Alveolar Osteitis and Osteomyelitis of the Jaws

Peter A. Krakowiak, DMD, FRCD(C)[a,b,c,*]

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

The author has nothing to disclose.
[a] Oral and Maxillofacial Surgery, Herman Ostrow School of Dentistry, University of Southern California, 925 West 34th Street, Room Den 146, Los Angeles, CA 90089, USA
[b] Private Practice, 265 San Jacinto River Road, Suite 101, Lake Elsinore, CA 92587, USA
[c] Private Practice, 5256 South Mission Road Suite, Bonsall, CA 92003, USA
* Lakeshore Oral and Maxillofacial Surgery, 265 San Jacinto River Road, Suite 101 Lake Elsinore, CA 92530.
E-mail address: pkoms@sbcglobal.net

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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]

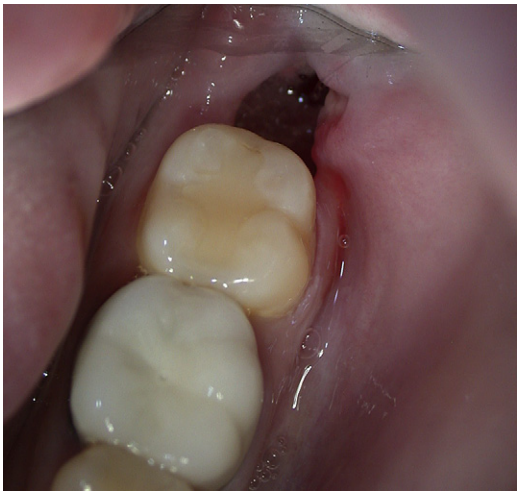

Fig. 1. Typical clinical presentation of extraction site devoid of blood clot.

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

for the initial healing period after surgical intervention until the normal healing process can occur. Therapy includes the application of topical analgesics and antimicrobial agents.[28]

The medications are usually applied on a carrier vehicle such as iodoform gauze, collagen, or gelatin sponge (**Fig. 2**). Many combinations exist for the treatment medication formulation, but the majority includes substances that contain eugenol with other additives such as benzocaine, guaiacol, balsam of Peru, chlorobutanol and iodoform, and others. The site is usually irrigated to remove any foreign debris and evaluated for any loose fragments of bone. Local anesthesia may be used for patient comfort but is usually not necessary. Limited manipulation of tissue is recommended. The dressing on the carrier is then applied into the site and packed to rest at the level of or slightly below the crest of the socket walls (**Fig. 3**). The drawback to placement of these carrier-based medications is that it retards some of the healing process because all dressings are foreign bodies.[28] The patient usually requires multiple dressing changes, and the exact number of changes is always dictated by the patient's relative comfort.

Premade dressings and carrier medications, such as Alvogyl (Septodont Inc Wilmington, DE, USA), that are marketed as place and dissolve dressings have been shown to produce delayed healing.[29,30] Moreover, any retained dressing can become a nidus for late infection and should be removed after 2 to 3 days in place and either discarded or replaced with a new dressing. Radiolabeled dressings, such as Dressol-X (Rainbow Specialty & Health Products Inc, Niagara Falls, NY, USA), are superior to regular iodoform dressings because they allow for easy identification if retained and overgrown by tissues.

Fig. 2. Topical medication being applied to nonresorbable iodoform gauze.

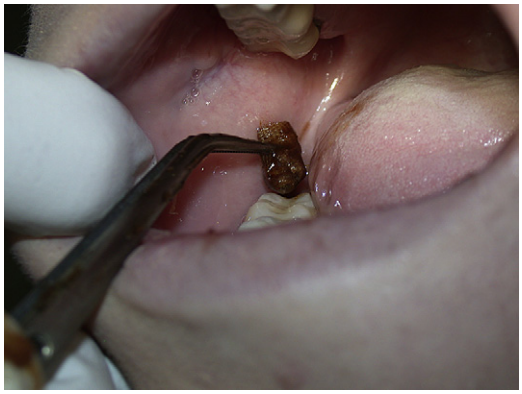

Fig. 3. Iodoform dressing being placed into third molar site using pick ups.

Patients can be prescribed additional analgesics and placed on gentle saline or 0.12% chlorhexidine rinses.[31] Moist warm heat compresses are helpful in increasing circulation and comfort in the area. Multiple agents including parahydroxybenzoic acid, polylactic acid, corticosteroids, 9-aminocrinide, and tranexamic acid have been used but have not been scientifically shown to be useful in the treatment or prevention of AO.[14,22]

AO SYNOPSIS

Although the incidence of AO is very high, especially in third molar extractions, it is a self-limiting concern, which benefits most from a few 1- to 2-day local topical treatments with eugenol-based compounds on a nonresorbable carrier. Patients who have identifiable risk factors should optimized medical therapy and appropriately counseled regarding their risks. At the same time, the practitioners should be able to differentiate between normal postoperative pain, which tends to improve after the initial 24 to 48 hours, and the increasing symptoms of AO, which become more pronounced after the 72-hour mark. In all cases, frank reassurance and prompt management of this common condition is paramount to the practitioner's ability to provide the necessary care for the patient with AO.

