), and cleaning the root canal.

Sometimes tooth removal is necessary. (See 'Pulpitis' above.)

- **Acute simple gingivitis** − Acute simple gingivitis rarely requires systemic antimicrobial therapy. Chlorhexidine 0.12% oral rinse can be used in most cases. Antibiotics are usually indicated for patients with rapidly advancing disease, severe pain, or an immunocompromising condition. Possible regimens include penicillin plus metronidazole, amoxicillin-clavulanate, or clindamycin (table 2). (See 'Plaque-associated acute gingivitis' above.)

- **Acute necrotizing ulcerative gingivitis** − Acute necrotizing ulcerative gingivitis, also known as Vincent angina or trench mouth, should be treated with systemic antimicrobials, such as metronidazole, amoxicillin-clavulanate, or clindamycin (table 2). (See 'Plaque-associated acute gingivitis' above.)

- **Periodontitis** − Certain types of severe periodontitis (Stage III or IV) are amenable to systemic antimicrobials in conjunction with mechanical debridement (scaling and root planing) (table 2). This approach has often obviated the need for radical surgical resection of periodontal tissues. For less severe periodontitis (Stage I or II), a topical antibiotic approach is used in conjunction with scaling and root planing. (See 'Plaque-associated periodontitis' above.)

- **Suppurative odontogenic infections**

  - Surgical drainage and removal of necrotic tissue is the most important therapeutic modality for pyogenic odontogenic infections. Needle aspiration by the extraoral route can be particularly helpful both for microbiologic sampling and for evacuation of pus. The need for definitive restoration or extraction of the infected tooth, the primary source of infection, is usually readily apparent. Deep periodontal scaling and endodontic treatments with root filling is required in most instances. (See 'Suppurative odontogenic infections' above.)

  - Antimicrobial therapy is generally indicated in addition to surgical management if fever and regional lymphadenopathy are present, or when infection has perforated the bony cortex and spread into surrounding soft tissue. Ampicillin-sulbactam (3 g intravenously every six hours) provides extended coverage against oral anaerobes, including those that produce beta-lactamases, and is the treatment of choice in immunocompetent patients (table 3). (See 'Antibiotic therapy' above.)

- **Prevention** − The single most cost-effective measure for reducing dental caries is fluoridation of public water supplies. Other preventive measures include regular brushing

with a fluoridated toothpaste, dental flossing, and reducing the ingestion of sugar-rich foods or beverages. (See 'Prevention' above.)

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Topic 3416 Version 33.0

GRAPHICS

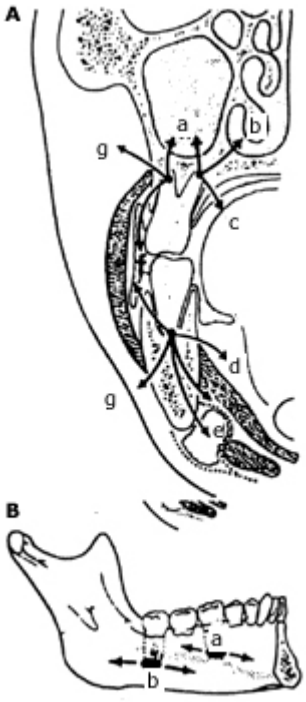
Clinical features of odontogenic orofacial and peripharyngeal "space" infections

Space infections	Usual site of origin	Clinical features				
		Pain	Trismus	Swelling	Dysphagia	Dyspnea
Masticator						
Masseteric and pterygoid	Molars (especially 3rd)	+	+++	May not be evident (deep)	-	-
Temporal	Post. maxillary molars	+	-	Face, orbit (late)	-	-
Buccal	Bicuspids, molars	±	±	Cheek (marked)	-	-
Canine	Maxillary canines, incisors	++	-	Upper lip, canine fossa	-	-
Infratemporal	Post. maxillary molars	+	++	Face, orbit (late)	±	±
Submental	Mandibular incisors	++	-	Chin (firm)	-	-
Submandibular	Masseteric spaces	+++	-	Angle of jaw (marked)	-	-
Sublingual	2nd, 3rd mandibular molars	+	±	Submandibular (brawny)	-	-
Lateral pharyngeal						
Anterior	Mandibular incisors	+	±	Floor of mouth (tender)	+ (if bilateral)	+ (if bilateral)
Posterior	Masticator spaces	+++	+++	Angle of jaw	+	±
Retropharyngeal (and "danger")	Masticator spaces	±	±	Post. pharynx	+	+++
	Lateral pharyngeal space, distant via lymphatics	+	±	Post. pharynx (midline)	+	+
Pretracheal	Retropharyngeal space, anterior esophagus	+	-	Hypopharynx	+	+++

±: minimal or occasional; +: present; ++: moderate; +++: prominent or severe.

Graphic 71760 Version 3.0

Routes of spread of odontogenic orofacial infections



Spread occurs along planes of least resistance.

