IV - FIBRO-OSSEOUS LESIONS:

- **Non-neoplastic lesions:**
  1. Fibrous dysplasia
  2. Cemento-osseous dysplasia

- **Neoplasms:**
  1. Ossifying fibroma
FIBRO – OSSEOUS LESIONS

Definition:
a diverse group of lesions characterized by replacement of normal bone by a fibrous tissue containing a newly formed, mineralized product.

These lesions include
1. Developmental
2. Reactive
3. Neoplastic lesions.
Classification:

(i) Non-neoplastic lesions:
   a. Fibrous dysplasia
   b. Cemento-osseous dysplasia
      1. Periapical cement-osseous dysplasia
      2. Focal cemento-osseous dysplasia
      3. Florid cemento-osseous dysplasia

(ii) Neoplastic:
   1. Ossifying fibroma
   2. Juvenile ossifying fibroma
FIBROUS DYSPLASIA

**Definition:**

a disease of bone maturation and remodeling in which the normal medullary bone and cortices are replaced by a disorganized fibrous woven bone.

The resultant fibro-osseous bone is more

1. **elastic**
2. **structurally weaker** than original bone.
Fibrous Dysplasia

- Monostotic Fibrous Dysplasia
- Polyostotic Fibrous Dysplasia
Etiology:

- Mutation in GNAS1 (a somatic mutation)

- It is caused by the deletion of a bone maturation protein during embryogenesis.

- There is no evidence to suggest a hereditary influence.
Prenatal life

Early stages embryologic life

Later stages of embryonic life

Undifferentiated stem cell
Zygote → Embryo → Fetus
Polyostotic FD

McCune-Albright syndrome
1. Multiple bone involved
2. Multiple cutaneous pigmentations
3. Hyperfunction of one or more endocrine glands

Jaffe type
1. with almost entire skeleton involved.
2. Cutaneous pigmentation
Post natal life

limited to one single bone.

III. Monostotic Fibrous dysplasia
Pathogenesis:

- At certain times in the histo-differentiation phase of the embryo, a genetic mutation or deletion occurs in the gene that encodes for an intra-cytoplasmic transducer protein required for bone maturation.

- If the mutation occurs in one of the undifferentiated stem cells during early embryologic life, the osteoblasts, melanocytes, and endocrine cells that represent the progeny of that mutated cell all will carry that mutation and express the mutated gene.

- The clinical presentation will be appear as multiple bone lesions, cutaneous pigmentation and endocrine disturbances would result.
Skeletal progenitor cells at later stages of embryonic development are assumed to migrate and differentiate as part of the process of normal skeletal formation.

If the mutation occurs during this later period, the progeny of the mutated cell will disperse and participate in the formation of the skeleton resulting in multiple bone lesions of fibrous dysplasia.

If the mutation occurs during postnatal life; the progeny of that mutated cell are essentially confined to one site, resulting in fibrous dysplasia affecting a single bone.
Clinical features:
- Age: 1st or 2nd decade of life.
- Sex: equal Site predilection
- Maxilla involved more than mandible.
- Mostly unilateral.
- Maxillary lesions often involve adjacent bones like zygoma, sphenoid (called Craniofacial FD).
Fibrous Dysplasia

**bones affected**

- **f** - frontal
- **m** - maxilla
- **md** - mandible
- **t** - temporal
- **s** - sphenoid
- **z** - zygomatic

**Craniofacial**
(multiple facial bones)

**Monostotic**
(single bone)
Signs & symptoms:

Affected bone:

- painless,

- gradually enlarging swelling.

- Teeth within affected jaws
  1. Remain firm (No mobility)
  2. But may be displaced by the mass.
  3. Reduced thickness of periodontal membrane space
Differential diagnosis:

Clinically:
1. Ossifying fibroma
2. Paget’s disease.

Radiographic appearance:
1. Hyperparathyroidism.
2. Paget’s disease (early stage).
Polyostotic fibrous dysplasia

Clinical features:

- **Age**: 1st decade of life or earlier.
- **Sex**: equal.
- **Site**: Can affect any bone in skeleton, but primarily the skull bones and long bones of skeleton.
Signs & symptoms:

**McCune-Albright (syndrome)**

(a) Polyostotic fibrous dysplasia
(b) Café au lait pigmentation
(c) Multiple endocrinopathies,
   - sexual precocity.
   - pituitary adenoma.
   - hyperthyroidism.

