Oral pathology

inflammatory diseases of the bone

Inflammatory diseases of bone can be divided into three broad but overlapping categories depending largely on the extent on involvement of the bone

1-Osteitis: - this term is used to describe a localized inflammation of bone with no progression through the marrow spaces. Particularly that associated with infected sockets following removal of teeth, (dry socket).

2-Osteomyelitis: - extensive inflammation of the interior of the bone involving, and typically spreading through the marrow spaces.

3-Periostitis: - inflammation of the periosteal spaces of the bone and may not be associated with osteomyelitis.

Osteomyelitis

Osteomyelitis of the jaw was a common complication of dental sepsis before the advent of antibiotics, now it is a rare disease. Various clinical subtypes were recognized, leading to confusion in typing and classification, due to variation in the clinical and pathological features of osteomyelitis being acute, chronic, suppurative or sclerotic, this reflecting the balance between the nature and severity of the irritant, the host defense, local and systemic predisposing factors.

The vast majorities of osteomyelitis cases are caused by bacterial infections and result in an expanding lytic destruction of the involved bone, with suppuration and sequestra formation. This condition (osteomyelitis) may appropriately be termed suppurative osteomyelitis, bacterial osteomyelitis or secondary osteomyelitis. Osteomyelitis may also result from bacteremia.

Another ill defined group of an idiopathic inflammatory disorder of bone that do not responds consistently to antibacterial medications and typically demonstrate sclerosis of bone without suppuration or sequestra formation. This second pattern of inflammatory bone disease is most appropriately termed primary chronic osteomyelitis but may be included under the term of diffuse sclerosing osteomyelitis. Other pattern unique patterns of inflammatory bone diseases include focal sclerosing osteomyelitis, proliferative periostits, and alveolar osteitis.

Suppurative oseomyelitis of the jaw is uncommon in developed countries, but it is a significant difficulty in developing nations. The most common cause is odontogenic infections and jaw fractures. In Africa an important cause is the presence of acute necrotizing gingivitis or NOMA.

Predisposing factors:

1- Chronic systemic diseases, immunocompromised status, and disorders associated with decreased vascularity of bone.
2- Tobacco use, alcohol abuse and intravenous drug abuse.
3- Diabetus mellitus.
4- exanthematous fever and malaria
5- sickle cell anemia
6- malnutrition
7- malignancy
8- collagen vascular disease
9- AIDS
10- Radiation.
11- osteopetrosis, dysosteosclerosis, pagets disease, end-stage cemento-osseous dysplasia, may result in hypovascularized bone that is predisposed to necrosis and inflammation.

Acute suppurative osteomyelitis the condition results when an acute inflammatory process spreads through the medullary spaces of the bone and insufficient time has passed for the body to react to the presence of the inflammatory infiltrate.

Chronic suppurative osteomyelitis: the condition result when the defensive response leads to the production of granulation tissue, which subsequently forms dense scar tissue in an attempt to wall of the infected area. The encircled dead space acts as a reservoir for bacteria, and antibiotics are difficult to reach the site. This pattern begins to evolve about one month after the spread of the initial acute infection and results in a smoldering process that is difficult to manage unless the problem is treated aggressively.

Acute osteomyelitis.

Patients with acute osteomyelitis have signs and symptoms of an acute inflammatory process that has typically been less than 1 month in duration, Fever, leukocytosis, lymphadenopathy, significant sensitivity and soft tissue swelling of the affected area may be present. The radiographs may be unremarkable or may demonstrate an ill-defined radiolucency. On occasion; Paresthesia of the lower lip occur, drainage or exfoliation of fragments of necrotic bone may be discovered. ***A fragment of necrotic bone that has separated from the adjacent vital bone is termed a sequestrum.***

Sequestra often exhibit spontaneous exfoliation, On occasion; ***Fragments of necrotic bone may become surrounded by vital bone and the mass of encased nonvital bone is called an involucrum.***

Chronic osteomyelitis.