OSTEOMYELITIS OF THE JAW

Most osteomyelitis in long bones arise from either local extension or hematogenous spread, but in maxillofacial skeleton, the spread is mostly by local extension from skin, oral cavity, or paranasal sinuses.[1,32,33] It is a relatively uncommon complication in patients undergoing extraction with normal immune function status because of the perceived excellent vascularity in this region of the body.[1,3,6,33,34] Highest rates of osteomyelitis

are noted in patients with vascular insufficiency and immune dysfunction as well as in those with bone metabolic abnormalities. These (metabolic bone) conditions include diabetes, fibrous dysplasia, florid osseous dysplasia, osteopetrosis, Paget disease, sickle cell anemia, osseous malignancies leukemia, agranulocytosis, systemic steroids, intravenous drug use, renal and hepatic failure, and human immunodeficiency virus infection.[1,3,6] Patients who take immunosuppressive agents, are malnourished, and consume significant amounts of alcohol[3,9] are also at a higher risk. Finally, patients who have received or are receiving osteochemotherapy with bisphosphonates and those who have undergone radiation therapy are a separate and highly risk-prone segment of patients who can develop maxillomandibular osteomyelitis. These 2 specific conditions are discussed elsewhere in this issue but need to be included in the list of contributing factors. However, 17% of patients who develop osteomyelitis have no identifiable underlying predisposing factors.[35]

CLASSIFICATION SCHEMES

The classification schemes for evaluating and treating AO have been based on clinical and radiographic findings, cause, pathogenesis, and associated anatomy. However, there is no 1 set standard. The most simplistic way to consider the condition is based on an arbitrary time line of 1 month and is considered to be either acute or chronic condition. The modifier for the picture can also include a suppurative attribute. Because the suppurative form is more aggressive and often it is hard to differentiate the chronic nonsuppurative entities from various fibro-osseous ones including clinically overlapping diffuse sclerosing osteomyelitis (DSO) or periostistis ossificans (PO) or Garré osteomyelitis lesions, the more suppurative variant is discussed in more depth in this article. It is also more related to acute complications of surgical therapy, which is the focus of this article. The chronic variant of osteomyelitis is also associated with synovitis acne pustulosis hyperostosis osteitis (SAPHO) syndrome, which is characteristic for synovitis, acne, pustulosis, hyperostosis, and osteitis and may be linked to HLA-B13 and HLA-B27–related autoimmune conditions.[32]

ACUTE OSTEOMYELITIS

Within this category, the patient may experience quite a range of symptoms and varied presentations. Most cases have significant pain in the jawbones, swelling, trismus, purulent drainage, and febrile episodes with potential hypoesthesias in more than 50% of the cases.[1,3,6] Additional clinical signs include lymphadenopathy, fistulous tracts, exposed bone, and sequestra formations (**Figs. 4** and **5**). Patients may report malaise and fatigue. Normal to slightly elevated leukocyte count is noted in most cases, but about one-third of the patients may have significantly increased leukocyte counts of more than 15,000.[6] Additional laboratory values of interest are erythrocyte sedimentation rate and C-reactive protein values, which may also be elevated. C-reactive protein values can be used to follow the resolution of infection and progress of therapy.[6,32,36] Patients who do not show significant symptoms during the acute phase and do not receive adequate therapy are considered to have subacute condition and most often progress to the chronic phase of the disease.[1,3,6,8]

CHRONIC OSTEOMYELITIS (SUPPURATIVE)

Chronic osteomyelitis occurs in patients in whom either a host resistance or a therapeutic failure occurs allowing the infectious process to continue past the 30-day mark. The symptoms and clinical presentation may be less severe than those of an acute form, but most patients still present with jaw pain, swelling, and suppuration.[6] Usually, the bone undergoes sequestra formation and demonstrates significant changes radiographically. With

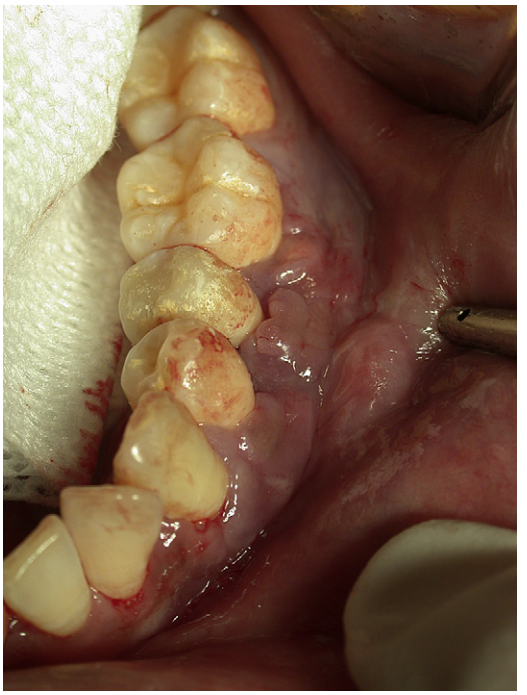

Fig. 4. Gingival edema and mobile dentition in the area of affected by osteomyelitis.

