Osteomyelitis of Jaw: A Review

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Abstract

Osteomyelitis of the jaw in dental practice has seen to be decreased due to the increasing awareness among the general population about oral hygiene and the various treatment modalities available in dentistry. Often the maxillofacial region is involved due to improper treatment, compromised immunity, fracture, or any other reason. It is due to the inoculation of bacteria because of multiple factors. The diagnosis is based on clinical findings, radiographs, and lab findings. Osteomyelitis is managed by both conservative and surgical management. With the availability of more potent antimicrobials and treatment under aseptic conditions, management of osteomyelitis has been possible.

Keywords: *Osteomyelitis, Sequestra, radiography, management.*

Introduction

Once being a dreaded disease in the pre-antibiotics era, the incidence of osteomyelitis of jaws has become less, because of awareness, availability of antimicrobials, and better dental health services. The word 'osseous' is from Latin origin which means bony, 'osteon' from Greek origin meaning bone, 'myelos' means marrow, and 'it is' means inflammation.[1]

Osteomyelitis may be defined as an inflammatory condition of the bone, that begins as an infection of the medullary cavity and Haversian system of the cortex and extends to involve the periosteum of the affected area. It is an inflammatory process with the tendency to progress. Because of poorly vascularized cortical plates, mandible shows a higher incidence of osteomyelitis.[2-5] Mandible gets its blood supply from the inferior alveolar neurovascular bundle. Maxilla being less dense shows less incidence as it is highly vascularized and gets its blood supply from numerous feeder vessels. Maxillary posterior region is less affected than the mandibular posterior region. Even in mandible,the most common sites are body, symphysis, angle, ascending ramus, and the condyle. The severity and high frequency of odontogenic infections in daily dental practice and the heavy bacterial load of the oral cavity is one of the major reasons why osteomyelitis of the jaw is more common than any other skeletal bone.[2-5]

Staphylococcus aureus or *Mycobacteria* are the most specific bacteria causing infection, other causes may include trauma, radiation, or certain drugs.[6] Diminished host defense mechanism whether local or systemic contributes to its emergence. Diabetes, malignancies, autoimmune diseases, malnutrition are some of the other causes that may associate with osteomyelitis.[3] Osteomyelitis can be described by inflammation of the mouth, exposed bone in the mouth which fails to heal, unhealing extraction socket, presence of fistula from mouth to lower skin, or development of sequestra.[7,8] It starts as an infection of the medullary cavity, spreads to the Haversian system, and gradually involves the periosteum of the infected area leading to severe tissue and bone destruction.[9-11] There is progressive inflammatory osteoclasia and ossification.[6] Radiation related osteomyelitis shows a delayed onset and tooth extraction and implant placement is also contraindicated in that area which has been irradiated.[12]

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Predisposing Factors: The predisposing factors of osteomyelitis are: (1) A delayed host defense mechanism or any condition that affects the host resistance, (2) Lack of vascularity of the host bone at a local or systemic level, and (3) virulence of microorganisms. The host defense system gets by the presence of any immunocompromising condition like Diabetes mellitus, leukemia, malnutrition, agranulocytosis, severe anemia, chronic alcoholism, drug abuse, sickle cell disease, typhoid, chemotherapy, irradiation, steroid use, hepatitis, HIV, glomerulonephritis, systemic lupus erythematosus, etc.[1,3,13] The vascularity of the host bone is altered due to therapeutic irradiation, Paget's disease, bone malignancy, fibrous dysplasia, osteoporosis, mercury; arsenic, and bismuth causing metallic bone necrosis. In the maxilla, osteomyelitis can occur due to any pansinusitis or facial cellulitis. Due to the lysosomal activity,certain organisms form thrombi. A protective barrier is formed around the infective foci by the pathogen born bioactive peptides and the chemoattracted leukocytic purulence which leads to lysosomal and enzymatic destruction of the tissues along with microvascular thrombi formation. The organisms further proliferate in the host medium but remain protected from the host defense mechanism. [1,3,13]

Etiology:

