Oral and Maxillofacial Surgery/Fourth Year
سلوان يوسف

**Osteomyelitis of the jaws**

It is an inflammation of the medullary portion of the bone. The pathological process is rarely confined to the endosteum, it usually encompasses the cortical bone and the periosteum as well. The infection becomes established in the calcified portion of the bone when pus and edema in the medullary cavity and underneath the periosteum compromise the blood supply, the infected bone becomes necrotic leading to sequestrum formation. What differentiates osteomyelitis from dentoalveolar abscess, dry socket and osteitis is the tendency to progression.

The incidence of osteomyelitis is much higher in the mandible due to the dense poorly vascularized cortical plates and the blood supply primarily from the inferior alveolar neurovascular bundle. It is much less common in the maxilla due to the excellent blood supply from multiple nutrient feeder vessels. In addition the maxillary bone is much less dense than the mandible.

In the preantibiotic time the main presentation of osteomyelitis of the jaws was of acute onset with later transition to secondary chronic process with massive clinical symptoms and widespread bone necrosis, large sequestra formation and extra and intra oral fistula formation. After the introduction of antibiotics, the acute phases were concealed by these drugs without totally eliminating the infection rendering the subacute and chronic forms more prominent.

**Classification**

Many classification systems were proposed, these were based on clinical picture, radiology, etiology pathophysiology and anatomy. Topazian 1994 classified osteomyelitis based on clinical picture, radiology and etiology into:

**Suppurative osteomyelitis**

- Acute suppurative osteomyelitis.
- Chronic suppurative osteomyelitis (primary or secondary).
- Infantile osteomyelitis.
Nonsuppurative osteomyelitis

- Chronic sclerosing osteomyelitis (focal or diffuse).
- Garre’s sclerosing osteomyelitis.
- Actinomycotic osteomyelitis.
- Radiation osteomyelitis and necrosis (osteoradionecrosis).

Predisposing factors

- Virulence of microorganisms.
- Conditions affecting host resistance including: diabetes, autoimmune states, malignancies, malnutrition, and acquired immunodeficiency syndrome. The medications linked to osteomyelitis are steroids, chemotherapeutic agents, and bisphosphonates.
- Conditions adversely affecting blood supply of the jaws; radiation, osteopetrosis, osteoporosis, Paget’s disease of bone, fibrous dysplasia, bone malignancy.

Etiology and Pathogenesis

Osteomyelitis initiates from contiguous focus of infection or by hematogenous spread, most of the cases of osteomyelitis of the long bones are hematogenous in origin, whereas that of the jaw bones arises mainly from a contiguous focus of infection caused primarily by odontogenic infections or trauma.

In most cases the process starts as an acute inflammation; hyperemia, increased capillary permeability, infiltration of leukocytes, tissue necrosis and pus formation. This leads to increased intramedullary pressure resulting in impaired blood supply and ischemia, the pus may travel through the nutrient canals and accumulates underneath the periosteum further reducing the blood supply. Extensive periosteal elevation is seen more frequently in children because the periosteum is presumed to be less firmly bound to bone than it is in the adults.

With time the process becomes chronic, the inflammation regresses and granulation tissue is formed. The new blood vessels cause lysis of bone separating the fragments of necrotic bone (sequestrum) from the viable bone. Small sequestra may be completely lysed, while larger ones may be isolated by a bed of granulation tissue encased in a sheath of new bone (involucrum).

The fate of sequestra is either, they may be revascularized, remain quiescent or continue to be chronically inflamed requiring surgical
removal. Occasionally the involucrum is penetrated by channels (cloacae) through which pus escapes from the sequestra to the epithelial surface.

**Microbiology**

In the past, staphylococcal species were considered the major pathogen in osteomyelitis of the jaws. However, with refinements in the collection and processing of microbiologic specimens, a better picture of the disease-causing organisms was revealed. As with most oral infections the prime pathogenic species are streptococci and anaerobic bacteria. The anaerobes responsible are generally bacteroides, fusobacterium and peptostreptococci species. Often, the infections are mixed, growing several pathogens.

**Clinical presentation**

In acute suppurative osteomyelitis patients may present with:

- Deep intense pain.
- Swelling and erythema of the overlying tissues.
- Lymphadenopathy.
- High, intermittent fever.
- Paresthesia of the inferior alveolar (dental) nerve.
- Trismus.
- Malaise.
- Pus may be expressed through the gingival sulcus or through cutaneous or mucosal fistula.
- Teeth in the involved are may become loose and tender to percussion.