If acute osteomyelitis is not resolved expeditiously, the enhancement of chronic osteomyelitis occurs, or the process may arise primarily without a previous acute episode. There may be swelling, pain, sinus formation, purulent discharge, sequestrum formation, tooth loss, or pathologic fracture, Patients may experience acute exacerbation or periods of decreased pain associated with chronic smoldering progression. Radiographs reveal a patchy, ragged and ill-defined radiolucency that often contains

central radiopaque sequestra, occasionally; the surrounding bone may exhibit an increased radiodensity, and the cortical surface can demonstrate significant osteogenic periosteal hyperplasia. Because of an anatomic peculiarity, large portions of each jawbone receive their blood supply through multiple arterial loops originating from a single vessel. Involvement of this single feeder vessel can lead to necrosis of a large portion of the affected bone. Sequestration that has involved an entire quadrant of the jaw has been reported in long-standing cases of chronic osteomyelitis.

Histopathologic Features

Acute osteomyelitis.

Generation of biopsy material from patients with acute osteomyelitis is not common because of the predominantly liquid content and lack of a soft-tissue component. When submitted, the material consists predominantly of necrotic bone. The bone shows a loss of the osteocytes from their lacunae. Peripheral resorption and bacterial colonization. The periphery of the bone and the haversian canals contain necrotic debris and an acute inflammatory infiltrate consisting of polymorphonuclear leukocytes. The submitted material will be diagnosed as a sequestrum unless a good clinicopathologic correlation points to the appropriate diagnosis of acute osteomyelitis.

Chronic osteomyelitis.

Biopsy material from patients with chronic osteomyelitis demonstrates a significant soft issue component that consists of chronically or sub acutely in flamed fibrous connective tissue filling the lntertrabecular areas of the bone. Scattered sequestra and pockets of abscess formation are common.

Treatment and Prognosis

Acute osteomyelitis.

If obvious abscess formation is note, the treatment of acute osteomyelitis consists of antibiotics and drainage. Microbiologic study of the infectious material typically reveals

a polymicrobial infection of organisms normally present in the oral cavity. The antibiotics most frequently selected include penicillin, clindamycin,cephalexin,cefotaxime, tobramycin, and gentamicin. In most patients, a sufficient and appropriate antibiotic regimen aborts the infection and averts the need for surgical intervention. Several investigators have suggested that antibiotic therapy can bring about sterilization of the sequestra; therefore, these non vital bone fragments should be allowed to remain in place as scaffolding for the future development of new bone.

\Chronic osteomyelitis

Chronic osteomyelitis is difficult to manage medically, presumably because pockets of dead bone and organisms are protected from antibiotics by the surrounding wall of fibrous connective tissue. Surgical intervention is mandatory. The antibiotics are similar to those used in the acute form but must be given intravenously in high doses. The extent of the surgical intervention depends on the spread of the process; removal of all infected material down to good bleeding bone is mandatory in all cases. For small lesions, curettage, removal of necrotic bone, and saucerization are sufficient.

In patients with more extensive osteomyelitis decortications or saucerization often is combined with transplantation of cancellous bone chips. In cases of persisting osteomyelitis, resection of the diseased bone followed by immediate reconstruction with an autologous graft is required. Weakened jawbones must be immobilized. The goal of surgery is removal of all infected tissue. Persistence of chronic osteomyelitis is typically due to incomplete removal of diseased tissue. Upon successful elimination of all infected material, resolution is expected. Adjunctive procedures (e.g. hyperbaric oxygen) are rarely necessary if thorough surgical curettage and sequestrectomy have been accomplished. Hyperbaric oxygen is primarily recommended for the rare patient who does not respond to standard therapy or for disease arising in hypovascularized bone (e.g., osteoradionecrosis, osteopetrosis, Paget's disease. cemento-osseous dysplasia).