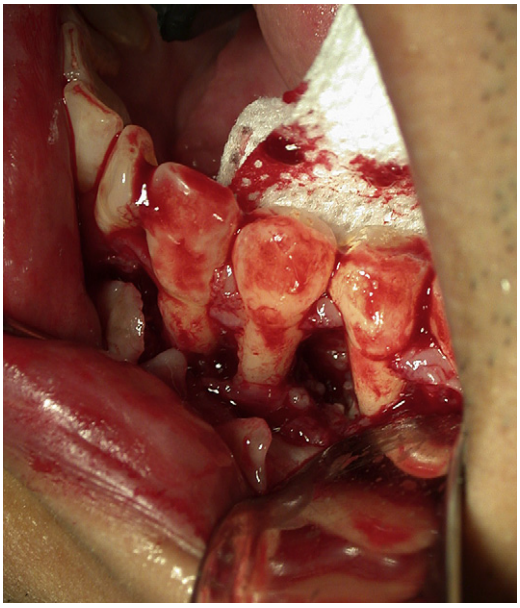

Fig. 5. After elevation of a full thickness flap extensive bone loss and necrosis is evident.

a severe degree of progression, there is a potential for pathologic fractures and formation of extraoral fistulae. The lower-grade processes tend to progress into the sclerosis variants on either DSO (medullary) or PO discussed as nonsuppurative variants.

CHRONIC OSTEOMYELITIS (NONSUPPURATIVE)

This form of medullary marrow infection is thought to be caused by overgrowth of *Actinomyces* and *Eikenella corrodens*.[3,32] It usually has milder symptoms and may be free of other clinical signs and symptoms with the exception of radiographic findings. In most cases, the disease is diagnosed several years into the disease process.[32] The lesions are often mistaken for fibro-osseous lesions and are difficult to definitively diagnose without biopsy and cultures.

OTHER RELATED CONDITIONS

PO, also known as Garré osteomyelitis, is named after Carl Garré, although he did not describe this specific condition in any of his late eighteenth century works. PO is characterized by deposition of immature bone layers over the existing cortical contour. Onion skin radiographic appearance of this expansile proliferative condition is classical but not pathognomonic because malignancy of bone may have similar appearance. No symptoms are evident in these cases.[6,32]

NEURALGIA-INDUCING CAVITATIONAL OSTEONECROSIS

Neuralgia-inducing cavitational osteonecrosis is a condition described by Dr Boquot who assigned osseous osteomyelitis–like changes to patients with atypical facial pain and neuralgia.[32] Patients were then subjected to experimental protocols including curettage and bone graft protocols that were aimed at reducing symptoms. Since gaining some attention in the early 1990s, the condition has not been more scientifically defined in the peer-reviewed literature, and the practitioners of this methodology have been involved in extended legal battles, disciplinary actions, and class action suits. Limited literature supports the existence of this condition, and most is the work of its inventors and proponents. There are many who have discredited the existence and validity of the treatments proposed for this obscure and controversial pathosis.[32,37,38]

DIAGNOSTIC IMAGING

The increasing availability of 3-dimensional imaging, magnetic resonance imaging (MRI), scintigraphy, and, now, PET/CT imaging has made it much easier to precisely delineate the extent of the disease process in a timely manner. The newest imaging modalities, such as scintigraphy and PET scan, are able to highlight biological as well as anatomic activity and may be coupled with navigational approaches to virtual surgical therapy and interventions.[6,39]

Conventional Radiography

Albeit the standard for many decades, the role of this modality is limited in detection of and therapy for osteomyelitis because it only shows changes after extensive bone abnormality has been present for prolonged periods.[40] However, it is readily available and exposes patients to minimal radiation. Panoramic projection is most useful in most maxillofacial cases (**Fig. 6**).

CT

The addition of cone beam computerized tomography, which is highly useful for imaging hard tissues of the head and neck in multiplanar slices, is ideal for visualizing decortications and periosteal changes.[41] Soft tissue changes can also be visualized in medical-grade CT scans by adding contrast medium (**Fig. 7**). The changes of early osteomyelitis are more clearly delineated and easier to interpret with CT images than with conventional radiography.[42] Its reconstruction capabilities can

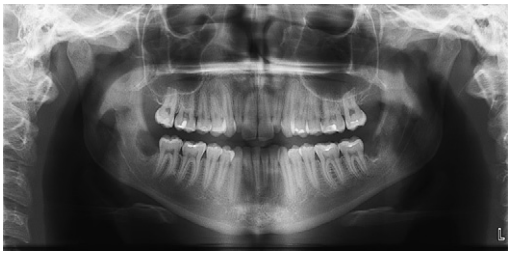

Fig. 6. Panoramic film of an acute osteomyelitis of number 17 site one month post extraction. Limited diagnostic changes are evident on this projection due to relatively long period required for lesion to affect bone density.

also be helpful in stereolythic model manufacturing and surgical treatment planning (**Figs. 8** and **9**).