In chronic suppurative osteomyelitis (secondary) which is due to the progression of the untreated or incompletely treated acute osteomyelitis (about one month after the onset of the acute osteomyelitis), the clinical findings are limited to fistulae, induration of soft tissues, with less pain and tenderness on palpation, sequestrum formation and exposed bone.

In chronic primary osteomyelitis, which is not preceded by an acute form; the onset is insidious, with slight pain, slow increase in jaw size, gradual development of sequestra and often without fistulae.
Diagnostic imaging

Imaging is accomplished using plain films, CT scans, MRI, and radionuclide bone scanning. In the early acute phase the radiographic findings lag behind the clinical presentation, since it is estimated that 30%-50% of the mineralized portion of bone must be destroyed before significant radiographic changes are apparent, so it may take about 2-3 weeks for the radiographic changes to be seen.

- The orthopantomogram OPG is indispensible view in the initial evaluation of osteomyelitis. The radiographic changes may include:
  - Scattered areas of bone destruction described as "moth-eaten" appearance, because of the enlargement of the medullary spaces secondary to lysis and replacement by granulation tissues with loss of the normal trabecular pattern.
  - Radiopaque sequestra surrounded by new bone usually separated from the sequestra by a zone of radiolucency (involucrum).
  - Subperiosteal deposition of new bone that appear in the form of layered, linear radiopacity separated by radiolucent line from the cortical bone.
  - Pathological fractures.

- Computerized tomography (CT) scans have become the standard in evaluating maxillofacial pathology such as osteomyelitis. They provide three dimensional imaging not available on an orthopanoramic view. The CT scan can give very detailed images as to early cortical erosion of bone in osteomyelitis. The extent of the lesion, bony sequestra, calcified periosteal reaction, areas of cancellous bone sclerosis and pathologic fractures can be determined. CT scanning, like plain films, requires 30 to 50% demineralization of bone before changes can be seen, thus presenting an essential delay in diagnosis of osteomyelitis.

- MRI can assist in the early diagnosis of osteomyelitis by loss of the marrow signal before cortical erosion or sequestrum of the bone appears. Thus, MRI may benefit in identifying the earlier stages of osteomyelitis.

- Scintigraphy (bone scanning) has evolved to aid in the diagnosis of osteomyelitis. Technetium 99 bone scan is very sensitive in
highlighting areas of increased bone turnover; however, the scan is not very specific to areas of infection. With the addition of gallium 67 or indium 111 study, one can differentiate areas of infection from trauma or postsurgical healing as these agents specifically bind to white blood cells. Technetium 99 labeled methylene diphosphonate is given intravenously, it concentrates in areas of increased blood flow and osteoblastic activity, it is then imaged, and the picture obtained reveals the distribution of radionuclide in the areas of bone activity. The complete bone scan consists of 3 phases:

1. Flow study; consists of images taken 1-2 minutes after injection of the radionuclide.
2. Blood-pool study; image obtained 5-10 minutes after injection.
3. Delayed or bone study; multiple views obtained 2-4 hours after the injection.

The main advantage of scintigraphy is that it can detect changes as early as 3 days after the onset of the symptoms of osteomyelitis before the radiographic bone changes, so that early treatment can be instituted.

- Positron emission tomography (PET); is a relatively new technique to investigate bone and soft tissue pathologies, it involves the administration of radioactive tracing substance which is fluorine-18-fluoro-D-glucose ($^{18}$FDG) and the provision of 3-dimensional images that depict the biological activity rather than anatomy.

**Treatment**

The management of osteomyelitis of the maxillofacial region requires both medical and surgical interventions, Antibiotic therapy is rarely curative in later-onset cases, and the overwhelming majority of osteomyelitis cases require surgical intervention.

The principles of management include:

1. Establish diagnosis, based on history, clinical examination and imaging.
2. Determine the extent of infected bone.
3. Evaluation and correction of the compromised host defenses.
4. Removal of the source of infection; teeth, foreign bodies or implants.
5. Local incision and drainage of pus, if present.
6. Local curettage with removal of superficial sequestra and saucerization if necessary.
7. Collection of specimen for Gram stain, culture and sensitivity and histopathology.
8. Begin with broad spectrum antibiotic therapy and change to culture-guided antibiotic therapy as soon as possible. Usually a long term antibiotic treatment is needed (about 6 weeks or sometimes more). Antibiotics like penicillin G, ampicillin, amoxicillin/clavulanic acid, clindamycin, ciprofloxacin, levofloxacin, aminoglycosides, ceftriaxone, ceftazidime, cefepime and rifampin, or a combination of these antibiotics have been reported in the treatment of osteomyelitis.
9. Consider the application of local antibiotics.
10. More extensive surgical debridement (decortication, resection) if necessary.
11. Possible adjunctive hyperbaric oxygen therapy.