Focal Sclerosing Osteitis

Etiology

Focal sclerosing osteitis is a relatively common phenomenon that is believed to represent a focal bony reaction to a low-grade inflammatory stimulus. It is usually seen at the apex of a tooth with long-standing pulpitis. This lesion may occasionally be adjacent to a sound, unrestored tooth, suggesting that other etiologic factors such as malocclusion may be operative. Synonyms for focal sclerosing osteitis include focal sclerosing osteomyelitis, bony scar, condensing osteitis, and sclerotic bone. The term focal periapical osteopetrosis has also been used to describe idiopathic lesions associated with normal, caries-free teeth.

Clinical Features

Focal sclerosing osteitis may be found at any age but is typically discovered in young adults. Patients are usually asymptomatic, and most lesions are discovered on routine radiographic examination. A majority are found at the apices of mandibular first molars, with a minority associated with mandibular second molars and premolars. When teeth are extracted, these lesions remain behind indefinitely.

Radiographically, one of several patterns may be seen. The lesion may be uniformly opaque, it may have a peripheral lucency with an opaque center, it may have an opaque periphery with a lucent center, or it may be composed of confluent or lobulated opaque masses.

Histopathology

Microscopically, these lesions are masses of dense sclerotic bone; Connective tissue is scant, as are inflammatory cells.

Differential Diagnosis

Differential diagnosis should include periapical cemental dysplasia, osteoma, complex odontoma, cementoblastoma, osteoblastoma, and hypercementosis. In most cases,

however, diagnosis can be made with confidence on the basis of historical and radiographic features.

Treatment

Because it is believed to represent a physiologic bone reaction to a known stimulus, the lesion itself need not be removed. A biopsy might be contemplated to rule out more significant lesions that received serious consideration in the differential diagnosis. The inflamed pulp that stimulated the focal sclerosing osteomyelitis should be treated. The decision about whether the tooth should be restored, treated endodontically, or extracted should be made on a case-by-case basis according to findings.

DIFFUSE SClEROSING OSTEOMYELITIS

Diffuse sclerosing osteomyelitis is an ill-defined, highly controversial, evolving area of dental medicine. This diagnosis encompasses a group of presentations that are characterized by pain, inflammation, and varying degrees of gnathic periosteal hyperplasia, sclerosis, and lucency. On occasion, diffuse sclerosing osteomyelitis can be confused with secondarily inflamed intraosseous pathoses (florid cementosseous dysplasia) or Paget's disease of bone. In spite of the clinical and radiographic similarities, these processes can be separated from diffuse sclerosing osteomyelitis because of various clinical, radiographic and histopatholog differences the remaining pathoses can be grouped under three major categories:

1-Diffuse sclerosing oseomyelitis

2-Primary chronic osteomylitis

3-Chronic tendoperiostitis

Etiology

Diffuse sclerosing osteomyelitis represents an inflammatory reaction in the mandible or maxilla, believed to be in response to a microorganism of low virulence. Bacteria are generally suspected as causative agents, although they are seldom specifically identified.

Chronic periodontal disease, which appears to provide a portal of entry for bacteria, is important in the etiology and progression of diffuse sclerosing osteomyelitis. Carious non vital teeth are less often implicated.

Clinical Features

This condition may be seen in any age, in either sex, and in any race, but it tends to occur most often in middle-aged black women. The disease is typified by a protracted chronic course with acute exacerbations of pain, swelling, and occasionally drainage.

Radiographically:

This process is diffuse, typically affecting a large part of the jaw. The lesion is ill defined. Early lucent zones may appear in association with sclerotic masses. In advanced stages, sclerosis dominates the radiographic picture. Periosteal thickening may also be seen. Scintigraphy may be particularly useful in evaluating the extent of this condition.

Histopathology

The microscopic changes of this condition are inflammatory, Fibrous replacement of marrow is noted; a chronic inflammatory cell infiltrate and occasionally a neutrophilic infiltrate are also seen. Bony trabeculae exhibit irregular size and shape and may be lined by numerous osteoblasts, Focal osteoclastic activity is also present. The characteristic sclerotic masses are composed of dense bone, often exhibiting numerous reversal lines.

