**Oral Pathology**

Lecture 5 Bone Pathology

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**Inflammatory Diseases of Bone**

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

The term 'osteitis' is used to describe a localized inflammation of bone with no progression through the marrow spaces, particularly which associated with infected sockets following removal of teeth (dry socket).

Osteomyelitis is a more extensive inflammation of the interior of the bone involving, and typically spreading through, the marrow spaces.

Periostitis means inflammation of the periosteal surface of the bone and may or may not be associated with osteomyelitis.

**OSTEOMYELITIS**

Osteomyelitis is an acute or chronic inflammatory process within the medullary spaces (trabecular) bone that involves the marrow spaces or cortical surfaces of bone that extends away from the initial site of involvement.

The vast majority of osteomyelitis cases are caused by bacterial infections and results in an expanding lytic destruction of the involved bone, with suppuration and sequestra formation before the advent of antibiotics; it is now a rare disease.

**Factors predisposing to osteomyelitis of the jaws:**

*Local f actors Systemic factors*

-Decreased vascularity/vitality of bone -Impaired host defense

- Trauma - Immunosuppression

- Radiarion injury - Diabetes mellitus

- Paget's disease - Malnutrition

- Osteopetrosis - Extreme of age

- Major vessel disease

**ACUTE OSTEOMYELITIS**: A rapidly destructive inflammatory process within bone that consists of granulation tissue, purulent exudate, and islands of nonvital bone (sequestra).

**Clinical and Radiographic Features**

Patients of all ages can be affected. There is a strong male predominance. Most cases involve the mandible due to its relatively poor vascular supply and dense cortical bone that is more susceptible to infection when compared to the maxilla. Fever, leukocytosis, lymphadenopathy and soft tissue swelling of the affected area may be present.

Radiographic examination may be normal in the early stages of the disease, but after 10-14 days sufficient bone resorption may have occurred to produce irregular, moth-eaten areas of radiolucency. Plain dental or panoramic radiographs may demonstrate an ill-defined radiolucency. Paresthesia of the lower lip, 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.

A fragment of necrotic bone may become surrounded by new vital bone, and the dead bone in this situation is known as an **involucrum**.

**Histopathologic Features**

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.

**Treatment**

1) Resolve the source of infection, 2) Establish drainage, 3) Removal of obviously infected bone; the sequestrum may be spontaneously exfoliated through a sinus or have to be surgically removed before healing can take place, and 4) Obtain bacteriologic samples for culture and antibiotic sensitivity testing.

**Chronic Suppurative Osteomyelitis**

Chronic differs greatly from the acute types by inducing bone to form and become denser. It occurs in response to a low-grade inflammatory process rather than the intense and destructive inflammation caused by virulent bacteria.

If acute osteomyelitis is not resolved the entrenchment of chronic osteomyelitis occurs, or the process may arise primarily without a previous acute episode.

**Clinical and Radiographic Features**

Swelling, pain, sinus formation, purulent discharge, sequestrum formation, tooth loss, or pathologic fracture may occur. Radiographs reveal a patchy, ragged, and ill-defined radiolucency that may contain central radiopaque sequestra and be intermixed with zones of radiodensity.

**Histopathologic Features**

Biopsy material from patient with chronic osteomyelitis demonstrates a significant soft tissue component that consists of chronically or subacutely inflamed fibrous connective tissue filling the intertrabecular areas of the bone. Scattered sequestra and pockets of abscess formation are common.

**Treatment:** Chronic osteomyelitis is difficult to manage medically because

pockets of dead bone and organisms are protected from antibiotic drugs by the surrounding wall of fibrous connective tissue. Surgical intervention is mandatory and the extent depends on the spread of the process; removal of all infected material down to good bleeding bone is mandatory in all cases. The antibiotic medications must be given IV in high doses.

**Focal Sclerosing Osteomyelitis (CONDENSING OSTEITIS)**

Condensing osteitis is termed to localized areas of bone sclerosis associated with the apices of tooth with pulpitis or pulpal necrosis.

**Etiology:** Osteosclerosis is one of the sequelae of periapical inflammation and may result from low grade irritation and / or high tissue resistance; represent a physiologic bone reaction to a known stimulus.

**Clinical and Radiographic Features**

This secondary sclerosis of bone is seen most frequently in children and young adults but also can occur in older adults. It is usually asymptomatic. Most cases occur in the premolar and molar areas of the mandible; most commonly the first permanent molar, that demonstrates pulpitis or necrosis, and may remain as a sclerotic area of bone following extraction.

The classic alteration consists of a localized, usually uniform zone of increased radiodensity adjacent to the apex of a tooth that exhibits an apical inflammatory lesion.

**Histopathogic features:** A localized increase in the number and thickness of the bone trabeculae is seen and there may be scattered lymphocytes and plasma cells in the surrounding scanty fibrosed marrow.

**Differential Diagnosis** includes focal cemental dysplasia, osteoblastoma,

idiopathic osteosclerosis, cementoblastoma, complex odontoma, and osteoma.

**Treatment and Prognosis:** After extraction or appropriate endodontic therapy of the involved tooth, approximately 85% of cases of condensing osteitis will regress, either partially or totally.