The classic surgical option includes sequestrectomy and saucerization. The aim is to débride the necrotic or poorly vascularized bony sequestra in the infected area to improve blood flow. **Sequestrectomy** involves removing infected and avascular pieces of bone.

**Saucerization** involves the removal of the adjacent bony margins overlying the focus of osteomyelitis and open packing to permit healing by secondary intention after the infected bone has been removed, it should be performed intraorally whenever possible.

**Decortication** of the mandible refers to the removal of the chronically infected lateral and inferior cortical plates of bone 1-2 cm beyond the area of involvement, the bone must be cut back to uninvolved area as evidenced by bleeding points in bone margins, it is indicated in the treatment of chronic osteomyelitis, it is performed intraorally.

In removing adjacent teeth and bone the clinician must be aware that these surgical procedures may weaken the jaw bone and make it susceptible to pathologic fracture. Supporting the weakened area with a fixation device (external fixator or reconstruction plate) and/or placing the patient in maxillomandibular fixation is frequently used to prevent pathologic fracture.

**Resection** of the area of osteomyelitis with immediate or delayed *reconstruction* may be necessary in treatment of persistant chronic osteomyelitis.

Some authors advocated the use of local antibiotics (e.g. irrigation system), this therapy works on the premise that high local levels of
antibiotics are made available, reducing the systemic antibiotics load and their associated complications.

Hyperbaric oxygen (HBO) treatment has also been advocated for the treatment of refractory osteomyelitis. This treatment method works by increasing tissue oxygenation levels that would help eliminating anaerobic bacteria present, but its use is still controversial.

**Other types of osteomyelitis**

**Infantile osteomyelitis**

It is seen in infants few weeks after birth, it affects the maxilla, delayed or inappropriate treatment may lead to major facial deformity. It occurs via hematogenous route or from perinatal trauma of the oral mucosa. Clinically; there is facial cellulitis centered about the orbits, palperbral edema, proptosis, irritability, fever, dehydration, convulsions, vomiting, a purulent discharge may be associated with the nose, intraorally the affected maxilla is swollen buccally and palatally. Staphylococcus aureus is usually the offending organism. Treatment should be prompt and aggressive to prevent permanent optic nerve damage, neurologic complications, loss of bone or teeth buds. It consists of intravenous antibiotics plus drainage of all fluctuant areas, supportive treatment with antipyretics, fluids and proper diet.

**Focal sclerosing osteomyelitis**

Localized areas of bone sclerosis associated with teeth with large carious lesions or deep restorations. It is also termed condensing osteitis, these lesions resemble other lesions that show the same pattern. It occurs in young individuals usually below 20 years of age, appears radiographically as a circumscribed radiopaque mass of sclerotic bone associated with a roots of teeth with thickened periodontal membrane. Clinically there is no expansion. Treatment is by elimination of the focus of infection either by extraction of the tooth or by appropriate endodontic treatment, in 85% of the cases this condition may regress, in some cases a residual area of sclerosis persists (bone scar).
**Diffuse sclerosing osteomyelitis**

It is highly controversial. It should be distinguished from fibrous dysplasia and florid cemento-osseous dysplasia. The diagnosis is made only when there is an infectious process directly responsible for the bone sclerosis.

It is mainly seen in patients in their mid-twenties to late-forties in age, clinically there is constant pain, mild bone expansion, with diffuse intramedullary sclerosis and poorly defined margins with focal areas of radiolucency.

Treatment consists of antibiotics for prolonged periods, surgical decortications with application of local antibiotics. Hyperbaric oxygen may be helpful.

**Garré's sclerosing osteomyelitis**

Also known as (chronic osteomyelitis with proliferative periostitis), (periostitis ossificans). **It is presumed to be first reported by Carl Garré (German physician) in 1893.** It represents a periosteal reaction to the presence of inflammation, and it is thought to occur due to low grade infection or irritation that influences the potentially active periosteum of young individuals to lay down new bone.

Clinically it is characterized by nontender bony swelling of the lateral and inferior aspects of the mandible with no systemic manifestations, it affects children and young adults (mean age 13 years) with a carious molar teeth usually mandibular first molar.

Radiographically there is a focal area of well calcified bone proliferation that is smooth and laminated usually described as "onion skin" appearance. The laminations of bone are parallel to each other and to the cortical surface often separated by radiolucent zones.

Treatment is by elimination of the source of infection either through extraction or endodontic treatment. Usually after successful treatment there is remodeling of the bony swelling, but if the deformity remains static surgical recontouring may be necessary.