Differential Diagnosis Chronic sclerosing osteomyelitis shares many clinical, radiographic, and histological features with florid osseous dysplasia. The two should be separated, because the former is an inflammatory/infectious process and the latter a bony dysplastic process. Treatment and prognosis are therefore dissimilar. Florid osseous dysplasia appears to be an extensive form of periapical cemental dysplasia and, unlike

diffuse sclerosing osteomyelitis, may exhibit anterior periapical lesions and traumatic or simple bone cysts. Furthermore, florid osseous dysplasia is usually asymptomatic and appears as a fibroosseous lesion lacking an inflammatory cell infiltrate.

Treatment

The management of diffuse sclerosing osteomyelitis is problematic because of the relative avascular nature of the affected tissue and because of the large size of the lesion. Even with aggressive treatment, the course is protracted. If an etiologic factor such as periodontal disease or a carious tooth can be identified, it should be eliminated. Antibiotics are the mainstay of treatment and are especially helpful during painful exacerbations. Surgical removal of the diseased area is usually an inappropriate procedure because of the extent of the disease. However, decortication of the affected site has resulted in improvement in some cases. Low-dose corticosteroids have also been used with some success. Hyperbaric oxygen therapy may prove to be a valuable adjunct. Recently, treatment with pamidronate has shown promising results.

Chronic Osteomyelitis with Proliferative Periostitis: (Garré's Osteomyelitis)

Etiology

Chronic osteomyelitis with proliferative periostitis, commonly known as Garré's osteomyelitis, is essentially a subtype of osteomyelitis that has a prominent periosteal inflammatory reaction as an additional component. It most often results from a periapical abscess of a mandibular molar tooth or an infection associated with tooth extraction or partially erupted molars, It is most common in children.

- The eponym Garré's osteomyelitis has been applied to this condition after the author, Dr. K. Garrés, who in1893 described the clinical features of 72 patients with osteomyelitis. The disease he described was most common in the femur, with

only three cases occurring in the jaws. In the absence of histological and radiographic findings, which were unavailable at the time of the report, it is likely that Garrés was describing a form of recalcitrant, acute osteomyelitis that occurred in both adults and children. It was not chronic osteomyelitis with proliferative periostitis. Therefore, the term Garré's osteomyelitis, although widely used in reference to this condition, is inaccurate.

Clinical Features

This variety of osteomyelitis is uncommonly encountered. It has been described in the tibia, and in the head and neck area, it is seen in the mandible. It typically involves the posterior mandible and is usually unilateral. Patients characteristically present with an asymptomatic bony, hard swelling with normal appearing overlying skin and mucosa. On occasion, slight tenderness may be noted.

This presentation necessitates the differentiation of this process from benign mandibular neoplasms. Radiographs and a biopsy provide a definitive diagnosis.

Radiographically, the lesion appears centrally as a mottled, predominantly lucent lesion in a pattern consistent with that of chronic osteomyelitis. The feature that provides the distinctive difference is the periosteal reaction. This, best viewed on an occlusal radiograph, appears as an expanded cortex, often with concentric or parallel opaque layers. Trabeculae perpendicular to the onion skin layers may also be apparent.

Histopathology

Reactive new bone typifies the subperiosteal cortical response. Perpendicular orientation of new trabeculae to redundant cortical bone is best seen under low magnification. Osteoblasts dominate in this area, and both osteoblasts and osteoclasts are seen centrally. Marrow spaces contain fibrous tissue with scattered lymphocytes and plasma cells.

Inflammatory cells are often surprisingly scant, making microscopic differentiation from fibroosseous lesions a diagnostic challenge.

Treatment : Identification and removal of the offending agent are of primary importance in chronic osteomyelitis with proliferative periostitis. Removal of the involved tooth is usually required. Antibiotics are generally included early in this treatment. The mandible then undergoes gradual remodeling without additional surgical intervention.