**Jaffe-Lichtenstein syndrome**

(a) Involvement of two or more bones is termed polyostotic fibrous dysplasia.
(b) Café au lait (coffee with milk) pigmentation
Café au lait pigmentation

1. Well defend
2. Unilateral
3. Irregular border
Radiographic features

- **Early lesions**
  are patchy radiolucencies but develop into weak radiopacities, with a ground glass or fine orange-peel texture.

- **Later on:** the lesion mineralizes. The degree of radiopacity increases, and late lesions are sclerotic and lack the trabecular pattern of normal bone.

- A fingerprint pattern of coarse trabeculae may be seen in very old lesions.

- The cortex and lamina dura are affected by the process, and their definition is lost radiographically. The cortex is expanded but thin.

- Lesions ill defined and blend into adjacent bone – limits of lesion cannot be defined.
Fibrous dysplasia affecting the left mandible with a patchy radiolucency. No lesion border can be identified.
Mixed radiolucent and radiopaque

Fig. 14.1 Fibrous dysplasia. This example is heavily calcified but shows the lack of definable margins of the lesion.
This well-established lesion with a rounded swelling merges imperceptibly with normal bone surrounding the canine. There is loss of lamina dura and cortex in the affected area, and the fine trabecular pattern produces a thumb-print appearance.
Ground glass appearance with sclerotic areas and ill-defined border
III- defined border
Histological features:

- In fibrous dysplasia, normal bone is replaced by a generally loose, cellular fibrous connective tissue.

- Lesion shows typical
  1. Irregular,
  2. Chinese character shaped
  3. Not connected to each other
  4. Trabeculae of immature woven bone.
  5. Not bordered by osteoblasts. These trabeculae believed to arise due to metaplasia
The surrounding stroma is highly cellular and vascular.

The lesion has no definable borders, and the osseous trabeculae blend into the normal surrounding bone.

Over time, fibrous dysplasia of the jaws may show maturation, which is characterized by formation of lamellar bone and parallel arrangement of the trabeculae.
PERIAPICAL GRANULOMA
(CHRONIC APICAL PERIODONTITIS)

**Definition:**
It refers to a mass of chronically inflamed granulation tissue at the apex of a non-vital tooth.
Formation of apical inflammatory lesions represents a defensive reaction secondary to the presence of bacteria in the root canal.
Etiology:

Periapical granulomas may arise:

1. Develop as the initial periapical pathosis.
2. After a periapical abscess
These lesion is not static:

1) Periapical cysts
2) Show acute exacerbations with abscess formation
Clinical Features

- Periapical granulomas represent approximately 75% of apical inflammatory lesions and of those that have failed to respond to conservative endodontic measures.

- The tooth is slightly sensitive to percussion.

- Asymptomatic but pain and sensitivity can develop if acute exacerbation occurs.

- The involved tooth is not vital.
Radiographic features

- Most lesions are discovered on routine radiographic examination.
- A radiolucency of variable size is present.
- The affected tooth shows loss of the apical lamina Dura.
- The lesion may be well or ill circumscribed.
- The size is variable ranging from small barely perceptible lesions to lucencies exceeding 2 cm.
- Root resorption may be seen.
Early stage of Lesion

Late stage of lesion
Histopathologic Features

- Periapical granuloma consists of inflamed granulation tissue surrounded by a fibrous connective tissue capsule firmly attached to the infected root.

- Dense lymphocytic infiltrate that is intermixed frequently with:
  1. Neutrophils,
  2. Plasma cells.
  3. Histocytes, and
  4. less frequently mast cells and eosinophils
If plasma cells are present:
1. Scattered eosinophilic globules of gamma globulin (Russell bodies) and
2. Basophilic particles (pyronine bodies)

Both of these are plasma cell products are not specific for the periapical granuloma and may be found within any accumulation of plasma cells.

Epithelial rests of Malassez may be identified within the granulation tissue its growth is responsible for cyst formation.
Collections of cholesterol clefts in microscope formerly were crystals which dissolved in xylol.

