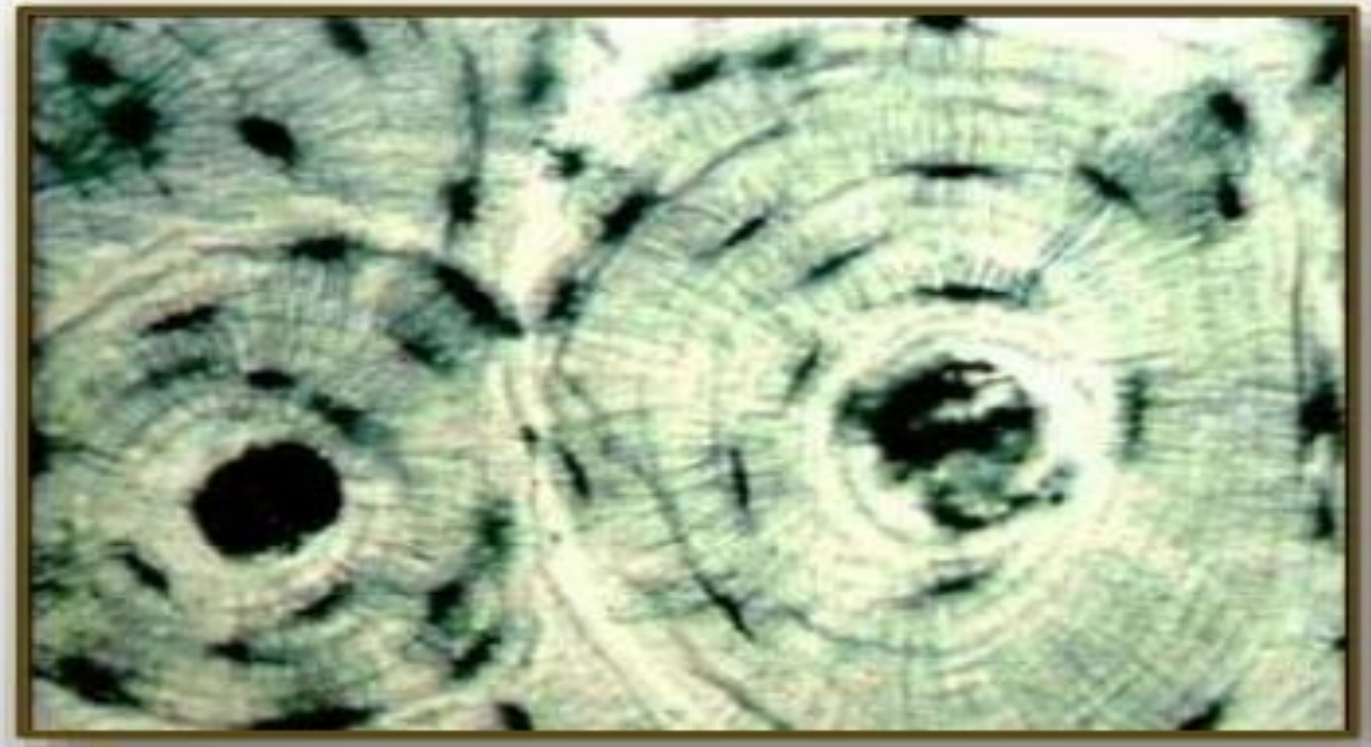
CONSTITUTION OF BONE TISSUE

- Extracellular Bone Matrix;
- Bone Cells.



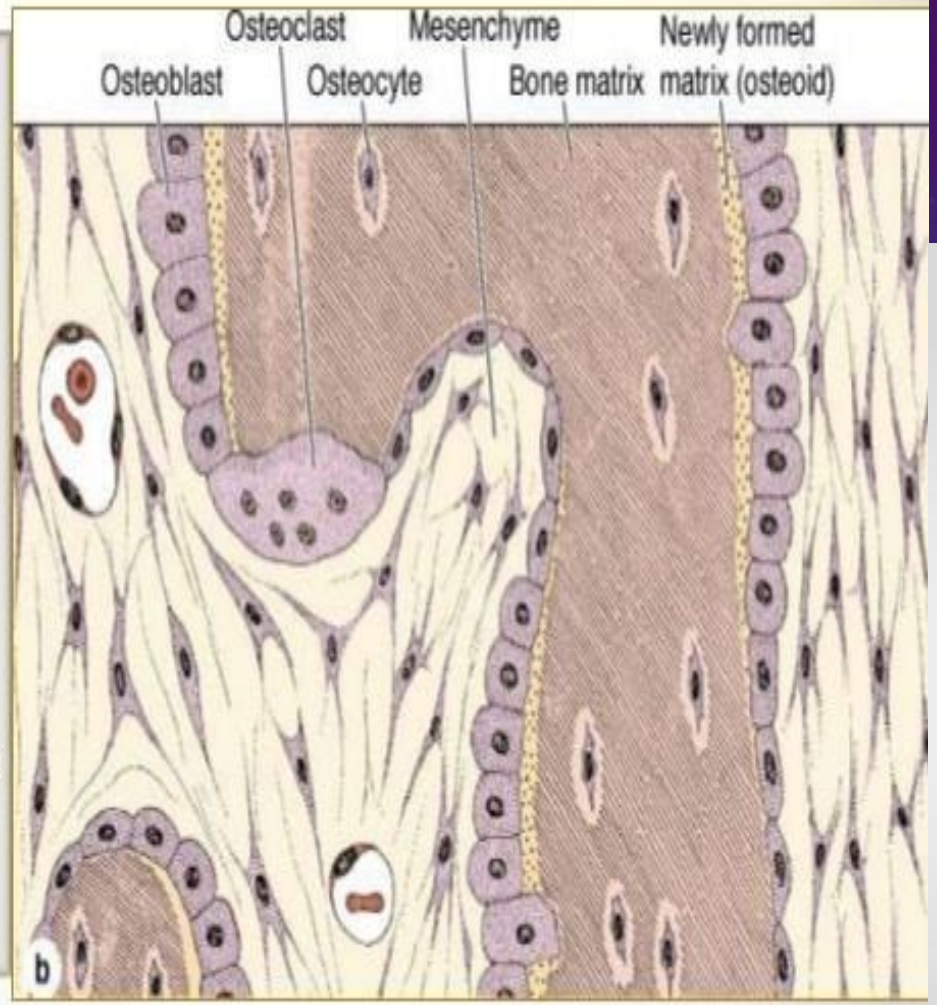
Functions:
1. Support;
2. Protection;
3. Movement;
4. Storage.