MRI

Use of gadolinium as a contrast agent can show early osteomyelitis changes in tissue by highlighting nonspecific disturbances in tissue-blood interfaces, which are common in infection, inflammation, trauma, or tumors. The changes are most often noted in the soft tissuesand can also be noticed in the medullary portion of the affected bone.[43,44] In the T1-weighted images, these changes show low signal, whereas T2-weighted images show bright signal at the sites of inflammation because of increased water content. MRIs have a poor ability to analyze the condition and involvement of mandibular cortex particularly in early acute osteomyelitis.[6,43]

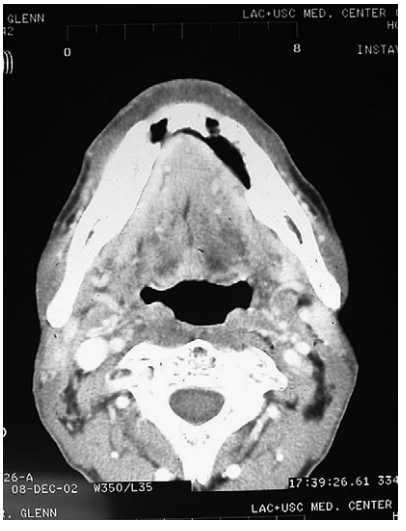

Fig. 7. CT with contrast showing both soft and hard tissue changes associated with chronic mandibular osteomyelitis.

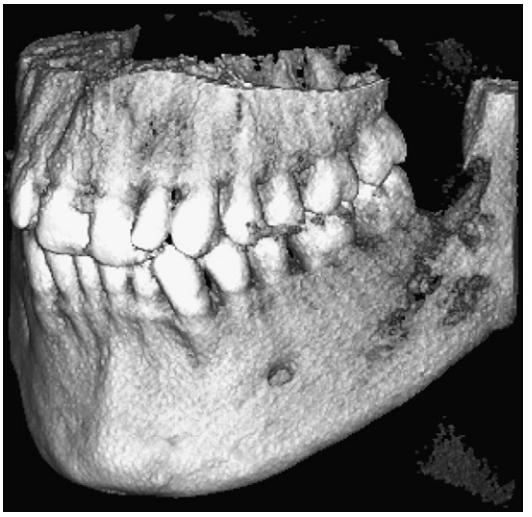

Fig. 8. 3D reconstruction of patient in **Fig. 6**, taken the same day but showing much greater osseous changes compared to the standard two dimensional modality.

Scintigraphy

The radioactive substances used to identify altered bone physiology are technetium 99m–labeled methylene diphosphonate, gallium 67, and indium 111. The most common scintigraphic agent is technetium 99m (**Fig. 10**), which is used to delineate increased bone turnover, and it is often coupled with the gallium 67 (**Fig. 11**) to distinguish the osteomyelitis lesions from tumor and trauma because gallium is sensitive to inflammatory

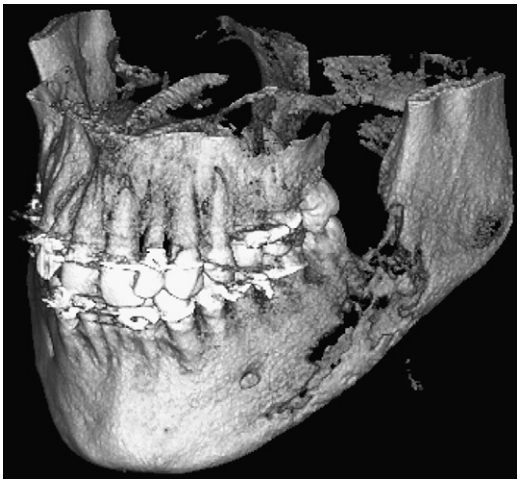

Fig. 9. Same patient as **Figs. 6** and **8** after additional two weeks of oral antibiotics therapy alone is showing the progression of the bone destruction and decalcification.

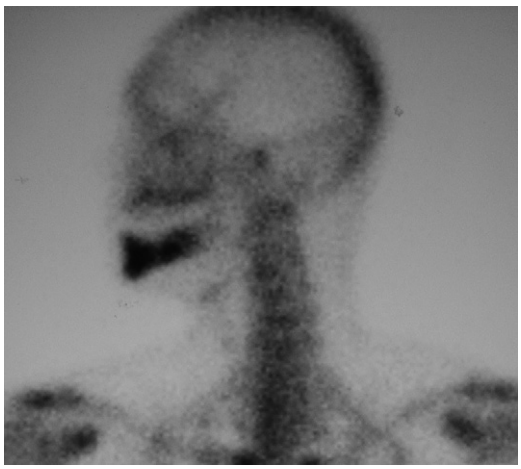

Fig. 10. Technetium scan showing uptake in the mandibular symphysis and body of an osteomyelitis patient.

changes.[6,40] The combined techniques have 98% sensitivity and can show changes as early as 3 days from the onset of infection.[1,40] Coupling of indium 111 is important when determining the activity of the lesion and the potential end point of therapy.[40,43] The downside of this technique is the exposure of the patient to a radiopharmaceutical. Therefore its use should be limited to cases in which a clear diagnostic benefit is expected.