Macrophages engulf cholesterol and change to foam cells

Macrophages may fuse together or nuclei divide to form multinucleated giant cells.
Ring of proliferating epithelium

Degeneration in the center of epithelium forming microcyst
• Granulation tissue contains lymphocyte intermixed with neutrophil, plasma cells and mast cell
• Number of young capillaries surrounded by a mixed inflammatory reaction
Early stage of periapical granuloma with predominantly foamy macrophages (a) and lymphocytes (b)
Periapical abscess

**Definition:**

It is the accumulation of acute inflammatory cells sat the apex of a non-vital tooth.

A periapical abscess is a collection of pus at the root of a tooth, usually caused by an infection that has spread from a tooth to the surrounding tissues.

The body attacks an infection with large numbers of white blood cells.

Pus is the accumulation of these white blood cells, dead tissue, and bacteria.
Etiology:

Acute inflammatory lesions with abscess formation may arise as:

1. Initial periapical pathosis
2. An acute exacerbation of a chronic periapical inflammatory lesion (phoenix abscess).
Frequently, the source of the infection is **Pulpal death** is always observed clinically except in case of **trauma** where the tooth may not contain a cavity or a restoration.

In the earliest stage, the periapical periodontal ligament fibers may exhibit acute inflammation but **no frank abscess formation**.

This localized alteration; best termed **(ACUTE APICAL PERIODONTITIS)** which is a process often occurs in association with a non-vital tooth.
Acute apical periodontitis may present in vital teeth secondary to:

1) Trauma high occlusal contacts or
2) Wedging by a foreign object

The clinical presentation often closely resembles that of a periapical abscess and must be considered in the differential diagnosis.
Clinical Features

- Many investigators wrongly subdivide periapical abscesses into acute and chronic types because both types represent acute inflammatory reactions.

- Periapical abscesses should be designated as symptomatic or asymptomatic on the basis of their clinical presentations.

- Periapical abscesses become symptomatic as the purulent material accumulates within the alveolus:
  1. Where pain becomes more intense
  2. Extreme sensitivity to percussion or
  3. Even touch with throbbing pain.
Patients with periapical abscesses typically have severe pain in the area of the non-vital tooth because of:

1. pressure and
2. the effects of inflammatory chemical mediators on nerve tissue.

The involved tooth is unresponsive to electric and thermal tests because of pulp necrosis.

The exudate and neutrophilic infiltrate of an abscess cause pressure on the surrounding tissue, often resulting in slight extrusion of the tooth from its socket.
Pus seeks the path of least resistance and spreads into contiguous structures.

The purulence may extend resulting in complications:

1) Pass through the medullary spaces away from the apical area causing osteomyelitis
2) Perforate the cortex and spread diffusely through the overlying soft tissue of the face in case of abscess from long roots causing facial cellulitis.
3) Canalize through the overlying soft tissue. The cortical plate may be perforated into the oral cavity. Creating a sessile swelling mass
4) Or perforate through the surface epithelium and drain through an intraoral sinus known as a parulis or (gum boil)
Ludwig’s angina
Facial cellulitis arising from an infected upper tooth. The tissues are red, tense and shiny, and the patient is incapacitated by the systemic effects of infection.
Phoenix abscess
Radiographic Features

Because of the rapidity with which this lesion develops, there is generally insufficient time for significant amounts of bone resorption to occur.

Therefore, radiographic changes are slight and are usually limited to mild radiographic thickening of the apical periodontal membrane space.

If a periapical abscess develops as a result of acute exacerbation of a chronic periapical granuloma, a radiolucent lesion is evident.
Periapical radiolucency

- **Apical granuloma**: < 1.5 cm corticated margin
- **Radicular cyst**: > 1.5 cm corticated margin
- **Abscess**: less well defined margin
Histopathology

Microscopically, a periapical abscess appears as a zone of liquefaction composed of proteinaceous exudate, necrotic tissue, and viable and dead neutrophils (pus).

Adjacent tissue containing dilated vessels and a neutrophilic (polymorphonuclear leukocytes) infiltrate surrounds the area of liquefaction necrosis.
Treatment:

- Early stages (acute apical periodontitis) could be treated by removal of the initiating cause such as premature contact.

- Acute periapical abscess is treated by:
  1. Extraction of the tooth
  2. or drainage of the pus through the root canal followed by endodontic treatment if the tooth is to be preserved

- The use of Antibiotic is conservative.
Osteomyelitis
Definition of Osteomyelitis (OM.)