Bone Matrix

- **Organic Part :**
- **Collagen;**
- **Proteoglycans;**
- **Glycoproteins.**

- **Inorganic Part:**

Calcium phosphate crystals called **hydroxyapatite:**



Bone Matrix

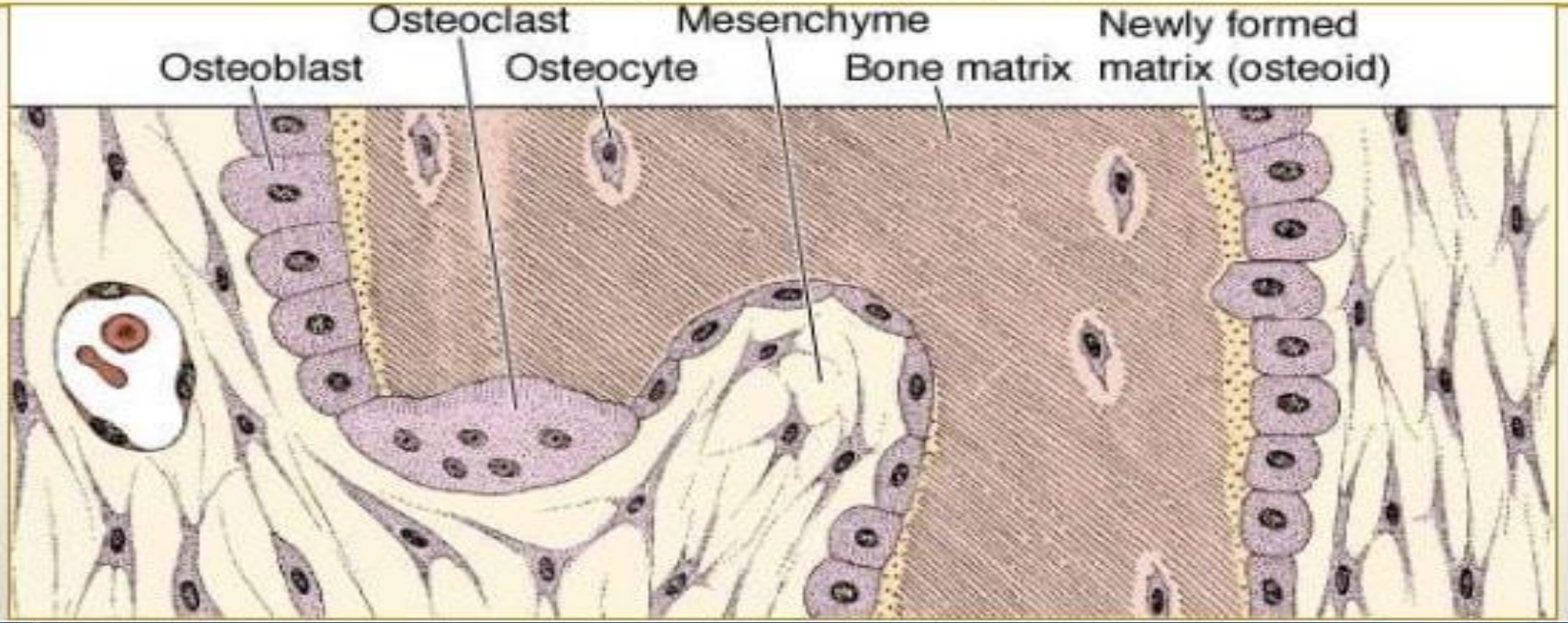
- The collagen and mineral components: Responsible for the major functional characteristics of bone.

Bone Cells

- Produce the bone matrix, become entrapped within it, and break it down so that new matrix can replace the old matrix.

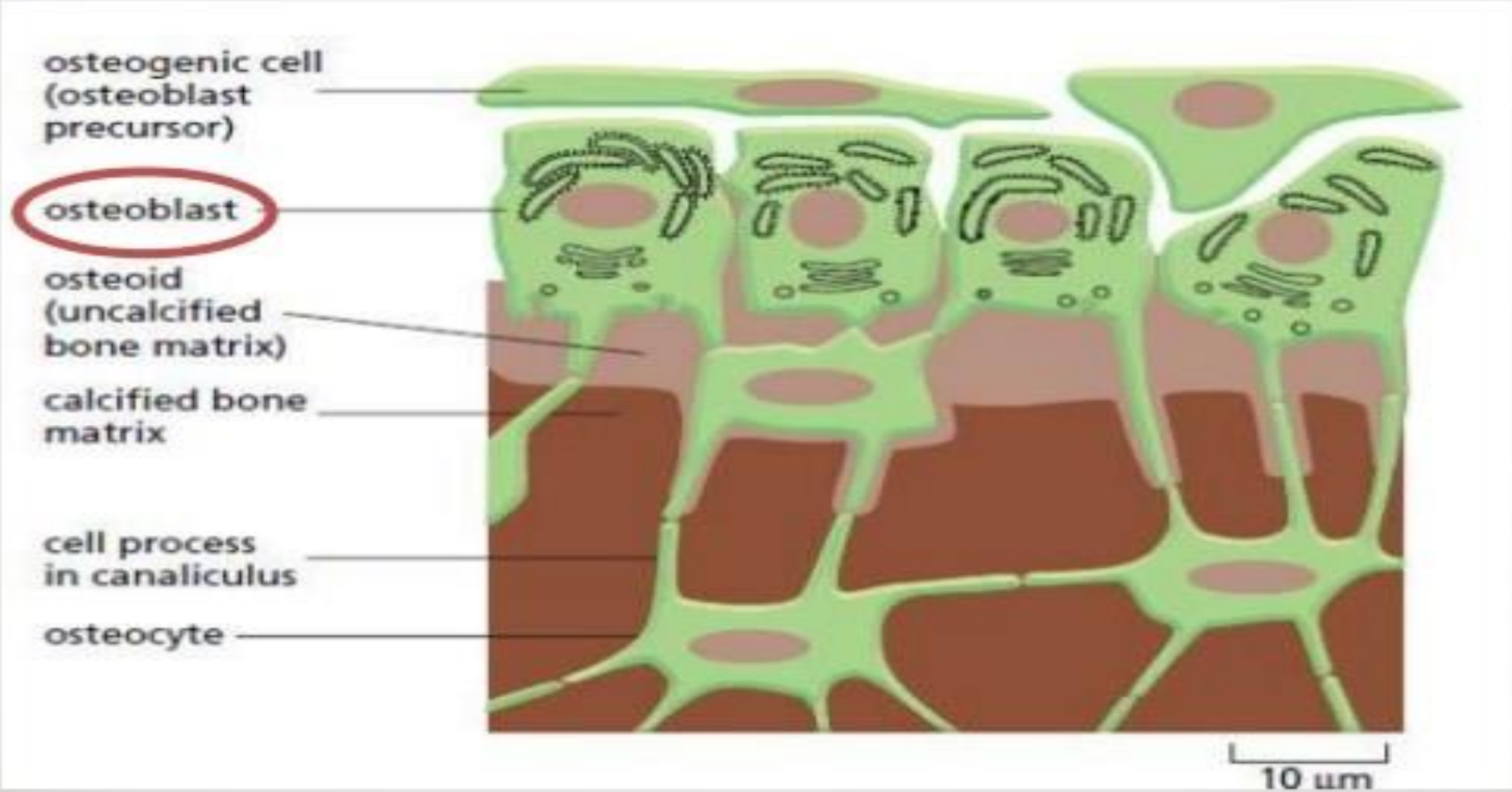
Bone cells are categorized as:

✓ **Osteoblasts; Osteocytes; Osteoclasts.**



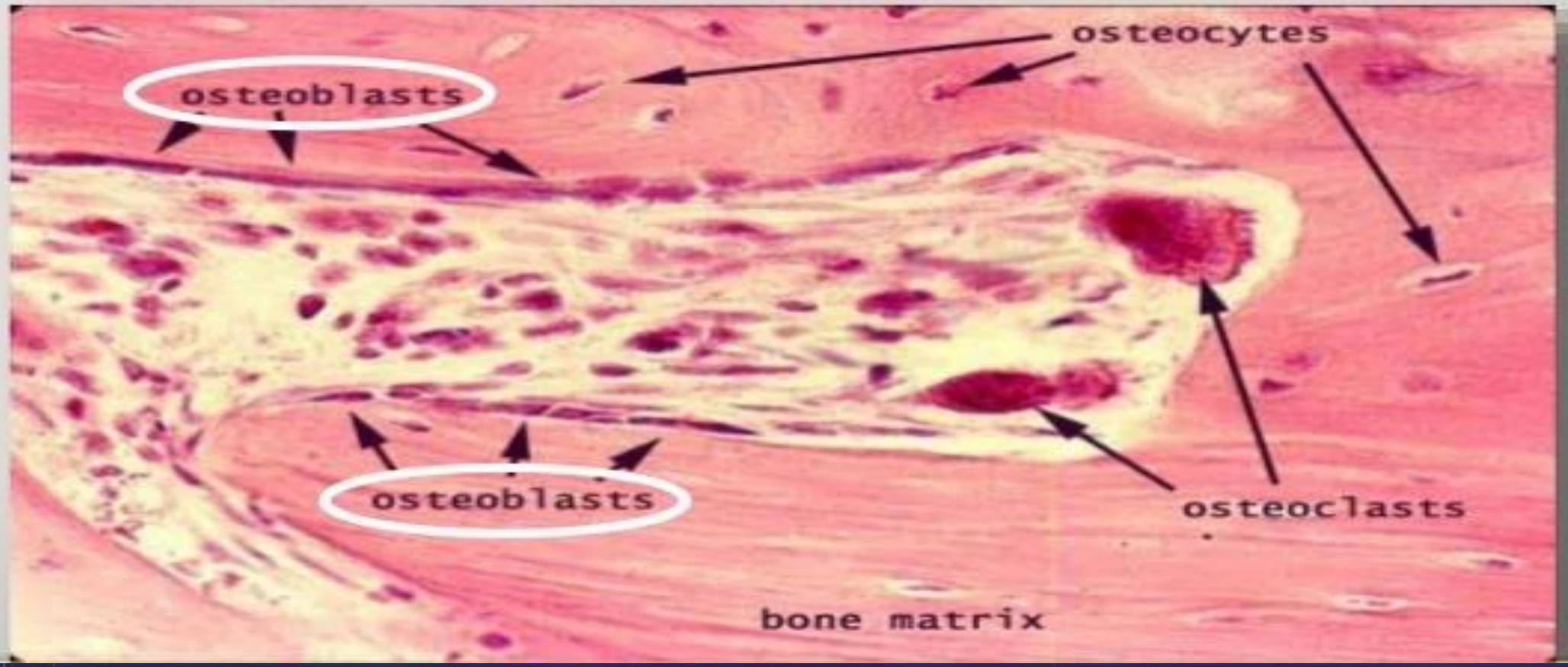
OSTEOBLASTS

- Extensive endoplasmic reticulum and numerous ribosomes;



OSTEOBLASTS

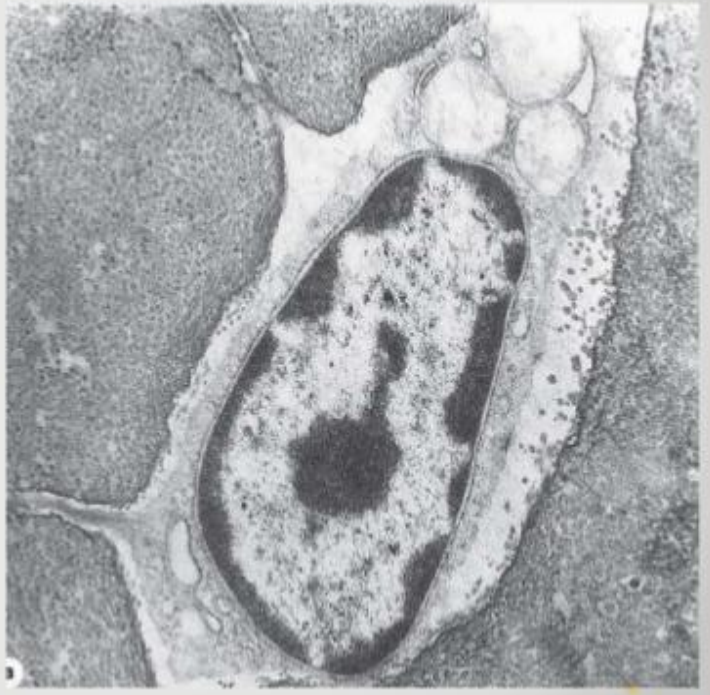
- Produce collagen and proteoglycans, which are packaged into vesicles by the Golgi apparatus and released from the cell by exocytosis;
- Form vesicles that accumulate calcium ions (Ca^{2+}), phosphate ions (PO^{2}_{4}), and various enzymes used to form hydroxyapatite crystals.