Odontogenic infection arising from pulpal, periapical, pericoronal tissues, infected cyst, socket, or tumor are one of the major cause of osteomyelitis. Trauma is the second most common cause especially compound fracture and any iatrogenic surgery.Oro-facial infections derived from periostitis following the gingival recession, infected lymph nodes from furuncles, peritonsillar abscess, lacerations are also seen to cause osteomyelitis. Furuncle on the face, upper respiratory tract infection, mastoiditis, wound on the skin, middle ear infection, systemic tuberculosis cause infection to spread via hematogenous route.[1,14]

Pathogenesis: The primary hematogenous osteomyelitis is very rare but can be seen at a very young. In adults, the primary cause being the inoculation of bacteria in the jaw bone. This insult results in the initiation of an inflammatory process or cascade. In a normal or healthy host, this may self-heal or self-limit itself, but in a compromised host it can lead to a process that is no more considered physiologic but pathologic. With increased inflammation, there is increased blood flow to that area and increased hyperemia. Further leucocytes are recruited to fight against the infection.[3] Pus formation occurs and a continuous supply of bacteria and cellular debris occurs that cannot be eliminated by the body host defense mechanism. When the pus and the inflammatory process starts in the bone marrow, the intramedullary pressure increases, and the blood supply decreases to this region. The pus then travels via Haversian and Volkmann's canals to spread through-out the medullary and cortical bone. Once the pus has entered the cortical bone and gets collected under the periosteum, the local conditions are aggravated by compromising the blood supply to that area.[3] Finally, the pus exits the soft tissue by intraoral or extraoral fistulas.[3] Osteomyelitis starts by bacterial invasion in the cancellous bone, followed by acute inflammation hyperemia, increased capillary permeability, and infiltration of granulocyte. It is an infectious process of the medullary myeloid cavity and only the mandible and the calvarium has myeloid compartment.[1]

Microbiology

Aerobic streptococci (alpha-hemolytic streptococci, *streptococci viridans*), anaerobic streptococci; and other anaerobes, such as Peptostreptococci, Fusobacteria, and Bacteroides cause many cases of osteomyelitis. Sometimes, anaerobic or microaerophilic cocci, Gram-negative organisms such as *Klebsiella, Pseudomonas,* and *Proteus* are also found. *M. tuberculosis, T. pallidum,* and *Actinomyces* species cause some specific kind of osteomyelitis. Marx et al. (1992), identified Actinomycoses, *Eikenella,* and *Arachnia* as pathogens of some other refractory osteomyelitis.[1,3, 14]

Classification:

Table 1. Classification of osteomyelitis of jaw

Historically accepted classification based on the clinical course: Hundson's classification of osteomyelitis of jaw:	**Acute forms** Contiguous focus: trauma, surgery, and odontogenic infection • Progressive: burns, sinusitis, and vascular insufficiency • Hematogenous (metastatic): developing skeleton (children)
	Chronic forms • Recurrent multifocal: Developing skeleton (children), Escalated osteogenic activity (< age 25 years) • Garre's: Unique proliferative subperiosteal reaction, Developing skeleton (children to young adults) • Suppurative or Non-suppurative: Inadequately treated forms, Systematically compromised forms, and Refractory forms (CROML: Chronic Refractive Osteomyelitis) • Diffuse sclerosing: Fastidious organisms, and Compromised host or pathogen interface.
Classification is based on the pathogenesis of altered vascular perfusion as a major contributing factor to the presence and persistence of osteomyelitis as a clinical disease. (Vibhagool et al 1993).	**There are three types:** • Hematogenous osteomyelitis • Osteomyelitis secondary to a contiguous focus of infection • Osteomyelitis associated with or without peripheral vascular disease
Classification based on the presence or absence of suppuration	**Suppurative osteomyelitis** a. Acute suppurative (pyogenic) osteomyelitis b. Chronic suppurative (pyogenic) osteomyelitis i. primary ii. secondary c. Infantile osteomyelitis
	Nonsuppurative osteomyelitis a. Chronic sclerosing osteomyelitis i. Focal sclerosing osteomyelitis ii. Diffuse sclerosing osteomyelitis b. Garre's sclerosing osteomyelitis c. Actinomycotic osteomyelitis d. Radiation osteomyelitis (osteoradionecrosis) e. Specific infective osteomyelitis i. Tuberculosis ii. Syphilis
Classification and Staging system of osteomyelitis developed by Clerny et al. and Vibhagool	**Anatomic type:** **Stage I** Medullary osteomyelitis It involved medullary bone. Without cortical involvement, usually hematogenous **Stage II** Superficial osteomyelitis Less than 2 cm of the bony defect without cancellous bone **Stage III** Localized osteomyelitis Less than 2 cm bony defect seen on the radiograph, the defect does not appear to involve both cortices **Stage IV** Diffuse osteomyelitis The defect is less than 2 cm Pathological fracture, infection, and nonunion
	Physiological type: A Host: Normal host; B Host: Systemic compromise (Bs); local compromise (Bl); C Host: Treatment is worse than the disease