Inflammation of bone and the bone marrow.
Etiology

1. An infecting organisms
   i. Bacteria, Viruses, Parasites and Fungi.
   ii. The commonest anerobic bacteria: bacteroids

2. Trauma

3. Vascular insufficiency
Classification of Osteomyelitis

Specific
1) Actinomycosis OM.
2) Syphilitic OM.
3) Tuberculosis OM.

Non-specific
1) Suppurative
2) Non-suppurative

Osteoradionecrosis
Non-specific

Suppurative OM.
1. Acute suppurative OM.
2. Chronic suppurative OM.

Non-suppurative OM.
1. Chronic Osteomyelitis with Proliferative Periostitis (Garré's Osteomyelitis)
2. Diffuse Sclerosing Osteomyelitis
3. Focal Sclerosing Osteomyelitis
Immune System

Virulence of the irritant
Acute Suppurative OM.

**Definition:**

Acute inflammation of the bone and bone marrow of the mandible and maxilla
Predisposing factors:
1. Extension of periapical abscess.
2. Physical injury such as fracture or surgery.
4. Presence of acute necrotizing ulcerative gingivitis (ANUG) or Noma in developing countries.
5. People with Chronic systemic diseases
6. Disorders associated with decreased vascularity of bone
7. Tobacco and alcohol abuse.
8. Intravenous drug abuse.
10. Anemia
11. Malnutrition
12. Malignancy
13. acquired immunodeficiency syndrome (AIDS)
14. Bone diseases such as osteopetrosis and Paget's disease.
Clinical Features
Signs and symptoms of acute infection:

1. Pain is the primary feature of this inflammatory process + Painful lymphadenopathy
2. Swelling due to inflammatory edema
3. Pyrexia
4. Redness
5. Lose of function
• Gingiva appears red, swollen & tender
• Associated teeth are:
  1. Tender.
  2. They may become loose, and
  3. pus may exude from an open socket or gingival margins.
Early complaints are severe, throbbing, deep-seated pain and swelling with external swelling due to inflammatory edema.

Later, distension of the periosteum with pus

Finally, subperiosteal bone formation cause the swelling to become firm.

Laboratory: Leukocytosis
Lose of function:

1. Muscle edema causes difficulty:
   - Trismus (difficulty opening mouth)
   - Dysphagia (difficulty swallowing)

2. Anesthesia or paresthesia (changes or decreased sensation) in (anterior chin, lower lip) caused by pressure on the inferior dental nerve and mental nerve distribution
Osteomyelitis of the newborn is a distinctive variant affecting the maxilla shortly after birth and is potentially fatal.

The cause is either birth injuries or uncontrolled middle ear infection.

Other than in children, the maxilla is very rarely affected.
Pathology

- Acute osteomyelitis is a suppurative infection with a mixed bacterial, much of which forms a biofilm on sequestra of bone.

- Oral bacteria, particularly anaerobes such as Bacteroides, Porphyromonas or Prevotella species, are important causes.

- Staphylococci may be responsible when osteomyelitis follows an open fracture and the bacteria enter from the skin.

- Infection and acute inflammation cannot escape, and the pressure spreads infection through the florae marrow spaces. It also compresses blood vessels confined within the rigid boundaries of the vascular canals.
Thrombosis and obstruction then lead to further bone necrosis.

Dead bone is recognizable microscopically by lacunae empty of osteocytes and medullary spaces filled with neutrophils and colonies of bacteria that proliferate in the dead tissue.

Pus, formed by liquefaction of necrotic soft tissue and inflammatory cells, is forced along the medulla and eventually penetrates the cortex to reach the subperiosteal region by resorption of bone.
At the boundaries between infected and healthy tissue, macrophage cells resorb the periphery of the dead bone, which eventually becomes separated as a sequestrum.

Once infection starts to localize, new bone forms around it, particularly subperiosteally.

Where bone has died and been removed or shed as sequestrum, healing is by granulation tissue with formation of woven bone in the proliferating connective tissue.

After resolution, woven bone is gradually replaced by compact bone and remodeled to restore normal morphology.
Osteomyelitis

Inflammation

Suppurative Osteomyelitis

Edema & pus in marrow/medullary cavity

Obstructs blood flow

"Necrosis of Bone"
A fragment of necrotic bone that has separated from the adjacent vital bone is termed a sequestrum.