Osteocytes

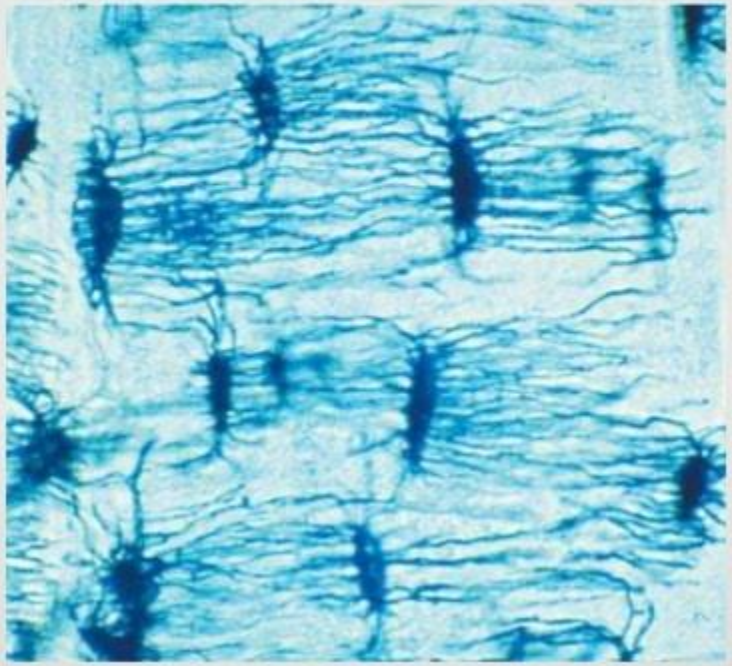
- They produce components needed to maintain the bone matrix;

- **Lacunae:** Spaces occupied by the osteocyte cell bodies;
- **Canaliculi:** Spaces occupied by the osteocyte cell processes;



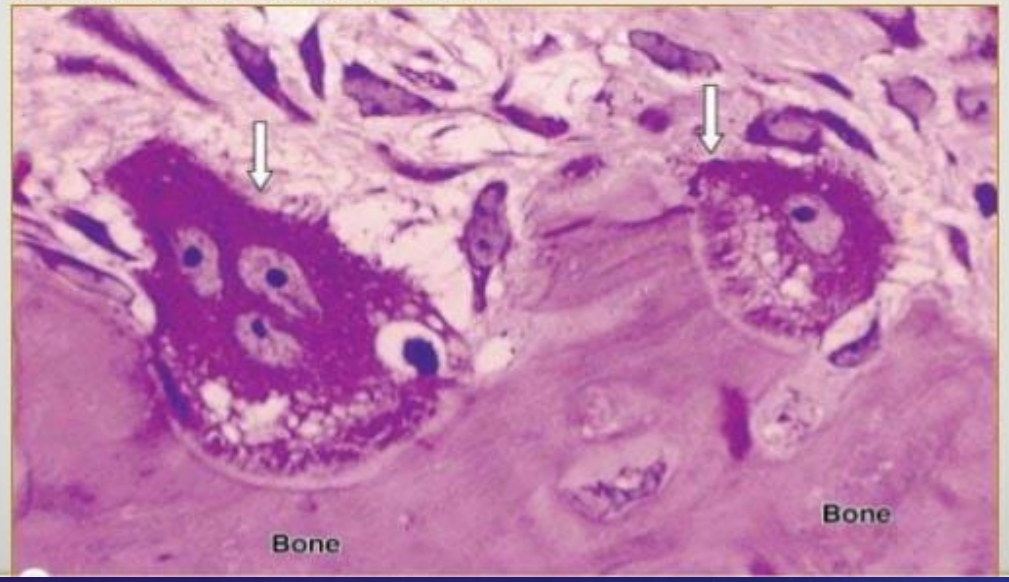
Osteocytes

- **Bone differs from cartilage in that the processes of bone cells are in contact with one another through the canaliculi**



OSTEOCLASTS

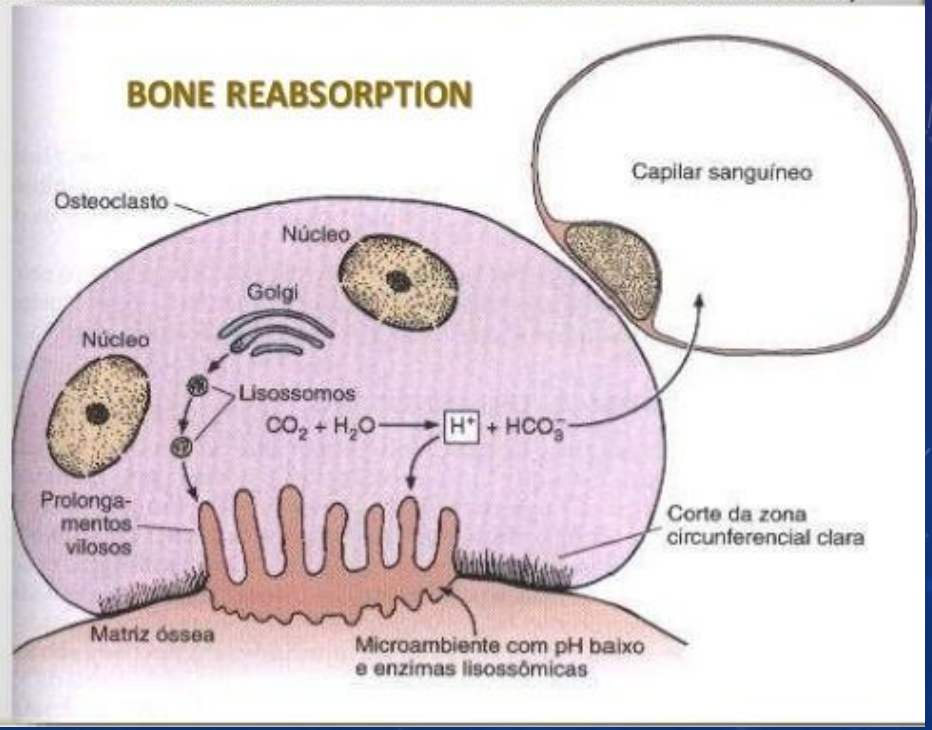
- Large cells with several nuclei;
- Responsible for the resorption, or breakdown of bone;
- **Ruffled border –** Projections where the plasma membrane of osteoclasts contacts bone matrix.



OSTEOCLASTS

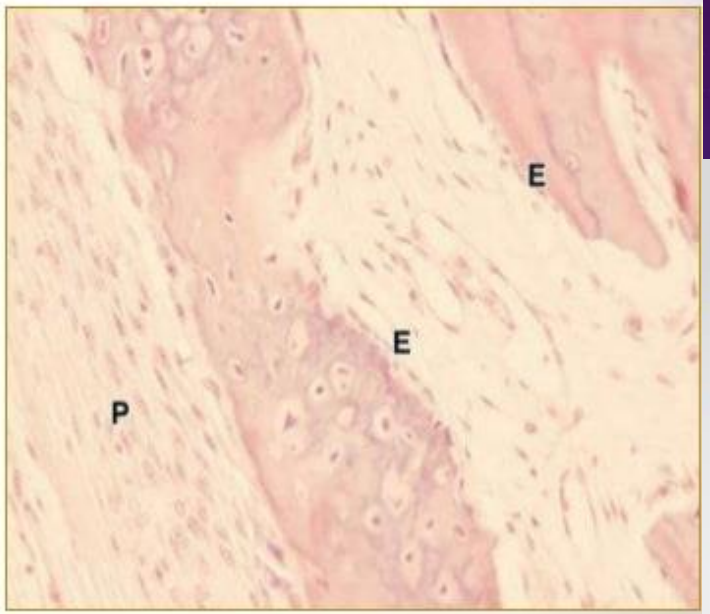
- Hydrogen ions are pumped across the ruffled border and produce an acid environment : Decalcification of the mineralized bone matrix;

By endocytosis some of the breakdown products are taken into the osteoclast.