PET/CT

The application of PET scan using fludeoxyglucose F 18 has shown promise in the identification of osteomyelitis in the jaws especially when applied with traditional CT. The 2 scanning modalities fuse together anatomic findings and a metabolic state finding in a real-time frame. Unlike other existing study modalities, a 3-dimensional image is obtained with high sensitivity and specificity.[45] Individual PET scans have a much higher rate of false-negative and false-positive results.[39] The linking of anatomic abnormalities with metabolic alterations is the key to pinpoint accuracy of the hybrid diagnostic modality in mandibular osteomyelitis.[46,47] The limited preliminary research data available show that the combined techniques have higher rates of specificity and sensitivity of traditional scintigraphy and leukocyte scintigraphy.[39,45] Because this is a relatively novel diagnostic approach, more research is needed to fully delineate all potential applications of this diagnostic and potentially surgically navigational technology. It is also hoped that as the PET scanning technology becomes more widely available and more economical, the access to this imaging will also improve for all practitioners in the community to routinely diagnose suspected osteomyelitis and measure real-time progress of the therapy.

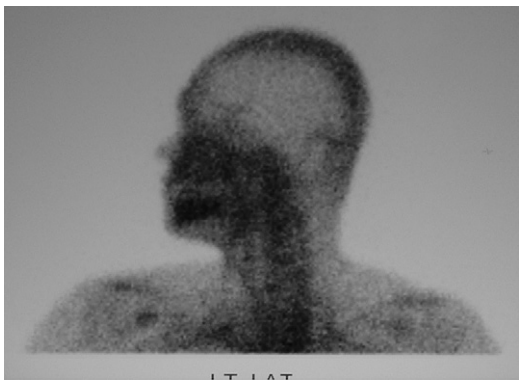

Fig. 11. Gallium scan showing similar mandibular involvement.

INCIDENCE

Acute and chronic osteomyelitis are much more relevant in the mandible than in the maxilla. The literature has noted that the overall incidence of mandibular pyogenic osteomyelitis is up to 3 to 19 times greater than maxillary cases.[2,35,48–51] Historically, most cases in the maxilla were related to dental infections, orthognathic procedures, and malignancies.[1–3,52] The few documented infections were mostly associated with the dental support structures. However, with increase in bisphosphonate-related osteonecrosis cases, the maxillary skeleton involvement may become more prevalent.[52] In the mandible, the most common sites of osteomyelitis are the body, followed by the symphysis (**Figs. 12–14**), angle, ascending ramus, and condyle,[51] Both sexes are affected almost equally based on overall data from demographic studies.[6,32,35,50–52] Chronic osteomyelitis cases are more frequent after the second decade of life peaking, and this may correlate better with changes of the immune and vascular health of the adult and aging patient.[50,52] A rare infantile osteomyelitis variety can occur in newborn and infants and can involve the maxilla as well as the mandible. It is thought to have more of a hematogenous origin as a pathway for seeding of the bacterial infection in infants.[1,9]

CAUSE, PATHOGENESIS, AND MICROBIOLOGY

In most patients who develop osteomyelitis of the jaws, there is local spread of microflora into some wound connected to the medullary space. The

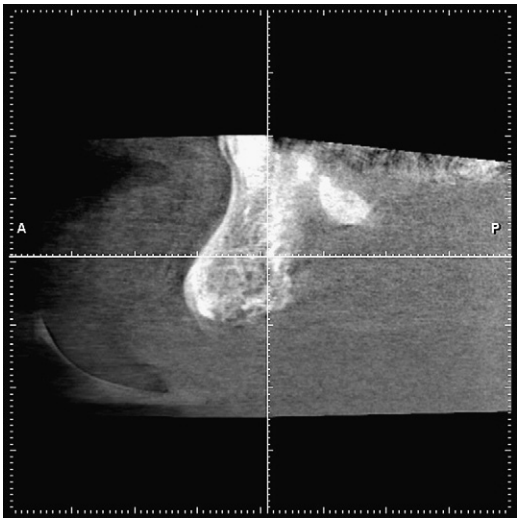

Fig. 12. Sagital cone beam CT showing mandibular symphysis osteomyelitis.

normal mixed microflora from oral and panfacial sinuses as well as skin in trauma cases has been implicated in the development of the disease. The bacteria associated with infected dentition, such as periodontal pathogens including *Staphylococcus aureus*, *Staphylococcus epidermidis*, Actinomyces, Prevotella species,[6,9,52] and Eikenella species, have been noted to be present in most chronic cases.[3,9,52] Candida infections were also noted in some of the cases of osteomyelitis.[52,53] The culturing of specific microorganism is very complex and often difficult to obtain in all clinical settings. Often, bone needs to be submitted in anaerobic medium or blood culture vials to prevent loss of anaerobic milieu. Gram staining and staining

