Osteomylitis of the jaw.

OSTEOMYELITIS is an inflammation of the medullary portion of the jaw bone which extends to involve the periosteum of the affected area. **Classification.** Osteomyelitis may be classified as suppurative or nonsuppurative and as acute or cronic.

Etiology and pathogenesis. The disease primary is caused by odontogenic infection originating from pulpal or periodontal tissues. Trauma, especialy compound fractures, is the second leading cause. Infections derived from periostitis following gingival ulceration or from lymph nodes infrcted from furuncles or lecerations and hematogenous origin account for small additional number of jaw infections.

The infection becomes established in the calcified portion of bone when pus in the medullary cavity or beneath the periosteum obstructs the blood supply. The infected bone becomes necrotic once ischemia sets in. An underlying alteration of host defenses is present in the majority of patients with osteomyelitis of the jaw. Conditions altering the vascularity of bone predispose the patient to the onset of osteomyelitis and include radiation, osteoporosis, osteopetrosis, Paget's disease of bone, and bone malignancy.

Osteomyelitis of the maxilla is much less frequent than that of the mandible because the maxillary blood supply is far more extensive. Compromise of the blood supply is a critical factor in the establishment of osteomyelitis. The mandible receives its major blood supply from the inferior alveolar artery. A secondary source is the periosteal supply giving off nutrient vessels that penetrate the cortical bone and anastomose with branches of the inferior alveolar artery.

Most periapical and periodontal infections are localized by the production of a protective pyogenic membrane or soft tissue abscess wall. If sufficiently virulent, microorganisms may destroy this barrier. The process leading to osteomyelitis is initiated by acute inflammation. Tissue necrosis occurs as proteolytic enzymes are liberated and as destruction of bacteria and vascular thrombosis ensue. When pus accumulates, intramedullary pressure increases, resulting in vascular collapse, venous stasis, and ischemia. Pus accumulates beneath the periosteum, elevating it from the cortex and thereby further reducing the vascular supply. If pus continues to accumulate, the periosteum is penetrated and mucosal and cutaneous abscesses and fistulae may develop.

Microbiology of osteomyelitis. The establishment of an infectin in bone is related to virulence of the organism, the integrity and effectivness of the host defenses, and anatomic and structural factors. Until recently, it was estimated that Staphyloccocus aureus and Staph. Epidermidis (albus) were responsible for 80 to 90 per cent of cases of ostemylitis of the jaws. Mixed bacterial cultures, hemolytic streptococci, pneumococci, typhoid and acid-fast bacilli, Escherichia coi, and Actinomyces accounted for the remaining infections.

An apparent decline in the predominance of Staph. Aureus osteomyelitis has occurred that is attributed to the use of antibiotics and to the employment of more sophisticated culture methods that results in more accurate indentification, of responsible organism. Consequently, organisms not previosly associated with osteomyelitis are nw known to be responsible for many bone infections. For example, anaerobes had been thought to account for fewer than 1 per cent of all instances of osteomyelitis. The number of negative cultures ranged from 16 to 50 per cent. it is now recognized that failure to isolate such organisms may have rezulted froms an inability to culture anaerobes. Currently, anaerobes are frequently found to be associated with aerobic organisms in ostemyelitis, and anaerobic osteomyelitis may also occur alone. Now, is known that only accasional cases of ostemylitis of the jaws are truly caused by Staphylococcus aureus; it is found in cultures of jaw osteomyelitis because of contamination

from the skin via wounds and fistulae. Most are caused by aerobic streptoccocus (Strepyoccocus viridans), anaerobic streptococci, and other anaerobes, particulary Peptostreptococcus, fusobacterium, and Bacteroides, Occasionally, anaerobic or microaerophilic cocci, n gram-negative organisms such as Klebsiella, Pseudomonas, and Proteus are found. In addition, Mycobacterium tuberculosis, Treponnema pallidum, and Actinomyces species produce specific forms of osteomyelitis.

Clinical findings. Clinically, four types of osteomyelitis of the jaws are seen; 1) acute suppurative; 2) secondary cronic, a form that begins as acute ostemyelitis and becomes cronic; 3) primary cronic, a form that has manifested no acute phase and has always been a law-grade infection; and 4) nonsuppurative. The clinical findings differ, depending on the type of deseas prezent.

Early acute suppurative osteomyelitis of the mandible is usually characterized by deep intense pain, high intermittent fever, parasthesia of the mental nerve and a clearly identifiable cause. In the initial phase of the acute form, teeth are not loose, swelling is minimal, and fistulae are not present. At this juncture, the process is true intramedullary osteomyelitis. Immediate aggressive antibiotic therapy may prevent progression to involvement of the periosteum.