**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.

Included in this category are three different pathoses: 1. Diffuse sclerosing osteomyelitis

2. Primary chronic osteomyelitis 3. Chronic tendoperiostitis

**Etiology:** Diffuse sclerosing osteomyelitis term should be used only when an obvious infectious process of a low virulence microorganism directly is responsible for sclerosis of bone. In these cases, a chronic intraosseous bacterial infection creates a mass of chronically inflamed granulation tissue that incites sclerosis of the surrounding bone.

**Clinical and Radiographic Features**

Diffuse sclerosing osteomyelitis arises almost exclusively in adulthood, does not exhibit sex predominance, and primarily occurs in the mandible.

An increased radiodensity develops around sites of chronic infection (e.g., periodontitis, pericoronitis, and apical inflammatory disease) in a manner very similar to the increased radiodensity that may be seen surrounding areas of chronic suppurative osteomyelitis.

**Histopathologic Features**

Diffuse sclerosing osteomyelitis demonstrates sclerosis and remodeling of bone. The haversian canals are scattered widely and little marrow tissue can be found. 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 treatment and prognosis are dissimilar due to etiology of the former is an inflammatory/infectious process and the latter a bony dysplastic process.

**Treatment and Prognosis**

Diffuse sclerosing osteomyelitis is treated best through resolution of the adjacent foci of chronic infection. After resolution of the infection, the sclerosis remodels in some patients but remains in others. The persistent sclerotic bone is avascular, does not exhibit typical remodeling, and is very sensitive to inflammation.

**GARRÉ OSTEOMYELITIS**

Garré osteomyelitis is an unusual hyperplastic reaction of the periosteum to a chronic osteomyelitis of the posterior mandible that is unique to young patients.

**Clinical and Radiographic Features**

Garré osteomyelitis is most frequently associated with advanced acute caries in young patients that has progressed to pulpitis and a periapical lesion.

They slowly exhibit a diffuse or focal enlargement of an area of the mandible, usually posteriorly. The other common cause of this unique process is a molar that is unable to fully erupt. The area is usually asymptomatic.

A radiograph taken using an occlusal projection will demonstrate the characteristic multiple thin layers of new bone, referred to as an “onion skin” appearance.

**Histopathology**

To become a Garré osteomyelitis the inflammatory response must extend through the bone to the outer surface, stimulating the periosteum to thicken and lay down excess layers of new bone.

The microscopic feature of the reactive bone that forms in response to the stimulated periosteum is less dense than normal cortical bone and deposited in a layered pattern. The trabecular spaces are wide and occupied with a cellular connective tissue.

**Treatment**

The condition slowly reverts to normal after the source of infection is identified and resolved. Sometimes extraction of the offending tooth or surgical recontouring of the tissue in the molar area is necessary.

**Alveolar Osteitis (Dry Socket; Fibrinolytic Alveolitis)**

After extraction of a tooth, premature fibrinolysis of the initial clot is to be responsible for the clinical condition known as alveolar osteitis.

Factors deemed to be associated with an increased prevalence include:

Preoperative infection, Difficult extraction, Tobacco use, Inexperienced surgeons, Surgical flap design,

Use of a local anesthetic with vasoconstrictor, Inadequate postoperative irrigation, Oral contraceptive use. The clot is lost secondary to transformation of plasminogen to plasmin, with subsequent lysis of fibrin and formation of kinins; these are potent pain mediators.

**Clinical Features**

The frequency of alveolar osteitis is higher in the mandible and the posterior areas. The frequency appears to be decreased when impacted teeth are removed prophylactically rather than for pericoronitis. The affected extraction site is filled initially with a dirty gray clot that is lost and leaves a bare bony socket (dry socket).

The diagnosis is confirmed by probing of the socket, which reveals exposed and extremely sensitive bone, a radiograph should be taken of the affected area to rule out the possibility of a retained root tip or a foreign body. Typically, severe pain, foul odor, and (less frequently) swelling and lymphadenopathy develop 3 to 4 days after extraction of the tooth. The signs and symptoms may last from 10 to 40 days.

**Radiation injury and osteoradionecrosis**

Osteoradionecrosis is primarily a problem of hypoxia, hypocellularity, and hypovascularity in which the presence of bacteria represents a secondary colonization of non-healing bone rather than a primary bacterial infection.

In addition, medication-related osteonecrosis represents unique and appears more strongly related to altered bone metabolism.

The non-vital bone which results from the reduction in blood supply is sterile and asymptomatic but is very susceptible to infection and to trauma. Infection may spread rapidly through the irradiated bone, resulting in extensive osteomyelitis and painful necrosis of the bone. Modern methods of radiotherapy and better treatment modalities and prevention have greatly reduced the incidence of this condition.

**Bisphosphonate-Associated Osteonecrosis**

Bisphosphonates inhibit bone resorption by suppressing osteoclast activity and frequently used to lessen bone destruction by metastases to bone from multiple myeloma, breast and prostate carcinoma.

Treatment with high – potency bisphosphonates, especially when administered long term and high dose intravenously can lead to osteonecrosis. Osteonecrosis affects primarily the jaws, usually mandible.

Typical presenting lesions are either non – healing extraction socket or exposed bone; they are refractory to conservative treatment and antibiotic therapy.

Discontinuation of the bisphosphonates does not appear to help because their effects last at least a year after administration and may be permanent.