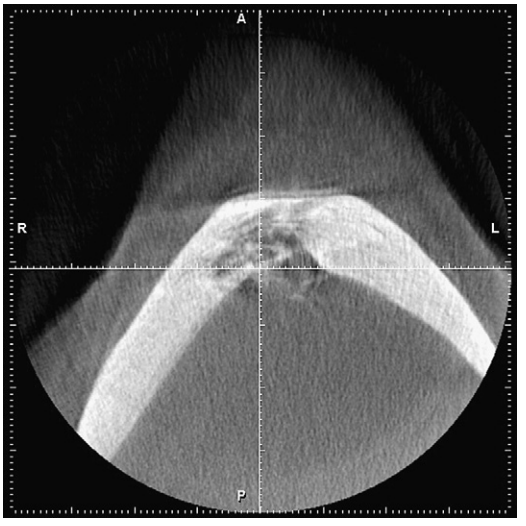

Fig. 13. Axial view of the same patient.

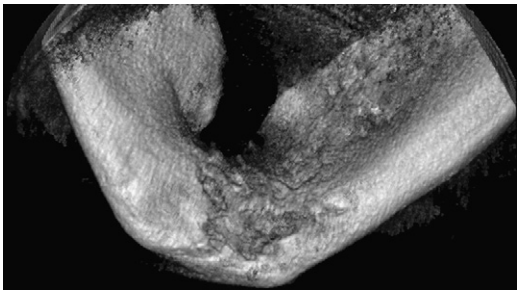

Fig. 14. 3D reconstruction of mandibular defect from worm's view.

with hematoxylin-eosin, van Gieson, Giemsa, and periodic acid–Schiff are the standard techniques helpful with early pathogen identification. The final results of culture and sensitivity test should guide the antimicrobial therapy after initial treatment with empirically derived regimens.[54–56]

Once seeded, the infections are thought to spread via the medullary marrow space and compromise the blood supply. This process is particularly damaging in the mandible, which has limited peripheral contributory supply and relies mainly on the inferior alveolar artery for blood flow. The affected bone is destroyed at a rapid rate in most suppurative cases with formation of sequestra and involucrum within cancellous and cortical portions.

Biopsied bone specimens from acute osteomyelitis have histologic findings of marrow spaces lined with neutrophilic granulocytes, necrotic bone, and inflammatory exudates (**Fig. 15**).[53] The increased pressure leads to further compromised vascularity and osteocyte necrosis. The sequestra become

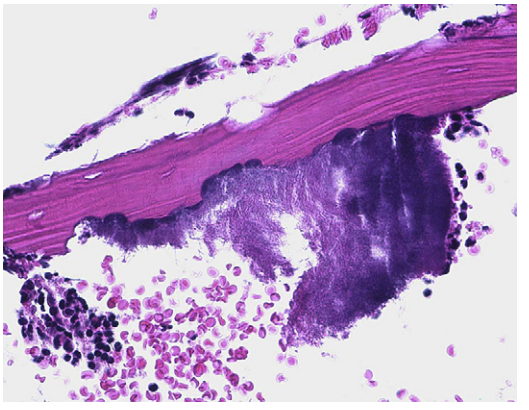

Fig. 15. H&E high power view of specimen from osteomyelitis showing sequestra of necrotic bone, inflammation and bleeding with overgrowth suggestive of actinomyces specie. (*Courtesy of* Paymon Parish Sedghizadeh, DDS, Los Angeles, CA.)

colonized with biofilm-forming microorganisms, which in turn leads to continued suppuration and chronicity of the process.

In chronic forms of osteomyelitis, the inflammatory infiltrate is composed of plasma cells, lymphocytes, and macrophages. Reactive bone formation is evident with irregular reversal lines seen similar to those of Paget disease.[53,55]

TREATMENT

Combined antimicrobial and surgical therapy is required in the management of all suppurative and chronic cases, with the exception of the infantile variety that may respond to intravenous medication alone.[1,3,32,54,56] The most important step in the process is a timely diagnosis before significant progression of the disease occurs. Early management reduces the morbidity and extent of surgical therapy required.

Other conditions including malignancies and metabolic disease should be excluded. The therapy is aimed at reducing bacterial challenge to the host's system. Surgical therapy physically reduces bacterial count but must be coupled with correction of any underlying medical conditions and well-targeted antimicrobial regimen. The addition of hyperbaric oxygen (HBO) therapy is also considered an important adjunct in swinging the pendulum in favor of host defensive and homeostatic systems.[57]

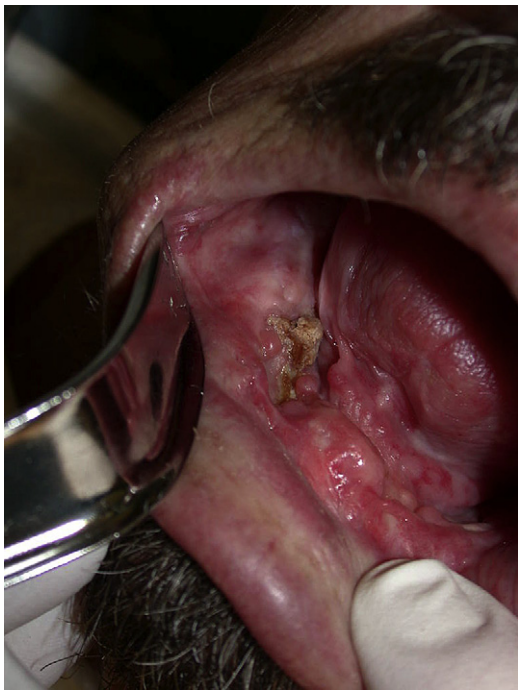

Fig. 16. Superficially located sequestra.