In established suppurative osteomyelitis, symptoms include deep pain, malaise, fever, and anorexia. Within 10 to 14 days after onset, teeth in the involved area begin to loosen and become sensitive to percussion. Pus exudes around the gingival sulcus or through mucosal and cutaneous fistulae. Firm cellulitis of the cheek, enlargement of the dimensions of the bone from increased periosteal activity, abscess formation with localized warmth, erythema, tenderness to palpation, and mental nerve paresthesia also may be noted. Trismus, submandibulary flegmone and regional lymphadenopathy is a constant finding in children. Temperature may reach 35 to 40 degree.

Leukocytosis consisting of a white blood cell count in the rang of 25000 to 35000 cells/mm2 with a shift to the left occurs. The erytrocyte sedimentation rate (ESR) may be slightly elevated.

Treatment. Osteomyelitis of the jaws usually requires both medical and surgical treatment. An underlying alteration of host defenses is present in many patients with osteomyelitis of the jaws. Steps should be taken to identify and correct factors that may delay recovery. Whenever possible, specimens should be obtained for Gram staining, aerobic and anaerobic cultures and sensitivity testing. Conventional radiographs and possibly bone scanning should be obtained to determine the extent of the disease, the existence of causative factors such as periapical abscesses and fractures, and the presence and location of sequestra. Extremely loose teeth and sequestra that are readily accessible should be removed early in the course of the disease. After the acute stages of the disease have subsided with intravenous or parenteral antibiotics and supportive measures, other treatment options include sequestrectomy, debridement, decortication, resection of infected bone, and immediate or late bone graft reconstruction.

Ostemyelitis of the jaws does not occur often these days. We can reduce the incidence further by using a rubber dam and apex locator during root canal therapy. Without a rubber dam, saliva can contaminate the tooth and our gloves, which in turn will transfer bacteria onto the reamers. Without an apex locator, we can over-instrument the canal and introduce bacteria on the reamer deep into the medullary portion of the jaw bone. This continuous introduction of bacteria into the medullary region can cause osteomyelitis in an immune-compromised patient.

Imaging. Imaging of suspected osteomyelitis of the mandible is accomplished by use plain films, supplimented as needed by computed tomiraphy (CT), magnetic resonanca imaging (MRI), and radionuclide bone scaning. Proper imaging aids in determining the

extend and degree of desease and the location of sequestra and assists in planing the approach and etend of surgery. An estimated 39 to 60 per cent of the mineralized portion of the bone must be destroyed before significant radiographic changes are noted. This degree of bone alteration requires a minimum of 4 to 8 days after the onset of acute ostemyelitis. The full extend of bo dissolution cannot be determined radiographically until 3 weeks after initiation of the ostemyelitic process.Therefore, in the early stage of the disease, the history and clinical finings may constitute the sole date base upon which a diagnosis is made.

Once ostemyelitis has become well estabished, radiographic changes usually demonstrate one of the following sets of characteristics described by Worth.

1. Scattered areas of bone destruction, varied in size and number, separated by variable distances and by bone that has normal or nerly normal appearance. There is a „motheaten"appearance because of enlagment of medullary spaces and widening of Volkmann's canals secondary to destruction by lysis and replacement with granulation tissur.
2. Bone destruction of varied extnd in which there are "islands", that is, sequestra, with a trabecular patten and marrow spaces. A sheath of new bone is often found, separated from the sequestra by a zone of radiolucency.
3. Stippled or granular densification of bone caused by subperiosteal depositon of new bone obscuring the intrinsic bone structure or deposition of new bone on surfaces of existing trabeculae at the expense of row spaces. The central sequestra usually present in osteomyelitis help to distinguish it from fibrous dysplasia.

Fibrous dysplasia, osteoid osteom, Paget's desease of bone, and malignant bone-producting tumors (such as ostesarcoma) may be confused with ostemyelitis, particularly when periosteal bone production has been marked, as can be the case in pre-adolescent patients. The laminated or „onion peel" appearance of bone in ostemyelitis is seen only rarely with tomors. Destructive jaw lesions such as lyphosarcoma, Ewing's tumor, and inraosseous squamous cell carcinoma may also mimic infection. Additionally, secondary infection may occur in bone tumors, potentially confusing the true diagnosis.

Careful history, physical examination, and radiogrphs adequate for visualization of the complete process with delineation of surface contours are necessary in distinguishing ostemyelitis from neoplasms.When history, physical findings, and radiographs are equivocal and resolution does not occur as expected, biopsy should be considered. For example, in a child without dental disease wo demonstrates a marked but atypical productive periostitis, biopsy shoul be done to destinguish Garre's ostemyelitis from sarcoma and to prevent delay in appropriate treatment.