- It occurs in late stages of acute infection.
- Sequestra often exhibit spontaneous exfoliation.

Fragments of necrotic bone may become surrounded by vital bone and the mass of encased non vital bone is called an involucrum.
A: healthy bone
B: periosteum
C: involucrum
D: sinus tract
E: sequestrum
More in mandible than maxilla due to:

1. Compact nature of mandibular bone
2. Single blood supply via mandibular canal
1. The trabecular bone mandible is more coarse than the maxilla.
2. The cortical bone is thick and dense.
3. The mandible is a force-absorbing element.

1. The dentate maxilla has a finer trabecular pattern compared with the mandible.
2. The cortical bone is more thin and porous.
3. The maxilla is a force distribution unit and is designed to protect the orbit and brain.
1. An early osteomyelitis may show a normal radiographic appearance. Do not appear until after at least 10 days.

2. If the infection continues or progresses:
   - there is loss of trabecular pattern and
   - areas of radiolucency indicating bone destruction and
   - sometimes widening of periodontal ligament.

3. An irregular radiolucent pattern with ragged borders develops indicative of bone necrosis and pathologic resorption. (Moth eaten appearance).

4. Some radiographs will show a portion of bone separated from the parent bone. This has been termed a sequestrum, and the radiolucent band separating it from the parent bone an involucrum.
Bone marrow contains an inflammatory exudate consisting essentially of neutrophils.

Loss of osteoblasts with increased bone resorption by osteoclasts.

Sometimes a portion of necrotic bone with no viable cells forms
High-power view of a sequestrum showing non-vital bone (the osteocyte lacunae are empty) and eroded outline with superficial lacunae, produced by osteoclastic resorption

Vital bone with lacunae osteocyte
Complications and resolution

- Acute osteomyelitis usually resolves fully following aggressive treatment.
- Anaesthesia of the lower lip usually recovers with elimination of the infection.
- Rare complications include:
  1. pathological fracture caused by extensive bone destruction,
  2. chronic osteomyelitis after inadequate treatment,
  3. cellulitis due to spread of exceptionally virulent bacteria or septicaemia in an immunodeficient patient
Chronic Suppurative Osteomyelitis

**Definition:**

Chronic suppurative inflammation of bone and bone marrow
Etiology

- Chronic osteomyelitis may:
  1. Follow the acute disease, or
  2. The chronic form may result de novo from a low-grade infection.

- Organisms are often difficult to identify
Clinical features

1. Swelling, pain and sinus formation.
2. Purulent discharge.
3. Sequestrum formation.
4. Tooth loss.
5. The picture is often dominated by persistent ache or pain, often relapsing, during a long period with a bad taste from pus draining to the mouth through sinuses.

6. In more active phases there is swelling, increased pain and discharge, and increased tooth mobility.

7. There may be exposed bone. Initially the original focus of infection can be identified, but chronic osteomyelitis may persist after its removal and the chronic infection becomes self-perpetuating in the bone.
5. pathologic fracture,

6. Patients may experience acute exacerbations or periods of decreased pain associated with chronic to acute progression

7. Chronic osteomyelitis of the mandible associated with periodontal disease.
Sequestration of the entire mandible following chronic osteomyelitis of odontogenic origin.
Radiographic features

1. Patchy, ragged and ill-defined radiolucency that often contains central radiopaque sequestra
2. The lucent pattern is often described as moth-eaten because of its mottled radiographic appearance
3. The surrounding bone may exhibit an increased radio density
4. The cortical surface can demonstrate significant osteogenic periosteal hyperplasia.
The inflammatory reaction in chronic osteomyelitis can vary from very mild to intense.

**In mild cases**

1. Microscopic diagnosis can be difficult because of similarities to fibro osseous lesions such as ossifying fibroma and fibrous dysplasia.

