Alveolar Osteitis and Osteomyelitis of the Jaws

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KEYWORDS

• Osteomyelitis • Delayed healing • Marrow infection
• *Actinomyces* • Dry socket • Immune compromised

OVERVIEW

Postoperative bone healing after oral surgical procedures occurs uneventfully in most cases because of exceptional vascularity of head and neck structures when compared with other anatomic sites. However, in certain patients, the normal process of osseous healing can be delayed and, in some cases, often because of multiple coexisting factors, the sites can become infected, with extension of the infection into medullary bone. This process is termed osteomyelitis. The exact definition of osteomyelitis is inflammation of the osseous medulla. The term osteitis reflects a more superficial inflammation of the cortex of the bone. Most often, infections of the medulla also involve the cortex by the pathways of haversian systems and often affect the overlying periosteum. Hence the term osteomyelitis is more commonly used to describe alveolar and basal bone infections. The infectious process in the marrow space of bones has been well documented in early man. The oldest known case of mandibular osteomyelitis dates back to the Pleistocene epoch about 1.6 million years ago and fossil findings in the jaw of a 12-year-old *Homo erectus* skeleton found in Kenya. Since the discovery of bacteria and the advancement in antimicrobial therapy, there has been a significant decrease in the incidence with improved outcomes in the care of these infectious conditions.[1,2] Over the years, multiple classification schemes have been proposed,[1–6] but most current literature on the topic suggest wisely using a simplified classification system based on clinical course time lines and appearance of the disease.[3] This simplified classification scheme is used in discussing the pathogenesis, diagnosis, and therapy for these conditions. Imaging techniques, including the new positron emission tomography/computed tomography (PET/CT) fusion techniques, are addressed. Pathogenesis, microbiology, and surgical and medical therapies are outlined. This article specifically addresses osteomyelitis cases related to patients with no documented history of radiation or bisphosphonate exposure and in whom the principal factor in the development of the condition is infection by pyogenic microorganisms.[3] The other subsets of infectious osseous pathosis are discussed by Leon A. Assael; and Sinha and colleagues specifically elsewhere in this issue.

DENTOALVEOLAR SURGICAL WOUND HEALING

Normal wound healing is aimed at restoring the site to the preinjury state. It is often a sequential process that starts at the time of injury and is based on cellular level messing that induces homeostatic, inflammatory, angiogenic, inductive, and mitogenic changes in local cell populations as well as circulating pluripotent cell recruitment and differentiation. Site regeneration involves both metabolic and catabolic changes, which are

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influenced by local and host factors including vascularity and oxygen supply. Hypoxia decreases normal antimicrobial activity of granulocytes by as much as 50%.[7] Bacterial virulence is also a significant factor in the development of early or late wound infections.[2,3,8] Finally, host vascular and immune factors and current immune status have been shown to affect the incidence of head and neck delayed healing and wound infections.[1,8–10]

ALVEOLAR OSTEITIS (DRY SOCKET)

Incidence

One of the best known and most referred to complications of dental extraction in the general public is alveolar osteitis (AO) better known as the dry socket. It is a common postoperative complication that occurs in less than 5% of patients undergoing tooth extraction.[11–17] The research, despite high incidence of this condition, is poorly structured with rates of incidence ranging broadly from 0.5% to 37.5%.[18,19] Third molar surgery carries the highest incidence of AO occurrence. Maxillary AO is very rare and is often misdiagnosed as normal postoperative discomfort. It is widely thought that this misdiagnosis is due to higher maxillary bone vascularity because of more circumferential sources of supply over the central endosseuous mandibular pattern. The best description of the condition is premature fibrinolysis of the clot, which may result in local and radiating pain, halitosis, and abdominal discomfort.[20,21]

Cause of AO

Despite the long-term awareness of the condition, the cause of AO is still not fully understood, but it has been widely noted that premature fibrinolytic breakdown of the initial platelet clot in the extraction site exposes the underlying and tooth socket bone (**Fig. 1**). Breakdown of the clot occurs as a result of plasminogen pathway activation whereby an activator substance is triggered by either physiologic or nonphysiologic mediators (including bacterial enzymes). Specific factors are debated and poorly understood, but all of them have some promoter effect on clot lysis, which leads to fibrinolysis.[22,23] It has been postulated that bacteria is limited to the surface of the bone and does not produce a true medullary bone infection.[3,6,22] Hence at present, AO is not categorized as a true infectious process of the bone.

Symptoms

The cardinal symptom of AO is pain that originates in the jaw and radiates either from the ear to temple and/or runs in the lower jaw along the trigeminal nerve distribution affecting all distal teeth and bone.[14,16,17] Other reported symptoms include low-grade fever,[20,21] halitosis,[21] exposed bone, and regional lymphadenopathy.[20,21,24]

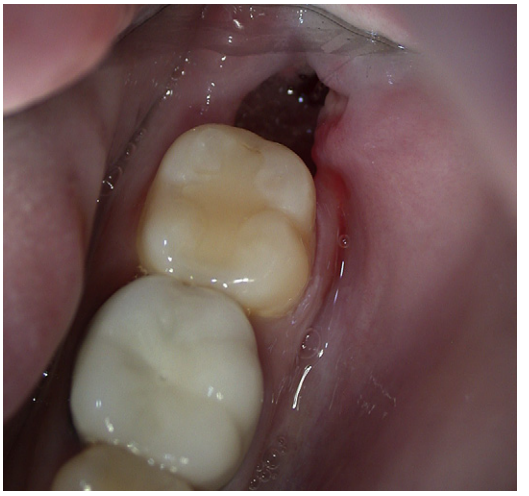

Fig. 1. Typical clinical presentation of extraction site devoid of blood clot.

Onset

Most patients diagnosed with AO have reported onset of symptoms after 3 to 5 days after the surgical procedure.[20,24,25] However, continued localized painful symptoms from the day of surgery are also possible. AO-like symptoms that become evident after 1 week from the surgery are not consistent with AO[11] time lines and therefore should be considered to be stemming from another process, which may include either food debris impaction or acute osteomyelitis.

Risk Cofactors in AO

The increased risk factors for development of AO have been well identified and documented in many studies and include preexisting infection, poor oral hygiene, partial impaction of tooth, periodontal disease, lack of operator experience, oral contraceptive use, tobacco use, and increased age.[16,24–27] Other factors are flap design, vasoconstrictor use, aggressiveness of site manipulation, saliva exposure, the patient's age, and the level of systemic health of patient, although their role has not been clearly demonstrated.[14,16,28]

Treatment of AO

No significant changes in the management of the condition have occurred in the past few decades. The main focus of the current therapeutic approach for AO is to maintain patient comfort