Osteoradionecrosis

Osteoradionecrosis is one of the most serious complications of radiation to the head and neck but is seen less frequently today because of better treatment modalities and prevention. The current prevalence rate is less than 4%, whereas the frequency approached 15% less than 20 years ago. Although the risk is low, it increases dramatically if a local surgical procedure is performed within 21 days of therapy initiation or between 4 and 12 months after therapy. Radiation of bone results in permanent damage:' to the osteocytes and microvasculature system. The altered bone becomes hypoxic, hypovascular, and hypocellular. Osteoradionecrosis is the result of nonhealing, dead bone; infection is not necessarily present.

Bisphosphonate-Associated Osteonecrosis

A similar type of jaw necrosis may be seen as a complication of bisphosphonate therapy (e.g., pamidronate, zoledronic acid). Bisphosphonates are currently used as part of the treatment regimen for patients with multiple myeloma, metastatic cancers to bone (e.g., breast or prostate cancer), Paget's disease, and osteoporosis because of their inhibitory effect on osteoclastic bone resorption.

Bisphosphonates, taken for an extended period (greater than 1 year), but the patient at risk for non infectious jaw necrosis.

The typical presenting clinical symptom of bisphosphonate-associated osteonecrosis is pain, and the characteristic sign is bone exposure. The lesion usually follows tooth extraction or other form of jaw surgery, although many cases seem to be spontaneous. As with osteoradionecrosis, the mandible is more commonly affected than the maxilla.

Alveolar osteitis

(drysocket; fibrinolytic alveolitis)

After extraction of a tooth, a blood clot is formed at the site, with eventual organization of the clot by granulation tissue, gradual replacement by coarse fibrillar bone, and, finally, replacement by mature bone. Destruction of the initial clot prevents appropriate healing and causes clinical syndrome known as alveolar osteitis . Extensive investigations have shown that the clot is lost secondary to transformation of plasminogen to plasmin, with subsequent lysis of fibrin and formation of kinins (fibrinolytic alveolitis): these are potent pain mediators. Local trauma, estrogens, and bacterial pyrogens are known to stimulate fibrinolysins. This knowledge correlates well with the increased frequency of alveolar osteitis in association with inexperienced surgeons, traumatic extractions, oral contraceptive use and presurgical infections. In addition, inadequate irrigation at surgery and the use of tobacco products have been related to the development of the problem.

Clinical Features

The frequency of alveolar osteitis is higher in the mandible and the posterior areas. After oral contraceptive use is taken into account. They do not appear to be a significant sex predilection. The prevalence is between 1% and 3% of all extractions, but it increases to 25% to *30%* for impacted mandibular third molars. The frequency appears to be

decreased when impacted teeth are prophylactically removed rather than for therapeutic reasons after development of chronic inflammation of pericoronal tissues.

The overall prevalence is highest between 20 and 40 years of age (when the majority of teeth are extracted. although the likelihood of developing alveolar osteitis appears greatest for extractions in the *40*- to 45-year-old age group The affected extraction site is filled initially with a dirty gray clot that is lost and leaves a bare bony socket (dry socket). The detection of the bare socket may be hindered by partial retention of the clot or by overlying inflamed tissue that covers the site. The diagnosis is confirmed by probing of the socket, which reveals exposed and extremely sensitive bone. Typically, severe pain, foul odor, and (less frequently) swelling and lymphadenopathy develop 3 to 4 days after ext reaction of the tooth. The signs and symptoms may last from 10 to 40 days.

Treatment and Prognosis

On evaluation of the patient complaining of postextraction pain, a radiograph should be taken of the affected area to rule out the possibility of a retained root tip or a foreign body. All sutures should be removed. The socket is irrigated with warm saline, followed by thorough clinical inspection of the socket for any unexpected pathosis. Curettage of the socket is not recommended, because this typically increases the associated pain. Potent oral analgesics should be prescribed, and the patient should be given a plastic syringe with instructions to keep the socket clean via home irrigation with a chlorhexidine or saline solution. This irrigation should continue until debris no longer collects within the healing socket (usually 3 to 4 weeks).