Systemic or local factors that affect immune surveillance, metabolism, and local vascularity.

a. Systemic (Bs): Malnutrition, renal or hepatic failure, diabetes mellitus, chronic hypoxia, immune deficiency or suppression, malignancy, extremes of age, autoimmune disease, and tobacco and alcohol abuse.

b. Local (Bl): Chronic lymphedema, venous stasis, major vessel disease, arteritis, extensive scarring, radiation fibrosis, small vessel disease, and loss of local sensation.

Clinical Presentation: The classical clinical features are pain, swelling, and erythema of the overlying tissues, fever, paresthesia of the inferior alveolar nerve, paresthesia of lower lip, adenopathy, trismus, malaise, fistulas, and sometimes pathologic fracture.[1-7] The pain is a generally deep and boring type. In the case of Acute osteomyelitis swelling and erythema is very common to see which indicates the cellulitic phase of the inflammatory process under the bone. Fever often accompanies it but in the case of chronic osteomyelitis, it is not seen. Due to the inflammatory process, there is continuous pressure on the inferior alveolar nerve, hence, paresthesia of the inferior alveolar nerve is the characteristic feature of osteomyelitis. In the case of inflammation in the muscle of mastication of the maxillofacial region, there is trismus. The patient gradually feels weak and malaise and may associate with any other systemic disease. Generally intraoral and extraoral fistulas are seen in the case of chronic osteomyelitis.[3]

In Acute osteomyelitis, the early cases are characterized by generalized symptoms like fever; malaise; nausea; vomiting; anorexia, deep-seated boring and continuous pain, intermittent paresthesia or anesthesia of lower lip, facial cellulitis or indurated swelling of moderate size, thrombosis of inferior alveolar vasa nervorum, rise in pressure from edema in the inferior alveolar canal, teeth are tender to percussion and loose and trismus. In established cases there is deep pain, malaise, fever, dehydration, anorexia, tender on percussion of the teeth in that area, teeth become loose, purulent discharge through sinuses, presence of fetid odor, trismus may be present, acidosis, toxemia, regional lymphadenopathy.[1,2]

In chronic cases, there is pain and tenderness but the pain is minimal, non-healing bony and soft tissue wounds with induration of the soft tissue, intraoral or extraoral draining fistula, thickened or wooden characteristic of bone, enlargement of the mandible because of deposition of subperiosteal new bone, a pathological fracture may occur. [1,2]

Lab findings: The laboratory studies show slightly raised ESR (Erythrocyte sedimentation rate), elevated C-reactive protein (CRP), moderate leukocytosis (PMNL), anemia, and albuminuria In acute cases leukocytosis is common but in chronic cases, it is not common.

