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Osteomyelitis of Jaws: A Narrative Review

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Abstract

Osteomyelitis of the jaws is a rare but potentially stubborn complication resulting from dental infections, trauma or compromised vascular supply. It is a localized infection more commonly seen in the immunocompromised patient, and usually affects the mandible more than the maxilla. Although it involves both the cancellous and cortical bone, strictly speaking, Osteomyelitis is the inflammation of the medullary portion and rarely stays confined to the endosteum. Oral microflora, especially Staphylococcus aureus, is the causative organism behind the disease and it is generally classified as either acute or chronic with varying clinical presentations. However, pain, swelling, fever and pus discharge can be considered the hallmark signs. Treatment generally involves surgical debridement and intensive antimicrobial therapy, however It's unpredictability and possible complications, make it a subject of interest to clinicians and researchers all over the world.

Keywords: Osteomyelitis, mandible, maxilla, bone, infection

Introduction

Ever since the antibiotic revolution in the year 1928, incidences of infectious pathologies have drastically decreased. Greater attention to sterilization and disinfection, standardization of procedures and patient education have also played a pivotal role. However, now, the world is re-entering a phase where bacteria have become a threat. In 2013, CDC warned that humanity is now in a "post-antibiotic era" and in 2014, WHO declared the unavoidable threat of antibiotic resistance.[1] Infection is a relatively rare but possible complication which may arise even after routine dental extraction. Although, mostly healing occurs uneventfully in the healthy patient, sometimes if the sterilization control is lacking or if the patient is immunocompromised, untoward events like infection with concurrent inflammation can occur.

Osteomyelitis can be defined as "an inflammation of bone marrow with a tendency for progression, involving the cortical plates and often periosteal tissues."[2] Despite the advances in the medical field, it still remains a difficult condition to diagnose and treat for practitioners

The infection usually spreads via the oral cavity, skin, sinuses or through a hematogenous route. Local factors, mostly, compromised oral health, periapical pathologies or trauma are the most common aetiology.

Classification

Several classification schemes have been proposed to facilitate easy differentiation and understanding of OM but because of its heterogenous nature, it is not easy to do so. Moreover, most of these classification systems were developed for long bones which are vastly different from the jaws. Broadly, OMJ can be classified as acute and chronic

The most popular classification scheme is the *Zürich system* [3], which relies on clinical presentation, aetiology and duration -

A. Acute Osteomyelitis:

1. Odontogenic origin
2. Trauma-associated.
3. Bone pathology–associated
4. Foreign body–induced
5. Immunosuppression-related
   - AIDS
   - Agranulocytosis
   - Anemia
6. Associated with systemic conditions
   - Diabetes
   - Autoimmune diseases

B. Secondary Chronic Osteomyelitis

(No further sub-classification)

C. Primary Chronic Osteomyelitis

1. Juvenile onset
2. Adult onset
3. Syndromic forms

The Topazian [4] system which factors in the presence of purulence along with the clinical presentation, imaging and etiology -

A. Suppurative Osteomyelitis

1. Acute Suppurative Osteomyelitis
2. Chronic Suppurative Osteomyelitis
   i. Primary
   ii. Secondary
   iii. Infantile

B. Non-suppurative Osteomyelitis

1. Diffuse Sclerosing Osteomyelitis
2. Focal Sclerosing Osteomyelitis (Condensing Osteitis)
3. Proliferative Periostitis (Garre's Osteomyelitis)
4. Osteoradionecrosis

Proliferative periostitis of Garre (sclerosing osteomyelitis)

Carl Garré in 1893 first described proliferative periostitis, also known as chronic non-suppurative sclerosing osteomyelitis, proliferative periostitis of Garré, sclerosing osteomyelitis, and periostitis ossificans, as focal thickening of the periosteum and cortical bone of the tibia induced by irritation. The disease primarily affects children and young adults but may occasionally occur in older individuals.[4]

Radiographic findings typically include bone hyperplasia with areas of bone destruction. Some features may mimic the radiographic appearance of malignant bone tumours. Characteristically, Garre's osteomyelitis presents as radiopaque laminations parallel to the cortical bone surface with periosteal thickening and subperiosteal new bone formation.[20]

SAPHO syndrome

In 1986, Chamot et al. described a syndrome associated with synovitis, acne, pustulosis, hyperostosis and osteitis (SAPHO syndrome), palmoplantar pustulosis is also a common finding in medical examination. Ideally, a whole-body scintigraphy should be performed to exclude other lesions especially if SAPHO syndrome is strongly suspected.