Periosteum e Endosteum

- Layers of osteogenic cells and conjuntive tissue that covers the internal and external surfaces of the bones.



- Outer layer: **periosteum;**

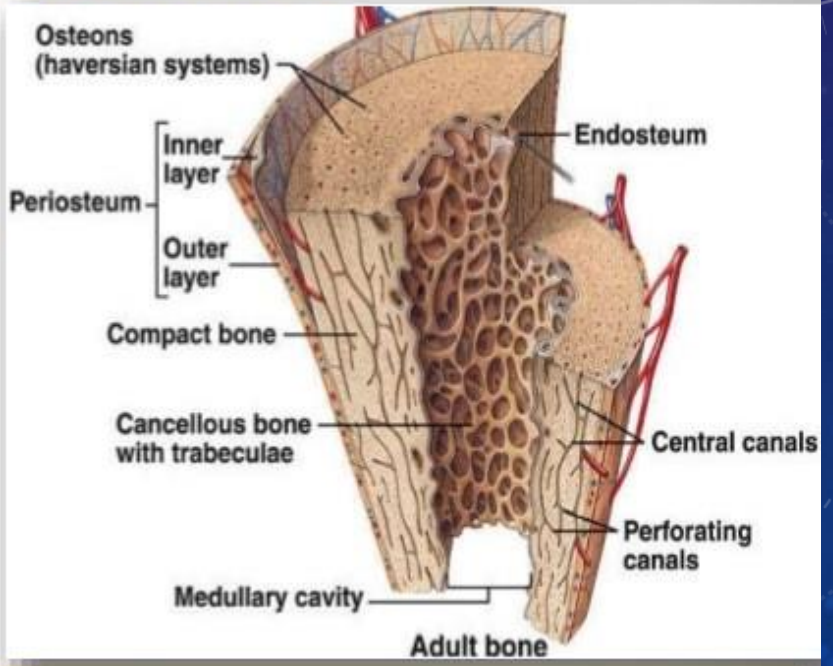
-**Collagen fibers** – Sharpey's fibers penetrate the bone and the periosteum hold firmly to the bone;

- **Fibroblasts.**

Periosteum e Endosteum

- Inner layer: **Endosteum**

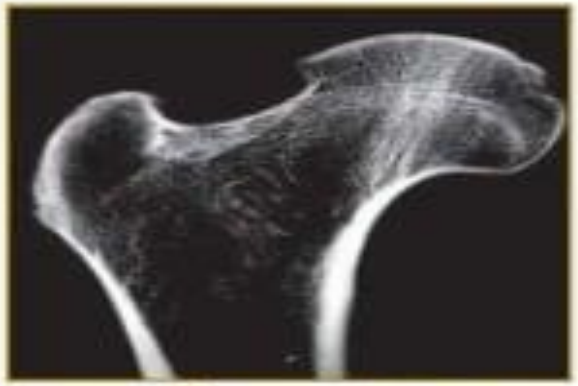
- **Osteogenic flattened cells** : Cover the trabecular bone cavity , the medullar channel, and Volkmann channel.



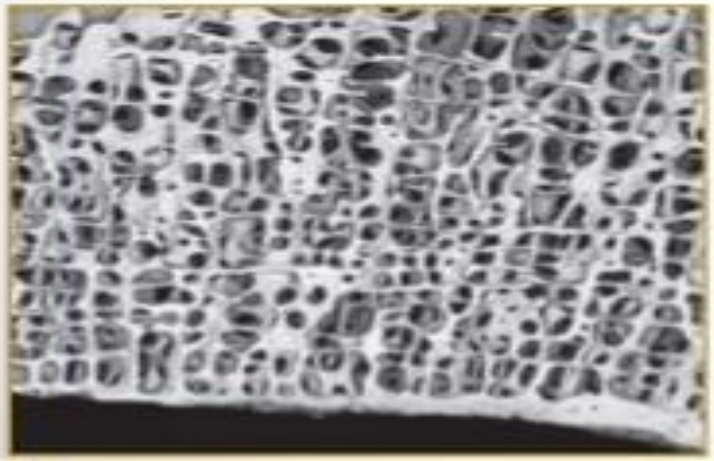
The aim of both layers is to promote the nutrition of bone tissue and provide new osteoblasts for bone growth and fracture repair.

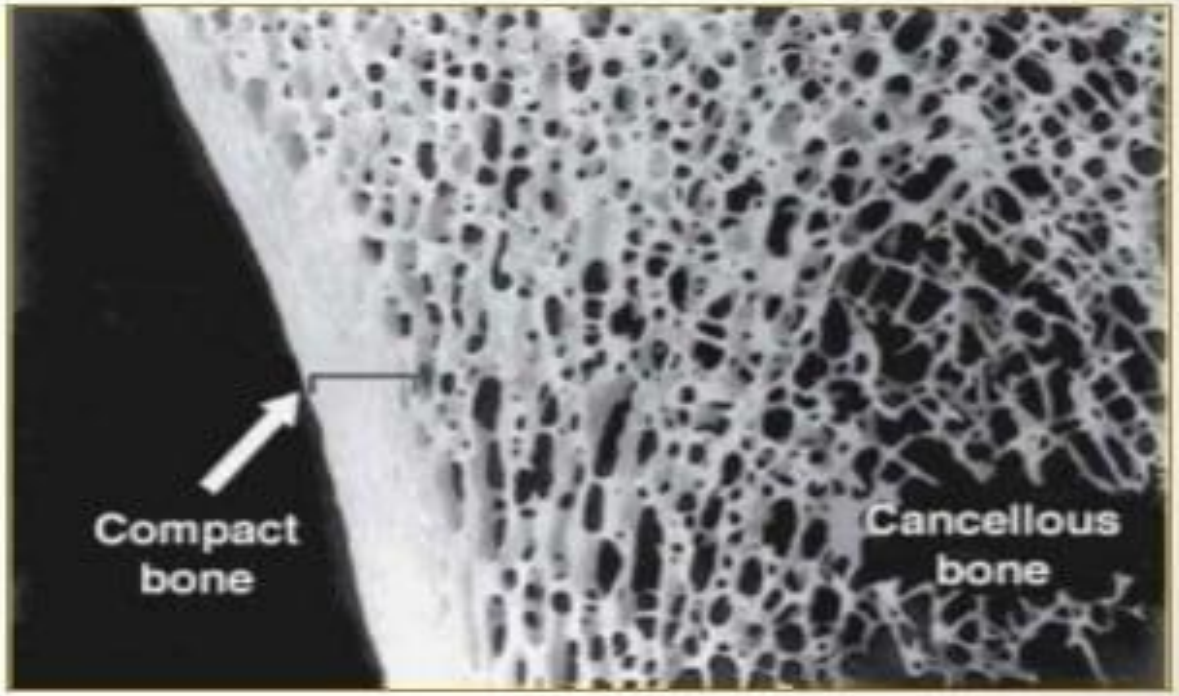
Types of Bone Tissue

- **Compact bone**
  - **No visible cavities**



- **Cancellous Bone**
  - **Full of interconnecting channels**





OSTEOMYELITIS

WHAT'S IN THE NAME?