SURGICAL CORRECTION

The removal of necrotic bacteria–containing debris is accomplished through 3 distinct modalities. Sequestrectomy removes the localized free-standing areas of necrosis in the central area of infection (**Fig. 16**). Saucerization is more aggressive with the removal of adjacent bony cortices followed by exposure of the deeper layers of the medullary bone to allow for placement of packing materials and healing of soft tissues by secondary intention (**Fig. 17**). This approach can be useful in the early stages of disease and diseases of limited extent. It also allows for decompression of medullary cavity without significant removal of supporting structures of the mandible. The drawback is that this approach may be more likely to contaminate the specimen for culture and sensitivity testing because it is an all-transoral technique.[58]

Decortication is a more extensive approach, with intraoral and extraoral approaches possible, involving large broad bone removal of cortical bone, and it often requires lateralization of neurovascular bundle and rigid fixation to reduce pathologic fractures (**Fig. 18**). Primary closure is attempted over the site with mucoperiosteal covering of the newly exposed medullary space. This approach is advocated for larger lesions in advance acute or chronic osteomyelitis.[56]

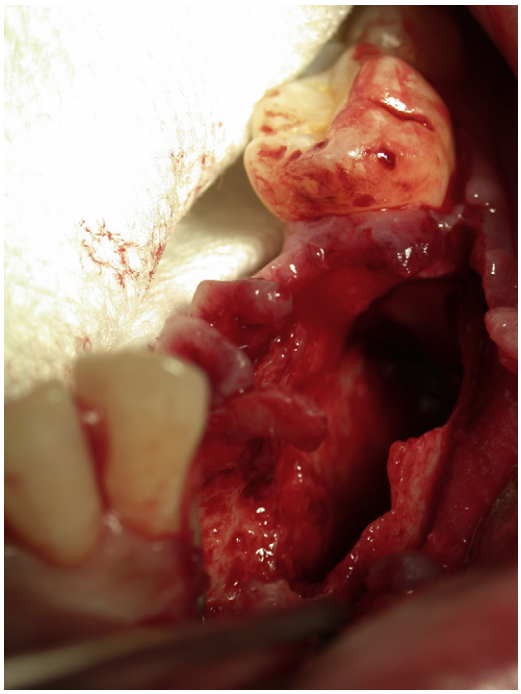

Fig. 17. Saucerization of mandibular defect maintains ridge continuity on lingual and buccal aspects of the body.

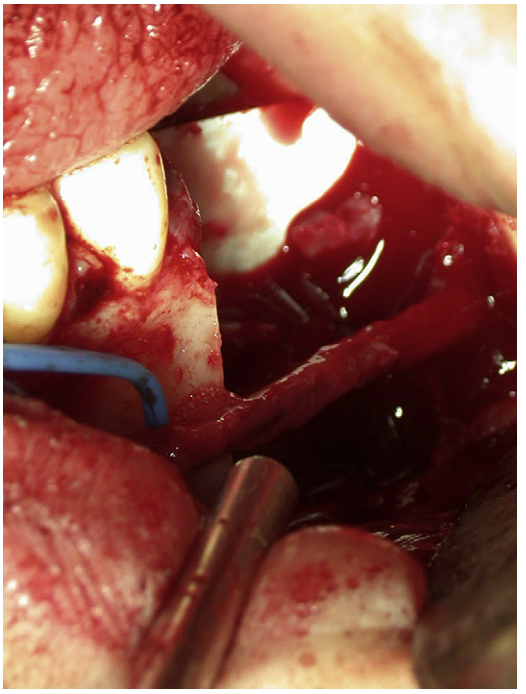

Fig. 18. Nerve laterization to allow for gross decortication of the defect.

In extensive defects or pathologic fractures, the site requires resection and subsequent reconstruction (**Fig. 19**). Early simultaneous resection and reconstruction has been performed and used by some,[54,59,60] but a staged approach may be more predictable.[3,56]

In all these approaches, clinical judgment directs the surgical intervention and is best based on imaging modalities such as CT, scintigraphy, and the new CT/PET fusion scan. The surgical end point of terminating resections at clinically viable bone stock with normal bone density and vascularity can be used when radiographic studies are not available or adequate (**Fig. 20**).