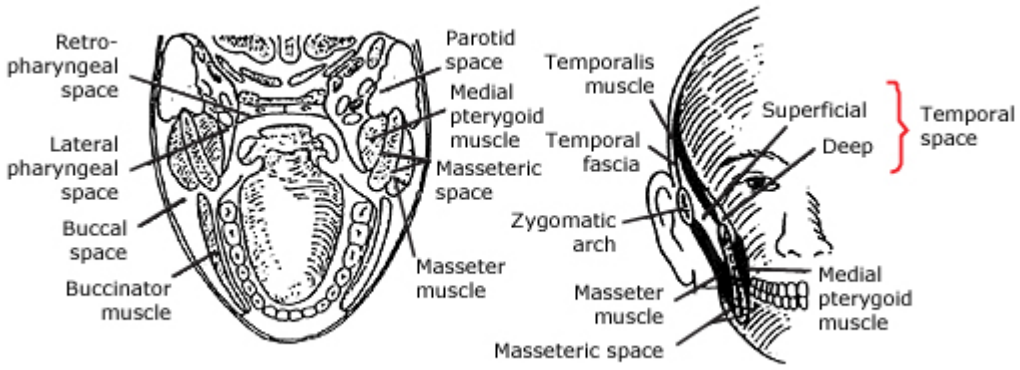
Top panel: Coronal section in the region of the first molar teeth: (a) maxillary antrum; (b) nasal cavity; (c) palatal plate; (d) sublingual space (above the mylohyoid muscle); (e) submandibular space (below the mylohyoid muscle); (f) intraoral presentation with infection spreading through the buccal plates inside the attachment of the buccinator muscle; (g) extraoral presentation to buccal space with infection spreading through the buccal plates outside the attachment of the buccinator muscle.

Bottom panel: Lingual aspect of the mandible: (a) apices of the involved tooth above the myohyoid muscle, with spread of infection to the sublingual space; (b) apices of involved tooth below the mylohyoid muscle, with spread of infection into the submandibular space.

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Graphic 77988 Version 3.0

Fascial spaces around the mouth and face



Left panel: Horizontal section at the level of the occlusal surface of the mandibular teeth.

Right panel: Frontal view of the face.

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Graphic 80359 Version 3.0

Antimicrobial regimens for the prevention of dental caries and the treatment of periodontal disease in adults

Clinical entity	Common causative organisms	Antimicrobial regimens
Supragingival dental plaque and dental caries prevention	*Streptococcus mutans*, other streptococci, *Actinomyces* spp	Fluoride-containing toothpaste (sodium fluoride, 1.1% or stannous fluoride, 0.4%) 2 or 3 times daily **AND/OR**
		Fluoride-containing varnishes (sodium fluoride, 5%) applied 3 o 4 times yearly **AND/OR**
		Chlorhexidine, 0.12% oral rinse
		Chlorhexidine chips
		25% metronidazole gel*
Ulcerative or acute necrotizing ulcerative gingivitis/periodontitis[¶]	*Prevotella intermedia*, *Fusobacterium* spp, *Tannerella forsythia*, *Treponema denticoli*, other oral spirochetes	Metronidazole 500 mg PO every hours **OR**
		Amoxicillin-clavulanate 875 mg PO every 12 hours or 500 mg PO every 8 hours **OR**
		Clindamycin 450 mg PO every 8 hours
Gingivitis		
Acute simple gingivitis[¶Δ]	Streptococci, *Actinomyces* spp, spirochetes	Penicillin VK 500 mg every 6 to 8 hours **PLUS** metronidazole 500 mg PO every 8 hours **OR**
		Amoxicillin-clavulanate 875 mg PO every 12 hours or 500 mg PO every 8 hours **OR**
		Clindamycin 450 mg PO every 8 hours
Periodontitis		
Stage III or IV adult and juvenile periodontitis[◇]	*Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Treponema denticola*, *Prevotella intermedia*	Amoxicillin 500 mg PO every 8 hours **PLUS** metronidazole 500 mg PO every 8 hours **OR**
		Amoxicillin-clavulanate 875 mg PO every 12 hours or 500 mg PO every 8 hours

Stage I or II adult periodontitis	*Treponema denticoli*, other oral spirochetes, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*	Topical minocycline microsphere (Arestin) **OR**
		Topical doxycycline hyclate periodontal extended-release liquid (Atridox)

IV: intravenous; MU: million units; PO: by mouth.

* 25% metronidazole gel is not available in the United States and Canada.