- The word "osteomyelitis" originates from the ancient Greek words osteon (bone) and muelinos (marrow) and literally means infection of medullary portion of the bone.

WHAT IS IT?

- It is an acute & chronic inflammatory process in the medullary spaces or cortical surfaces of bone that extends away from the initial site of involvement.

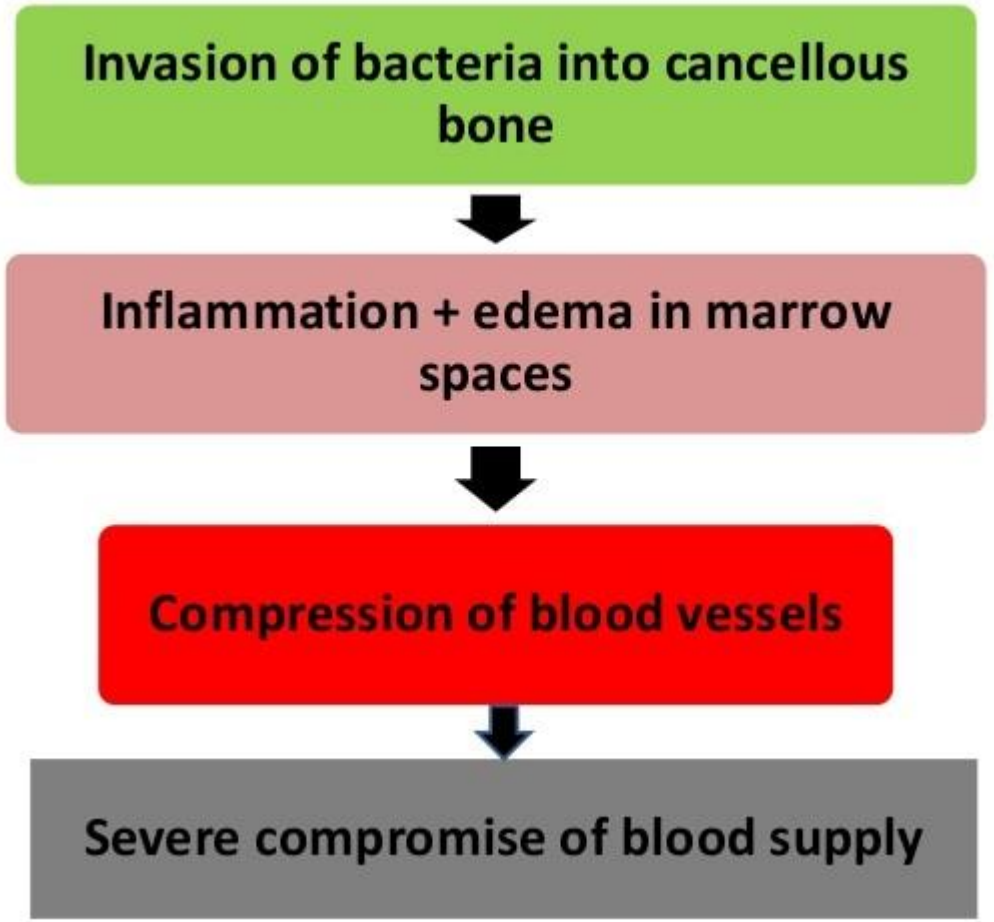
1-Osteitis: - this term is used to describe a localized inflammation of bone with no progression through the marrow spaces. Particularly that associated with infected sockets following removal of teeth, (**dry socket**).

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

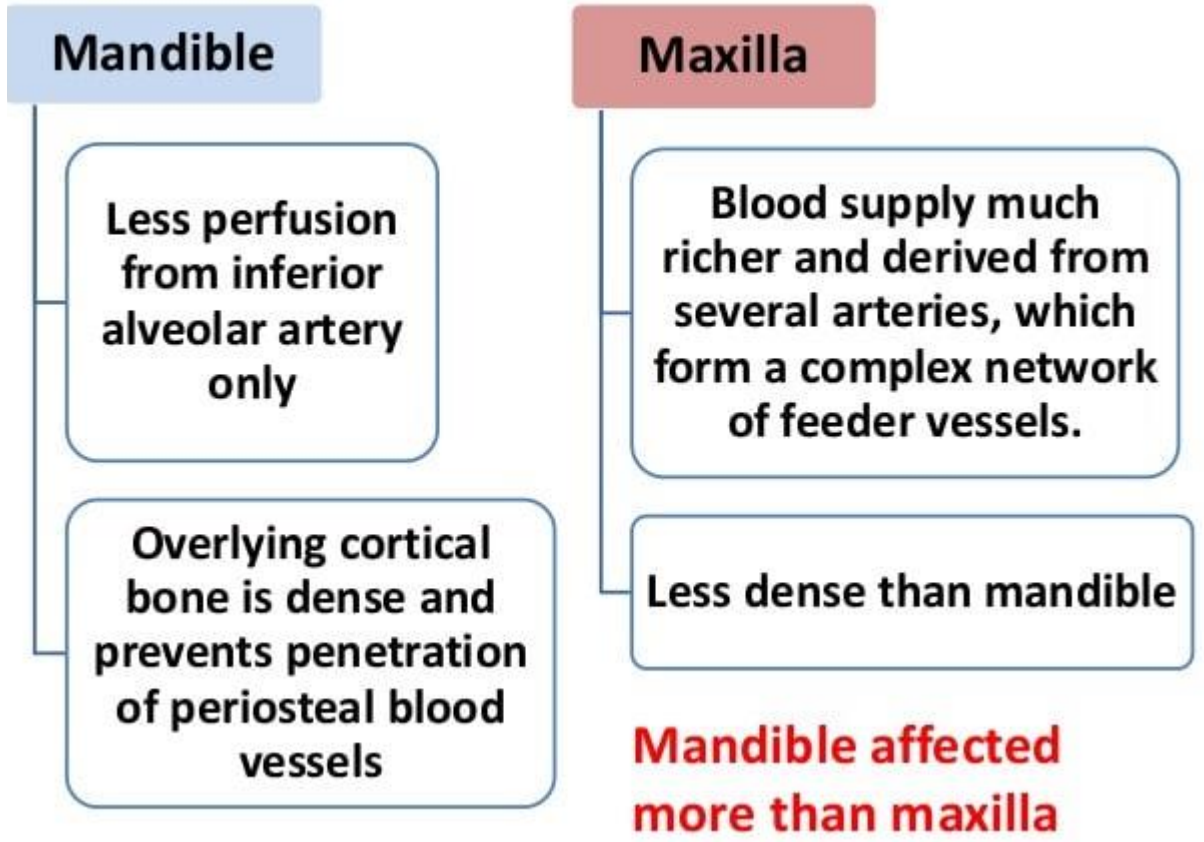
3-Periostitis: - inflammation of the periosteal spaces of the bone and may not be associated with osteomyelitis.

