Microbiology of Acute and Chronic Osteomyelitis and Antibiotic Treatment

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KEYWORDS

• Osteomyelitis • Jaw • Mandible • Microbiology • Antibiotic

KEY POINTS

- Osteomyelitis is an inflammation of bone marrow with a tendency for progression, involving the cortical plates and often periosteal tissues, with most cases occurring after trauma to bone or bone surgery or secondary to vascular insufficiency.
- Antimicrobial therapy and surgical débridement are the primary modalities of osteomyelitis treatment, although often it is associated with a prolonged course, requiring a large commitment between patient and clinician as well as sizable health care costs.
- Despite surgical and chemotherapeutic advancements, osteomyelitis remains difficult to treat, and no universally accepted protocol for treatment exists.

Osteomyelitis is an inflammation of bone marrow with a tendency for progression, involving the cortical plates and often periosteal tissues,[1] with most cases occurring after trauma to bone or bone surgery or secondary to vascular insufficiency.[2] Osteomyelitis is also encountered more commonly in tooth-bearing areas[3] and, prior to the advent of antibiotics, osteomyelitis of the mandible was not an uncommon occurrence.[1] With the use of antibiotics, however, osteomyelitis has now become a rare disease.[1] Recently, however, antibiotics have become less effective, presenting therapeutic challenges to surgeons that can result in the loss of teeth or bone.[1] Antimicrobial therapy and surgical débridement are the primary modalities of osteomyelitis treatment, although often it is associated with a prolonged course, requiring a large commitment between patient and clinician as well as sizable health care costs.[2] Despite surgical and chemotherapeutic advancements, osteomyelitis remains difficult to treat, and no universally accepted protocol for treatment exists.[2]

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PATHOGENESIS

Osteomyelitis of the mandible is much more common than of the maxilla due to its dense and poorly vascularized cortical plates along with the vasculature originating from the inferior alveolar neurovascular bundle.[1] Maxillary bone, meanwhile, is much less dense and receives its vasculature from multiple vessels and thus more resistant to developing an osteomyelitis.[1]

Host defenses also play a role in the progression of osteomyelitis. Osteomyelitis has been associated with systemic diseases, such as diabetes mellitus, autoimmune disorders, malignancy, malnutrition, and AIDS.[1] Additional factors that predispose patients to osteomyelitis of the jaws include noncompliant patients who are refractory to health care delivery, patient age, nutritional status, immunosuppression, microvascular disease, and inaccessibility to health care.[4] Medications with roles in osteomyelitis include steroids, chemotherapeutic agents, and bisphosphonates.[1] Local factors that compromise vascularity, such as osteoporosis, bone pathology, and radiation therapy, also increase the possible risk for bony infection.[1]

Osteomyelitis typically occurs as a result of spread of an odontogenic infection or as a result of trauma.[1] The presence of teeth provides a direct pathway to the bone by pulpal or periodontal disease.[3] A hematogenous origin is rare and primarily occurs in young children.[1] The adult process begins with bacteria spreading to the jaw bones, either through extraction of teeth, root canal therapy, or fracture of the jaw bones, resulting in a bacteria-induced inflammatory process.[1] Although commonly self-limiting, there is a potential for progression to a pathologic process.[1] Inflammation leads to hyperemia, increased blood flow, and leukocytes to the affected area.[1] Pus is formed when bacteria and cellular debris cannot be eliminated by the body's natural defense mechanisms.[1] When the pus and inflammatory response occur in the bone marrow, elevated intramedullary pressure is created, which further decreases blood flow to jaw bones.[1] Pus travels via the haversian system and Volkmann canals to spread throughout the medullary and cortical bones, and, as pus perforates the cortical bone, it collects under the periosteum, further compromising the periosteal blood supply.[1] After some time, ultimately the purulence exits the soft tissues via intraoral or extraoral fistulas.[1]

CLASSIFICATION

Many classification systems for osteomyelitis have been presented in the past, such as suppurative or nonsuppurative, hematogenous or secondary to a contiguous focus of infection, and acute or chronic, with the latter becoming the predominant classification system.[1] The differentiation of acute versus chronic is based on the presence of disease for 1 month.[1] Another classification system is based on Zurich, in which chronic osteomyelitis can be divided into suppurative chronic osteomyelitis, osteonecrosis of the jaw, and bisphosphonate-related osteonecrosis of the jaw (BRONJ).[5] Chronic nonsuppurative osteomyelitis of the mandible is also referred to in the literature as primary chronic osteomyelitis (PCO), diffuse sclerosing osteomyelitis (DSO), Garré osteomyelitis, and juvenile mandibular chronic osteomyelitis (JMCO).[6]

Osteomyelitis may be classified into acute and chronic forms.[7] Acute osteomyelitis can be further subdivided into suppurative and nonsuppurative forms as well as progressive or hematogenous forms.[7] Chronic osteomyelitis may be classified by the causative agent or as suppurative or nonsuppurative forms or sclerosing with subclassifications of diffuse or focal disease.[7]

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