2. A few chronic inflammatory cells (lymphocytes and plasma cells) are seen in a fibrous marrow.
In advanced (intense) chronic osteomyelitis

1. Both osteoblastic and osteoclastic activity may be seen, along with irregular bony trabeculae.

2. Necrotic bone (sequestrum) may be present, as evidenced by both necrotic marrow and necrotic osteocytes.

3. Reversal lines reflect the waves of deposition and resorption of bone. Inflammatory cells are more numerous and osteoclastic activity more prominent than in mild cases.
chronic inflammatory cells

BV

giant cell

reversal line

empty lacuna of death osteocyte
Treatment

The basic treatment of Suppurative osteomyelitis centers on the:

1. Selection of appropriate antibiotics after culture
2. The proper timing of surgical intervention.
3. Culture and sensitivity testing should be carried out.
Non-suppurative OM.

1) Chronic Osteomyelitis with Proliferative Periostitis (Garré's Osteomyelitis)
2) Diffuse Sclerosing Osteomyelitis
3) Focal Sclerosing Osteomyelitis
Chronic Osteomyelitis with Proliferative Periostitis (Garré's Osteomyelitis)

**Definition:**

Subtype of osteomyelitis that has a prominent periosteal inflammatory reaction as an additional component.
Etiology:

It most often results from:

1. a periapical abscess of a mandibular molar tooth or
2. an infection associated with tooth extraction or partially erupted molars.
Clinical Features

- It is most common in children.
- 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:
  1. an asymptomatic bony,
  2. hard swelling
  3. with normal appearing overlying skin and mucosa.
  4. On occasion, slight tenderness may be noted
Radiographic features

- **In panoramic radiograph**

  The lesion appears centrally as a mottled, predominantly lucent lesion in a pattern consistent with that of chronic osteomyelitis.
Orthopantomographic image showing a deep caries cavity in the left mandibular second premolar tooth and a radiolucent area in its apical region.
In occlusal radiograph

1. 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.

2. Trabeculae perpendicular to the onionskin layers may also be apparent.
Histopathology

1. Sub periosteal reactive new bone formation with Perpendicular orientation to the cortical bone is best seen under low magnification.

2. Osteoblasts dominate in this area, and both osteoblasts and osteoclasts are seen centrally in the mandible.

3. Marrow spaces contain fibrous tissue with scattered lymphocytes and plasma cells.

4. Inflammatory cells are often surprisingly scant, making Microscopic differentiation from fibro osseous 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.
Diffuse Sclerosing Osteomyelitis

**Definition:**

An inflammatory reaction in the mandible or maxilla, believed to be in response to a microorganism of low virulence.
**Etiology**

1. **Bacteria** are generally suspected as causative agents.
2. Combination of an *Actinomyces* species and *E. corrodens* produces a sclerosis.
3. In bone these organisms are known to be normal inhabitants of the oral flora, they become pathogens upon gaining a portal of entry into bone, where they establish and maintain an anaerobic environment via sclerosis and fibrosis.
4. **a portal of entry:**
   - Chronic periodontal
   - Carious non vital teeth pulpal infection,
   - endodontic therapy.
Clinical Features

1. 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.

2. The disease is typified by a protracted chronic course with acute exacerbations of pain, swelling, and occasionally drainage.

3. The mandible is involved mostly in the body, angle, and ramus area.

4. Similar cases have been seen in the maxilla but in much fewer numbers.

5. The mandible may be tender to palpation, particularly at the buccal cortex.
Radiographic features

In early stages

1. Diffuse,
2. Typically ill-defined lucent zones and
3. May appear in association with sclerotic masses affecting a large part of the jaw.
In advanced stages

1. Prominent endosteal sclerosis
2. Without prominent cortical bone loss and
3. With minimal or no periosteal bone formation
There is extensive patchy sclerosis of the mandible, poorly localized and without a clear focus of radiolucent infection.
1. a **chronic inflammatory** process with reactive and reparative features.

2. The marrow will appear **fibrous**, and varying quantities of inflammatory cells, predominantly lymphocytes and plasma cells, will be seen, sometimes with admixed neutrophils.

3. Irregular, thick, osseous trabeculae are seen with increased **resting lines** which are denser in sclerotic component.