An investigation by Suei et al.[21] revealed characteristic radiographic findings of the mandibular lesion in SAPHO syndrome: which were solid periosteal reactions, bone resorption affecting the external surfaces and enlargement of affected bone. [21] They also suggested that the periosteum rather than the bone is the site of inflammation because it is the periosteum which produces pro-inflammatory cytokines (IL-8 and TNF-α) which stimulate osteoclasts and osteoblasts, thereby promoting periosteal bone formation and cortical bone destruction respectively.[21, 22]

Clinical Features

Clinically, four types of osteomyelitis of the jaws are encountered.[4]

(1) acute suppurative;

(2) secondary chronic, when it begins as acute osteomyelitis and becomes chronic with time;

(3) primary chronic, a form having always been a low-grade infection and has manifested no acute phase previously;

(4) nonsuppurative

Acute osteomyelitis may present as moderate grade fever, malaise, facial cellulitis, trismus, and significant leukocytosis.[5] Swelling with erythema of the overlying skin typically reflects the cellulitic phase of the inflammation of affected bone and is most commonly seen in acute phase. [9] Other clinical manifestations may include swelling of local lymph nodes, draining sinus tracts, bone exposure and sequestrum formation.[10]

If acute osteomyelitis is not treated adequately, generally past the 30-day mark, it progresses into a subacute of chronic form.[4, 9] As the osteomyelitis turns chronic, the clinical signs diminish and become limited to fistulas,

In chronic secondary osteomyelitis, the clinical findings usually are limited to fistulas, tissues induration, and a "wooden" consistency to the affected area with a deep, boring type of pain, usually associated with palpation.[4, 10]

If a suspected case of chronic osteomyelitis is not preceded by any acute symptoms, and is associated with dull pain, slow growth in size and sequestra formation with an absence of fistulas, it is termed primary chronic osteomyelitis.[4] Clinically, primary chronic osteomyelitis can be linked with episodes of varying intensity of symptoms, lasting from days to weeks. During periods of activity, pain, swelling, restricted mouth opening along with regional lymphadenopathy may be observed.[11]

Imaging

Conventional Radiographs: Positive radiographic findings are usually delayed or secondary, except in the cases of associated fractures.[5] The full extent of bone dissolution cannot be determined radiographically until 3 weeks after initiation of the osteomyelitic process.[4] However, as it is inexpensive and readily available, Orthopantomograms are still widely used.

Computed Tomography: Because of its sensitivity, high-resolution CT detects early bone changes before they can be seen on conventional films.[4] A retrospective study classified CT patterns of osteomyelitis into four types, lytic, mixed, sclerotic, and sequestrum patterns, according to the amount of bony sclerosis, osteolysis, and the presence of a sequestrum. [13] According to Yoshiura K. et al, lesions with a lytic or sequestrum pattern were relatively localized, whereas the majority of the mixed and sclerotic patterns were diffuse. Mixed and sclerotic cases showed significantly larger extents than the lytic cases.[13]

Scintigraphy: Methylene diphosphonate, gallium 67, and indium 111 are some of the radioisotopes used to identify altered bone physiology.[10] These radioactive substances are distributed and taken up by the entire skeleton, eventually getting accumulated in areas of increased osteoblastic activity. Sodium iodide containing, rectilinear scanner or a scintillation camera, emits light, which is then used to obtain images of the isotope-containing areas.[4]

Etiopathogenesis

Microbiology

More than 500 species of bacteria have been identified in the mouth.2 Until recently estimates of the involvement of S. aureus and S. epidermidis in osteomyelitis of the jaws ranged between 80% and 90%; Staphylococcus continues to be listed frequently in connection with osteomyelitis of the jaws [4] However, the picture of S. aureus as the primary offending pathogen does not hold true with regard to osteomyelitis of tooth-bearing bone.[5] Because of the abundant and vast microflora of the oral cavity, osteomyelitis of this region can be due to any invading bacteria which breaches the mucosa.