Infantile osteomyelitis. Infantile osteomyelitis is seen most often in individuals a few weeks after birth and usually involves the maxilla. Before the advent of antibiotics, the mortality rate approached 30 per cent. It is thought to occur via a hematogenous route or from perinatal trama of the oral mucosa from the obstetrician's finger or the mucus suction bulb used to clear the airway immediatly after birth. Infection involving the maxillary sinus and contaminated human or artificial nipples have also been implicated as sources of infant infection.

Clinically, the patient presents with a facial cellulitis centered about the orbit. Irritability and malaise precede frank cellulitis and are followed by hyperpyrexia, anorexia, and dehidration. Convulsions and vomiting may occur.

Inner canthal swelling, palpebral edema, closure of the eye, conjunctivitis , and proptosis may result. A purulent discharge may be associated with the nose or eith an inner canthal sinus.

Intra-oraly, the maxilla on the affected side is swollen both buccaly and palatally, expecially in the molar region. Fluctuance is often present, and fistulae may exist in the

alveolar mucosa. During the early acute phase, little radiographic change is noted. Leukocytosis is generally present, with a shift to the left.

Staphylococcus aureus is usually the offending organism, although many ather organisms may occasionally be found, paticularly streptococci. Treatment should be prompt and aggressive to prevent permanent optic damage, neurologic complications, and loss of the tooth buds and bone. Instances of extention into the dural sinuses have been reported.

Intravenous penicillin and a penicillinase-resistant penicillin or clindamycin should be given, and drainage of all fluctuant areas should be established. Speciments should be obtained repeatedly for sensitivity testing and the antibiotic regimen appropriately adjusted. Supportive treatment with antipyretics, fluids, and proper diet should be provided.

Antibiotics and incision and drainage usually suffice for total managment of desease.

Antibiotics should be continued orally for 2 to 4 weeks after all signs of infection have disappeared. A conservative approach to sequestrectomy is advisable because of danger of damage to tooth buds. Occationally, totth buds are extruded, and sequestra form. When teeth in the area eventually erupt, they may be discolored. Scarring beneath eyelid has also been noted, causing an ectropion. Blepharoplasty is sometimes requied for correction.

Chronic recurrent Multifocal Osteomyelitis of children. An uncommon form of osteomyelitis of the jaws has been described involving children averaging 14 years of age and characterized by unpredictable periods of exacerbation and remission. Mandibular lesions are bilateral, irregular, motteled and multilocular and located in the mandibular rami. Antibiotics and debridment appear to have little effect on the prolonged course of the disease.

Garre's Sclerosing Osteomyelitis. Garre's Sclerosing Osteomyelitis also known as chronic nonsuppurative sclerosing osteomyelitis, proliferative periostitis of Garre, chronic osteomyelitis with proliferative periostitis and periostitis ossificans, was first described in 1893 by Carl Garre as an irritation-induced focal thickning of the periosteu.

Clinically, it is characterized by a localized, hard, nontender bony swelling of the lateral and inferior aspects of the mandibule. Lymphadepaphy, hyperpyrexia, and leucocytosis usually are not found. It is commonly associated with a cariousmolar, usually the first molar, and a hystory of past toothache. On accasion, no dental etiology or radiolucency may be found in the mandible.

Radiographically, a focal area of well-calcified bone priliferation ma be seen that is smooth and often has a laminated or „onion-skin" appereance.

Garre's ostemyelitis as thought to occur because of a low-grade infection or irrtation that influences the potentially active periosteum of young individuals to lay down new bone. Stafilococcus aureus and Staph. epidermidis have been found in specimens of bone that were obtaind by biopsy of lesions.

Ewing's sarcoma, osteosarcoma, and cortical hyperostosis (Gaffey's disease) are similar in radiographic appearence and must be differentiated from Garre's osteomyelitis. When no dental disease exists or when the lesion persists after treatment of dental pathology, biopsy should be considering. To establish the diagnosis. Histologically, the lesion consists of new bone formation with fibrous marrow containing chronic inflammatory cells.

Treatment is derected toward removing identifiable sources of inflammation. When the involved tooth is not restorable, extraction is indicated. Endodontic therapy has been succesfully employed, with resolution of the mass. Antibiotics not be administered unless signs of infection are present. Post-treatment follow-up is essential. If the lesion continues to increase in size after apparently successful treatment of the infection, early biopsy is indicate. Following successful treatment of the dental pathology, remodeling of the mandible generaly occurs naturally, but the deformity may remain static, and surgical recontouring may be required.

1 . Richard G. Topazian, "Osteomyelitis of the Jaws" Chapter 10 of *Oral and Maxillofacial Infections*