Histopathology of biopsies shows scattered inflammatory infiltration, mainly lymphocytes, histiocytes, plasma cells, and neutrophil granulocytes, increased osteoblast, thickened bony trabecula, and fibrous marrow replacement.[8-15s]

Radiographic findings:

The following method can be used for radiographic analysis:

Conventional radiography: The radiographic changes can be appreciated after 3 weeks of the onset of osteomyelitis. At least 40-60% of bone destruction should be there to mark any radiographic changes. This alteration is seen 4-8 days after the onset of acute osteomyelitis. In the early stages, areas of sclerosis and osteolysis with varying degrees of periosteal reaction is seen as mixed radiodensities. There is a "mottled appearance"due to the widening of marrow space and enlargement of Volkmann's canals. The irregular lines and zones of radiolucency give "moth-eaten appearance" due to the presence of granulation tissues between the livings and dead bone. In later stages, gradually the cortical bone gets involved and an island of cortical bone gets mobilized by the osteolytic process which is devitalized and is favorable for the precipitation of calcium. These separated devitalized calcified bone with resorbed periphery act as a foreign body and are prominent in the radiograph which is called a sequestrum. The subperiosteal new bone is seen as opaque lines over the cortex called Involucrum. A "fingerprint" or "onion peel appearance" is seen at the place where the new bone is superimposed upon the jaw.[1-4]

Imaging: This includes computerized tomography (CT), Positron emission tomography (PET), radioisotope scanning. Computed tomography gives a more clear picture of the involved calcified bone and the cortical plates. There is less radiation exposure to the patient.

Radioisotope Tc-99m methylene diphosphonate bone scanning helps to identify the margins of calcified tissue involvement, but, it has very poor resolution. Positron emission tomography (PET) recently have been more promising for evaluating metabolic activities. It uses radioisotopes of physiologically active compounds like glucose, ammonia, fluoride and the changes can be seen as early as 3 days.[1]

Treatment: The treatment of osteomyelitis requires both medical and surgical approach. Very rarely it requires intravenous antibiotic therapy like in the case of infantile osteomyelitis [3]. Based on the histopathological features i.e. gram stain results of the exudate or suspected pathogen, empirical antibiotic treatment should be started.Culture and sensitive reports may take time but guide the surgeon for treating the patient.[3]

In general, treatment[1] consists of:

i) Debridement

ii) Control of infection: acute infection is controlled through antibiotic therapy. However, as there is no circulation in the necrotic bone so the antibiotic does not reach.

iii) Hospitalization

iv) Other supportive treatment: a) hydration: fluid therapy, b) high protein and vitamin diet.

v) Analgesics: Narcotic and non-narcotic analgesics. Bupivacaine (Marcaine), alcohol nerve blocks, nerve avulsion, rhizotomy.

vi) Good oral hygiene: oral rinses, such as 1% sodium fluoride gel, 1% chlorhexidine gluconate, and plain water help to prevent radiation-induced caries from the xerostomia and mucositis by providing some local augmentation of the host immune antimicrobial activity.

vii) Frequent irrigations of wounds.

viii) Exposed dead bone: small pieces of bone may become loose and can be removed easily.

ix) Hahn and Corgill in 1967 gave the treatment of small areas by drilling multiple holes into vital bone as a means to encourage sequestration.

x) Sequestrectomy is performed intraorally, because of skin and vascular damage resulting from irradiation.

xi) Pathological fractures are not so common. These may occur from minor injuries. The fractures do not heal readily. Excision of necrotic ends of both fragments and replacement with a large graft is the best treatment. Reconstruction of bone defects usually warrants major soft tissue flap revascularization support.

xii) If there is persistent pain, infection, or pathological fracture, bone resection is performed. It is done intraorally to avoid orocutaneous fistula in radiation compromised skin.

xiii) Hyperbaric oxygen therapy is a useful adjunct. It increases the tissue oxygenation levels which helps to fight any anaerobic bacteria present in these wounds.[1]

Surgical Options: The classic treatment is sequestrectomy and sauterization. The main aim is to remove the necrotic or less vascularised bony sequestra and improve the blood flow. In sequestrectomy infected and avascular pieces of bone are removed, it is generally the cortical plates in the infected area. In saucerization adjacent, bony cortices are removed up to a good bleeding bone and an open pack is given for healing by secondary intention. Decortication is also done in which removal of dense, basically chronically infected and poorly vascularized bony cortex is performed and a vascular periosteum adjacent to the medullary bone is placed to increase the blood supply.[3]

Conclusion

Osteomyelitis of the jaw is an inflammatory condition that is multifactorial. A dentist should be aware of the predisposing conditions and know how to manage the condition.

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