In one study, S. sanguinis, S. viridans, S. saprophyticus and P. acnes were the isolated species. [6] Staphylococcus, Actinomyces, Bacteroides, Klebsiella, Fusobacterium, Lactobacillus, and Haemophilus were also reported.[7] Acute osteomyelitis of the jaws is usually a polymicrobial disease, including the involvement of streptococci, bacteroides, peptostreptococci, and other opportunistic bacterias.[4]

Definitive antimicrobial therapy, however, should be based on the final culture and sensitivities.[2] At the same time the difficulties in obtaining pus and representative specimens in cases of osteomyelitis of the jaws must be acknowledged.[4] Antimicrobial agents are routinely administered in chronic osteomyelitis without sufficient bacterial information, however, resulting in treatment failures.

Progression of disease

Osteomyelitis is initiated by a contiguous focus of infection or hematogenous spread.[4] The initial source of the infection, which later exacerbates and spreads, is typically a type of dental infection—most often a dento-alveolar abscess. These infections generally spreads mechanically through newly opened pathways, allowing the bacteria to enter healthy blood vessels within the bone cortex and marrow.[14] When pus and inflammation develop in the bone marrow, the resulting increase in intramedullary pressure further reduces blood flow to the bone.[9] As the effectiveness of host defenses and therapy increases the osteomyelitic process may become chronic. Fragments of necrotic bone (sequestra) may separate

from viable bone. Smaller sections of bone may be lysed while larger ones may be isolated by a bed of granulation tissue, encased in new bone (involucrum)[4]

Predisposing factors

Both local and systemic factors, which diminish host defenses can contribute significantly to the emergence and clinical course of the disease. [9] Diabetes, autoimmune disease, agranulocytosis, leukemia, severe anemia, malnutrition, syphilis, cancer chemotherapy, steroid drug use, sickle cell disease, and acquired immunodeficiency syndrome are all conditions associated with osteomyelitis of jaws. [4]

Additionally, individuals who are undergoing or have previously undergone osteo-chemotherapy with bisphosphonates, or who have received radiation therapy, represent a distinct and high-risk group for developing osteomyelitis in the jaw (maxillomandibular region).[10]

Management

The first step in the management of osteomyelitis is ideally, diagnosing the condition correctly. A tentative diagnosis should be made from clinical and radiographic evaluation, and tissue diagnosis. Often, certain malignancies mimic the presentation of osteomyelitis and must be ruled out first prior to starting treatment by a histopathological examination. [9] The mainstay of treatment remains a combined surgical and antimicrobial therapy for all suppurative and chronic cases. Infantile osteomyelitis which itself is a rare entity, may however respond to intravenous medication alone. [10]

Antimicrobial therapy

Precise identification of the pathogen or pathogens and treatment with the most effective narrow-spectrum antibiotic to which the organisms are susceptible is pertinent.5 An antibiotic usually should not be used until culture material is obtained and Gram stain examination and culture and sensitivity tests have been performed. However, time is critical in patients with acute osteomyelitis or those with clear systemic involvement, empirical antibiotic choices often must be made without aid of laboratory results. [4]

Several penicillin resistant bacteria like Porphyromonas, Prevotella, and Fusobacterium have been isolated from affected jaws. Additionally, antibiotics like metronidazole must be included to thus covering aerobic streptococci as well as penicillin-resistant anaerobes. [4] Clindamycin alone, clindamycin plus metronidazole, and cephalosporins are some other drugs found to be effective. 4 In presence of methicillin-resistant S aureus strain, a combination therapy including vancomycin or rifampin followed by oral ciprofloxacin or levofloxacin may be considered. [10]

The recommended duration varies from 2 weeks to 6 weeks, typically beginning with intravenous antibiotics followed by a variable period of oral antibiotics. [16] Kim et al. suggested a combination of surgery with antibiotic treatment for at least 8 weeks after surgery. [15] With first 2 weeks of parenteral administration and the following 6 weeks of oral antibiotics. A successful outcome was seen in 94.9% of patients with their new treatment protocol, and in 60% of control patients. [15]

Local antibiotic delivery

Surgical drains or rubber tubes may be placed against the involved bone in order to allow drainage of pus and provide a route for irrigation with antimicrobial solutions, thereby reducing the bacterial load. Unless surgical debridement to the point of bleeding is achieved, irrigation alone is unlikely to be effective and only delays the treatment. [4] A suction-irrigation system may be used after appropriate debridement of the diseased bone which includes:

2 drainage tubes of 1.6mm diameter are placed percutaneously at each end of the wound, against the

surface of the mandible. These tubes are sutured to the skin and the wound is closed. Then an irrigating solution, usually normal saline with a beta lactam class antibiotic, is pushed through one tube and the other tube serves as the drain and may be connected to a source of suction. [17] Antibiotic-Impregnated Beads Antibiotic-impregnated acrylic beads have been used in the treatment of osteomyelitis to deliver high concentrations of antibiotics into the wound bed and in immediate proximity to the infected bone

Surgical interventions

The main aim of treatment is to improve the blood flow which is achieved by debriding the necrotic or poorly vascularized bony sequestrum. Sequestrectomy and saucerization generally is the treatment of choice. [9] Sequestrectomy involves debridement and removal of the avascular sequestrum and only the cortical plates in the infected area are removed. In saucerization, the adjacent bony cortices are also removed and open packing is performed to permit healing by secondary intention. [9]

Decortication, which is a more extensive approach, may require rigid fixation to reduce chances of pathologic fractures. Neurovascular bundle lateralization may also need to be performed based on the area of involvement. Both intraoral and extraoral approaches can be used, but it should permit large removal of cortical bone. The steps in decortication are [4]

(1) creation of a buccal flap by a crestal incision extending along the necks of the teeth,

(2) reflection of the mucoperiosteum to the inferior border;

(3) removal of teeth in the involved area, and

(4) removal of the lateral cortical plate and inferior border with chisels.

Jaw resection is typically reserved as a last resort when previous surgeries or therapies have been unsuccessful. 9 In a study by Baur DA et al. partial resection with simultaneous reconstruction was performed in 4 patients and in 4 cases the reconstruction was delayed. [7] Rigid internal fixation has simplified the postoperative course by providing a means for immediate function of the jaws. Advances in technology and rigid technology have allowed 3-D printing of models and plates which allow for extremely precise ablative and reconstructive surgery.[9]

Baur et al. suggest that sub therapeutic conservative management of the necrotic bone in the jaws often results in a prolonged treatment period, multiple surgeries, and increased cost that could potentially lead to the level of morbidity that is no better than that from an aggressive surgical management. Therefore, they find it safe to state that, even with an effective antibiotic treatment or adjunctive therapies, a thorough surgical removal of the diseased segment is the essential component of the treatment. [7]

Hyperbaric Oxygen Therapy

It is a well-known fact that infection is an oxygen consuming process which leads to local hypoxia, which further encourages the spread of infection and inculcates the growth of anaerobes. [18] HBO is indicated in chronic (duration more than 3 months) cases refractory to adequate treatment, in cases of concomitant systemic disease compromising the vascularisation or in cases of severely compromised immune response. The following protocol was recommended 20 sessions 2.8-3.0 atm 1oo% O2 for 90 min followed by 20 postoperative sessions. [19] The use of HBO as an adjuvant to surgical procedure has been practiced by several clinicians due to the high-quality evidence including several in-vitro and in-vivo studies. [18] They have reported improvement or success when HBO was added to a difficult or desperate clinical situation. [18] The benefit of HBO is believed to be

the greatest for dominantly anaerobic infections. However, the efficacy of the intervention in early or acute osteomyelitis need large scale prospective trials and further research. 10 Further, data must be gathered to demonstrate the value of this therapy in non–radiation-related osteomyelitis. [10]

Complications

Chen et al. in their study, reported that an age > 65 years, admission temperature > 39 degree Celsius, and an admission WBC count >15×109/L were found to be the risk factors for life-threatening complications.[23] Additionally, presence of infected teeth, Pre-admission antibiotic administration without consultation, involvement of ramus lingually and the condylar neck, were also associated with high rates of recurrence. [23]

The most common complication of osteomyelitis of the jaw is formation of abscess in the adjacent soft parts. Septicaemia with its grave prognosis can also be encountered. [24] Cerebral abscess, meningitis, pansinusitis, erysipelas and tetanus have all been noted. Late complications sometimes seen are deformities of various types due to loss or maldevelopment of bone, and ankylosis of the temporomandibular joint [23, 24]

Conclusion

Osteomyelitis of jaws is an anomalous condition which impacts patients all over the world. Although rare, it is essential for clinicians to have an in-depth understanding of the condition to facilitate early diagnosis and management. With the current era of antibiotic resistance, it is prudent to focus on prevention rather than cure and maintain sterility even while performing a humble extraction.

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