Definition

- Inflammation of the bone forming elements with tendency to progression.



- Inadequate blood supply is a main factor as the involved area becomes ischemic and bone becomes necrotic.
- Bacteria can then proliferate, because normal blood-borne defenses do not reach the tissue, and the osteomyelitis spreads until it is stopped by medical and surgical therapy.



FACTORS PREDISPOSING TO OSTEOMYELITIS

LOCAL FACTORS
(decreased vascularity/vitality of bone)

- Trauma.
- Radiation injury.
- Paget's disease.
- Osteoporosis.
- Major vessel disease.

SYSTEMIC FACTORS
(impaired host defense)

- Immune deficiency states.
- Immunosuppression
- Diabetes mellitus.
- Malnutrition.
- Extremes of age.

PATHOGENESIS OF OSTEOMYELITIS

Inflammatory process of entire bone including cortex & periosteum, not just confined to endosteum

↓

Inflammatory condition beginning in medullary cavity & havarsian system & extending to involve periosteum of affected area

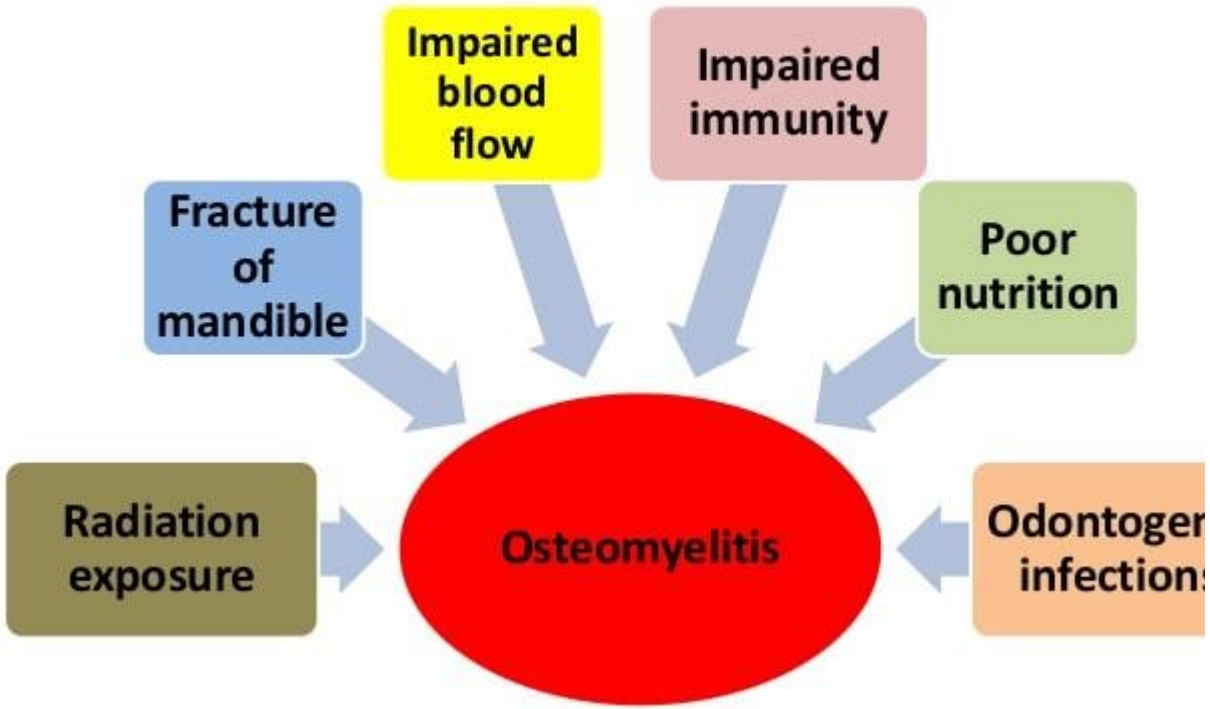
↓

Local factors decreases the vitality of bone

↓

Systemic conditions comprises the defense system of the host

Predisposing factors

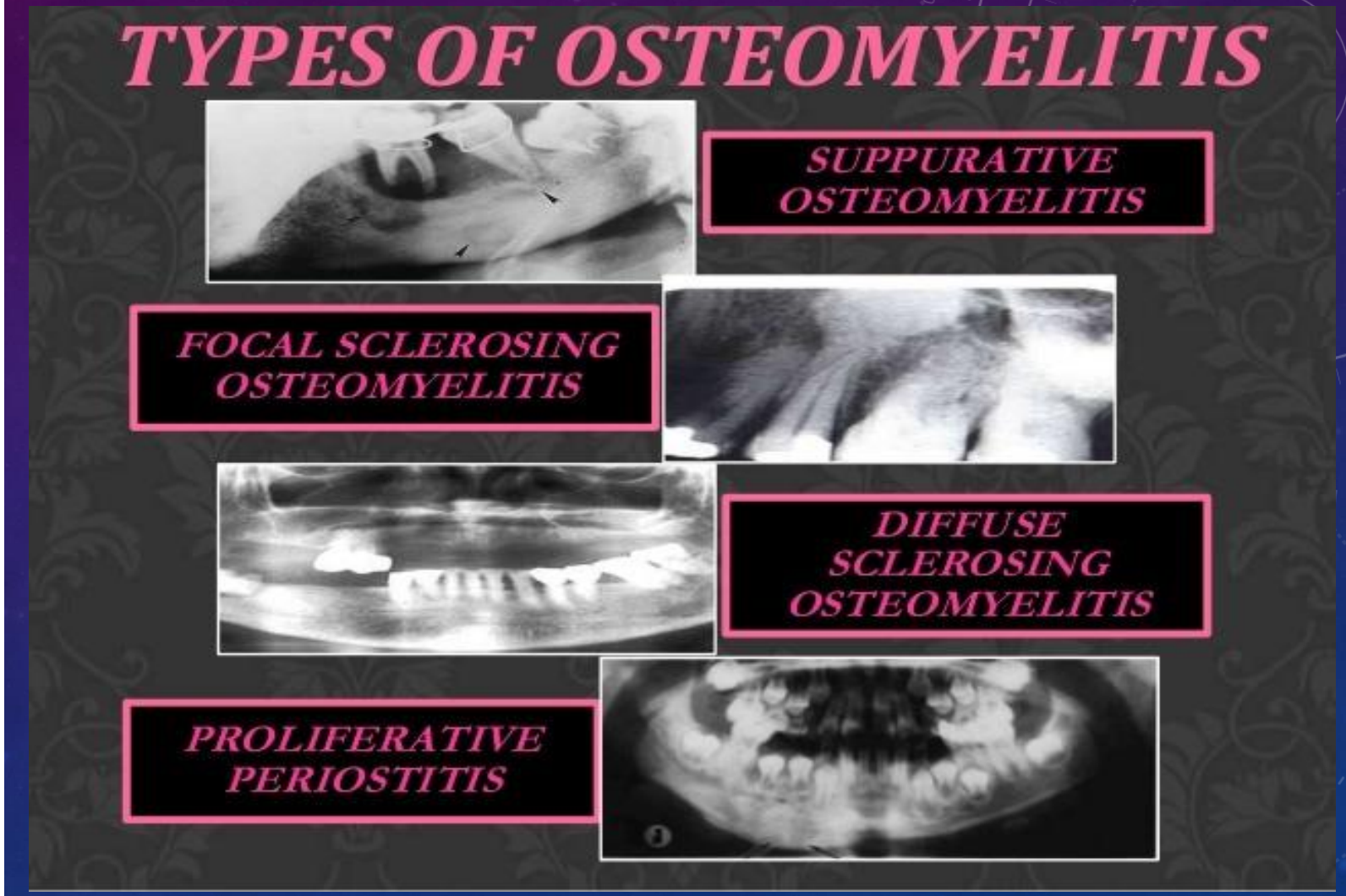


Microbiology

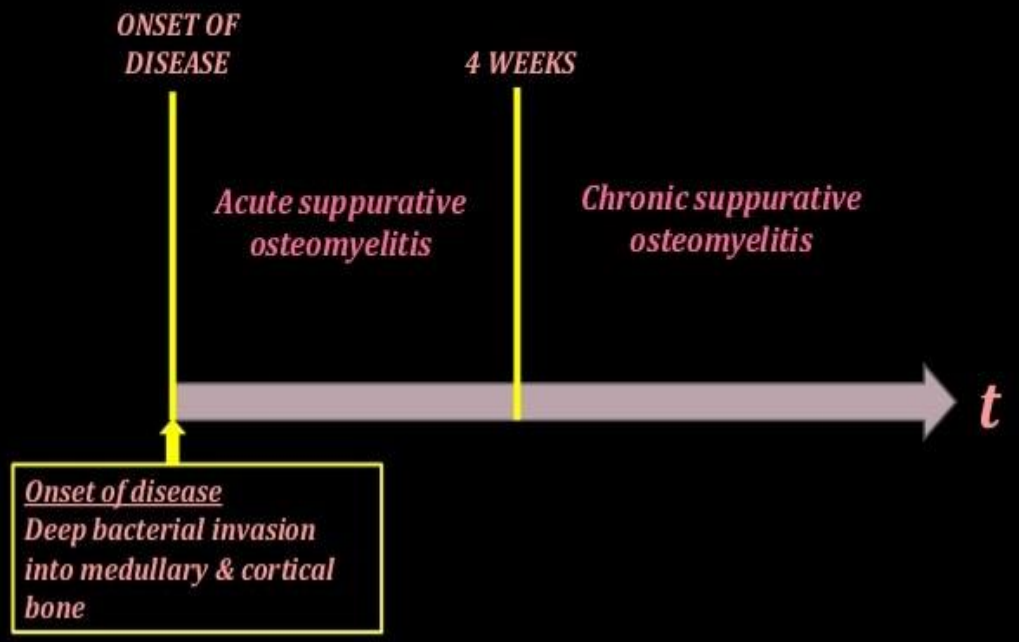
- Similar to those of odontogenic infections
  - Viridan streptococci
  - Strict anaerobes:
    - Bacteroides
    - Prevotella
    - Fusobacterium