The wounds can be treated with acrylic beads laced with gentamicin as well as copious pulsed irrigation techniques using antimicrobial irrigants analogous to orthopedic long bone irrigation protocols.[48,56,61]

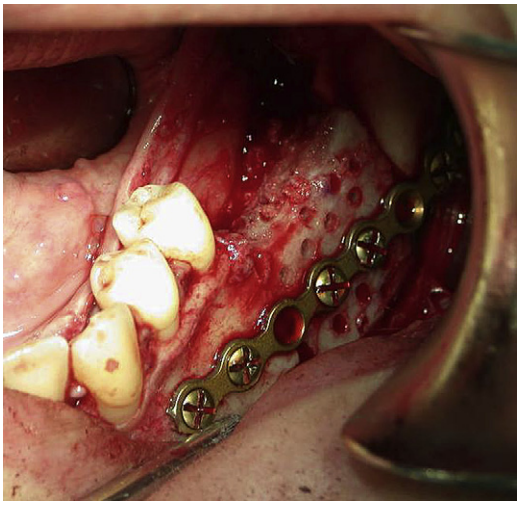

Fig. 19. Block resection and immediate iliac crest reconstruction of the body defect.

ANTIMICROBIAL THERAPY

As stated earlier, the initial antimicrobial therapy for osteomyelitis is based on using empirical coverage for the common causative pathogens. With treatment failures, a closer look at culture and sensitivity micropathology data when it becomes available is important. The first-line agents for this are clindamycin or amoxicillin/clavulanic acid combination regimens for minimum of 6 weeks.[62,63] Alternatively, for methicillin-susceptible *S aureus* infections, combination of flucloxacillin, ciprofloxacin, or levofloxacin with rifampin is advocated. For methicillin-resistant *S aureus*, combined vancomycin and rifampin therapy followed by oral ciprofloxacin or levofloxacin is considered appropriate. Peripherally inserted central catheter line should be placed and utilized during the IV therapy with frequent attention paid to condition and cleanliness of the access site (**Fig. 21**).

In cases of chronic osteomyelitis, similar regimens can be used, but intravenous medications can be limited to the initial 2 weeks of therapy and can then be followed by oral medication.[63] It is always a good idea to involve an infectious disease expert in coordinating the pharmacologic agent used in these more complex cases, especially in cases with past antibiotic therapeutic failures or when patient's multiple agent allergies are of concern.[3,54,56]

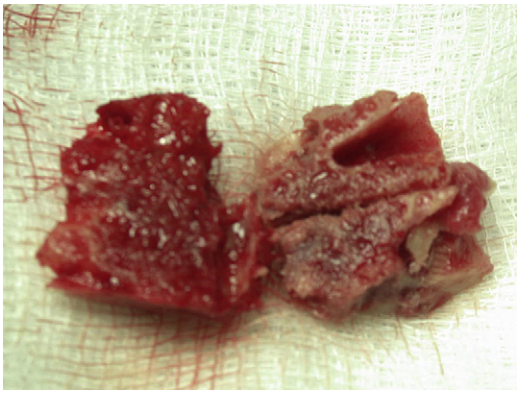

Fig. 20. Viable and necrotic bone from the osteomyelitic defect of the mandible showing differences in vascularity.

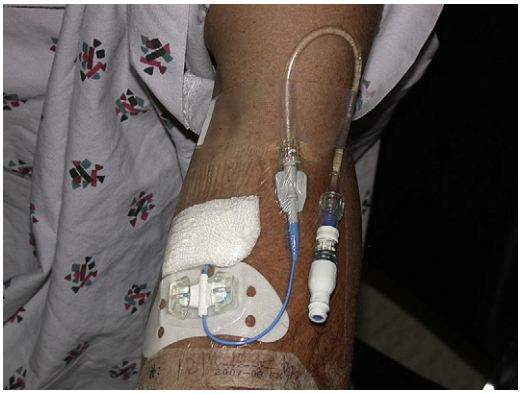

Fig. 21. Patient with a PICC line secured for IV antibiotic delivery.

HBO

An adjunct that has become available in the past 2 decades for therapy of osteomyelitis of the jaws is HBO therapy, which counters local hypoxia effects of medullary infections. For strictly anaerobic infections, the benefit of HBO is thought to be the greatest. However, no large-spectrum human prospective data studies support its use for early or acute osteomyelitis. Further, data must be gathered to demonstrate the value of this therapy in non–radiation-related osteomyelitis. In the meantime, it may be a modality to consider in refractory or host system incompetence cases.[3,54]

FOLLOW-UP AND CONTINUED CARE

The important concept in the management of infectious osteomyelitis is successful assessment of clinical interventions and the need for additional treatment in cases of failure or poor progress. The clinical picture is most important because it gives a real-time view of the process. Laboratory and radiographic values can be compared with baseline. At least a 2-year follow-up is important for acute osteomyelitis cases to ensure that no relapse is occurring. Reactivation of chronic osteomyelitis scan occurs even 10 years after primary therapy is concluded.[51] Reconstructive and regenerative procedures can be undertaken upon full resolution of the condition as long as the predisposing co–risk factors are well controlled. Otherwise, the patient may experience recurrence of the condition.

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