4. Sometimes colonies of organisms are seen on the bone
**Treatment**

The management of diffuse sclerosing osteomyelitis is problematic because of the:

1. Relative avascular nature of the affected tissue and
2. 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.
Focal Sclerosing Osteitis

**Definition:**
Is a relatively common phenomenon that is believed to represent a focal bony reaction to a low-grade inflammatory stimulus.
Etiology:

- 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.
Other names for focal sclerosing osteitis include:

1. Focal sclerosing osteomyelitis,
2. Bony scar,
3. Condensing osteitis, and
4. Sclerotic bone.
5. The term *focal perialpical osteopetrosis* has also been used to describe:
   - Idiopathic lesions associated with
   - Normal, caries-free teeth.
Clinical Features

1. Focal sclerosing osteitis may be found at any age but is typically discovered in young adults.

2. Patients are usually asymptomatic, and most lesions are discovered on routine radiographic examination.

3. A majority are found at the apices of mandibular first molars, with a minority associated with mandibular second molars and premolars

4. When teeth are extracted, these lesions remain behind indefinitely
Radiographic features

1. The lesion most frequently is uniformly opaque
2. It may have a peripheral lunacy with an opaque center
3. Localized but uniform radiodensity related to tooth with widened periodontal ligament space or periapical area
4. 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 the inflammatory cells.
ALVEOLAR OSTEITIS
(DRY SOCKET; FIBRINOLYTIC ALVEOLITIS)

Definition:

- Osteitis simply means inflamed bone, not infection.
- Is a complication of wound healing following a tooth extraction, the term alveolar refer to the alveolus, which is the part of the jawbone that surround teeth, osteitis which means bone inflammation.
Etiology

1) Local trauma,
2) Estrogens,
3) Bacteria,
4) Traumatic extraction,
5) Oral contraceptive use
6) Pre surgical infections,
7) Inadequate irrigation at surgery and
8) The use of tobacco products
Pathogenesis:

Normally after extraction of a tooth,

Blood clot is formed at the site with then organization of the clot with by granulation tissue,

Which gradually replaced by coarse fibrillar bone and, finally by mature bone.

Destruction of the initial clot prevents appropriate healing and causes the clinical syndrome known as alveolar osteitis.
Dry Socket Following Tooth Extraction

Blood clot forms after tooth extraction which leads to healing and new bone formation.

If the blood clot doesn't form or is lost too early, a painful 'dry socket' occurs.
Extensive investigations have shown that the clot is lost secondary:

1. Activation transformation of plasminogen to plasmin, with subsequent lysis of fibrin and
2. Formation of kinins (fibrinolytic alveolitis), kinins are potent pain mediators.
Trauma and/or infection → Inflammation of bone marrow → Release of tissue activators → Conversion to Plasminogen → Conversion to Plasmin → Lysis of fibrin → Formation of kinins → Dissolution of blood clot → Pain
Clinical Features

- Mandible and the posterior areas are most frequent sites.

- 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.

- The overall prevalence is highest between 20 and 40 years of but most frequent is in the 40 to 45 year-old age group.
It is not really an infection but leads to superficial bacterial contamination of exposed bone and can progress to osteomyelitis, though extremely uncommonly.

Pain usually starts a few days after the extraction, but sometimes may be delayed for a week or more.

It is deep-seated, severe or throbbing.

The mucosa around the socket is red and tender.

There is no clot in the socket, which contains, instead, saliva and often decomposing food debris.
Sequestration of the socket wall may sometimes be seen radiographically. Radiograph performs no useful purpose except to exclude retention of a root fragment.
Features of Dry socket

1) **Bare bone.**

- After extraction the 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 delayed:
  1. by partial retention of the clot or
  2. by overlying inflamed tissue that covers the site.

- The diagnosis is confirmed by probing of the socket, which reveals exposed and extremely sensitive bone.
2) Severe pain

3) Foul odor,

4) 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.
Treatment and Prognosis

- Patient complaining of post extraction pain, a radiograph should be taken of the affected area to rule out the possibility of a retained root tip or a foreign body.
- 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 it increases the associated pain.
- Finally, the socket is packed with an antiseptic dressing,
- The dressing is changed every 24 hours for the first 3 days, then every 2 to 3 days until granulation tissue covers the exposed bone.
Prevention of dry socket

• Preoperative infection control
  1. Scaling teeth before extraction
  2. Chlorhexidine rinsing preoperatively and for 3 days postoperatively

• Atraumatic extraction

• Adherence to postoperative instructions
  1. No rinsing or forceful spitting
  2. No hot fluids
  3. No smoking

• Postoperative antibiotics only for those at particular risk