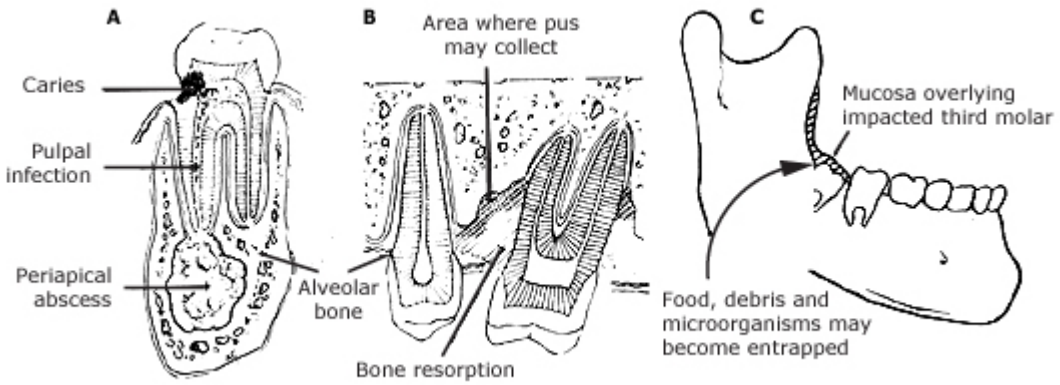
¶ We continue antibiotics until oral lesions have healed and pain has subsided, generally 5 to 7 days for gingivitis and 7 to 14 days for periodontitis. Rarely, IV antibiotics may be warranted for patient with intense gingivostomatitis who cannot tolerate oral intake. Appropriate regimens include ampicillin-sulbactam 1.5 to 3 g IV every 6 hours or clindamycin 600 mg IV every 8 hours.

Δ Acute simple gingivitis rarely requires systemic antimicrobial therapy, which are reserved for patients with rapidly advancing disease, severe pain, or an immunocompromising condition.

◇ For patients with penicillin allergies, other options include second- and third-generation oral cephalosporins with metronidazole. Refer to related UpToDate content for other alternative options.

Graphic 77718 Version 9.0

Odontogenic infections



(Panel A) Dental caries, pulpal infection, and periapical abscess.

(Panel B) Periodontal infection with destruction of supporting structures.

(Panel C) Pericoronal infection.

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Graphic 65240 Version 4.0

Antimicrobial regimens for odontogenic soft tissue infections in adults*¶

Patient population	Common causative organisms	Antimicrobial regimens[Δ]
Immunocompetent patients	*Viridans* and other streptococci, *Peptostreptococcus* spp, *Bacteroides* spp, and other oral anaerobes	Ampicillin-sulbactam 3 g IV every 6 hours **OR**
		Penicillin G 2 to 4 million units IV every 4 to 6 hours **PLUS** Metronidazole 500 mg IV or PO every 8 hours **OR**
		Cefoxitin 2 g IV every 6 hours **OR**
		Cefotetan 2 g IV every 12 hours **OR**
		Ceftriaxone 2 g IV every 24 hours **PLUS** Metronidazole 500 mg IV or PO every 8 hours **OR**
		Metronidazole 500 mg IV or PO every 8 hours[◇] **PLUS** Levofloxacin 750 mg IV or PO every 24 hours
Immunocompromised patients	*Viridans* and other streptococci, *Peptostreptococcus* spp, *Bacteroides* spp, and other oral anaerobes, facultative gram-negative bacilli (including *Pseudomonas aeruginosa*)	Piperacillin-tazobactam 4.5 g IV every 6 hours **OR**
		Cefepime 2 g IV every 12 hours **PLUS** Metronidazole 500 mg IV or PO every 8 hours **OR**
		Meropenem 1 g IV every 8 hours **OR**
		Levofloxacin 750 mg IV or PO every 24 hours[¶] **PLUS** Metronidazole 500 mg IV or PO every 8 hours

For patients with local soft tissue odontogenic infections, antimicrobial therapy is generally indicated if the patient has fever or regional lymphadenopathy, or if infection has perforated the bony cortex and

spread into surrounding soft tissue. Refer to other UpToDate content on management of patients with odontogenic deep neck space infections.

IV: intravenous; MRSA: methicillin-resistant *Staphylococcus aureus*; PO: by mouth.

* The doses recommended in this table are intended for patients with normal renal and hepatic function.

¶ Local and institutional rates of antibiotic resistance should be considered before choosing an antibiotic regimen. This is particularly important for immunocompromised patients, since there are substantial rates of fluoroquinolone resistance among *Pseudomonas aeruginosa* and other gram-negative bacteria in some regions.

Δ For patients with risk factors for MRSA infection, we typically add vancomycin (or other MRSA-active antibiotic) to the initial empiric regimen.

◇ This is an option for immunocompetent patients with penicillin allergy. Meropenem is another option, depending on the type of allergy.

Graphic 70669 Version 13.0

Contributor Disclosures

Anthony W Chow, MD, FRCPC, FACP Consultant/Advisory Boards: Qu Biologics Inc [Site-specific immune enhancers in the elderly]. All of the relevant financial relationships listed have been mitigated. **Marlene L Durand, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Milana Bogorodskaya, MD** No relevant financial relationship(s) with ineligible companies to disclose.

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