    - Peptostreptococci species

Clinical features of osteomyelitis of facial region

- Pain
- Swelling and erythema of overlying tissues
- Adenopathy
- Fever
- Paresthesia of the inferior alveolar nerve
- Trismus
- Malaise
- Fistulas



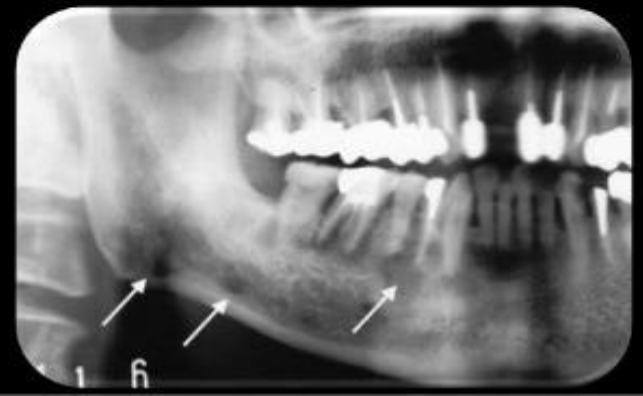
SUPPURATIVE OSTEOMYELITIS



SUPPURATIVE OSTEOMYELITIS

- *Source of infection is usually an adjacent focus of infection associated with teeth or with local trauma.*
- *It is a polymicrobial infection, predominating anaerobes such as Bacteriods, Porphyromonas or Provetella.*
- *Staphylococci may be a cause when an open fracture is involved.*
- *Mandible is more prone than maxilla as vascular supply is readily compromised.*

Cropped panoramic radiograph of suppurative osteomyelitis at the right side of mandible.



ACUTE SUPPURATIVE OSTEOMYELITIS

Organisms entry into the jaw, mostly mandible, compromising the vascular supply

↓

Medullary infection spreads through marrow spaces

↓

Thrombosis in vessels leading to extensive necrosis of bone

↓

Lacunae empty of osteocytes but filled with pus , proliferate in the dead tissue

↓

Suppurative inflammation extend through the cortical bone to involve the periosteum

↓

Stripping of periosteum comprises blood supply to cortical plate, predispose to further bone necrosis

↓

Sequestrum is formed bathed in pus, separated from surrounding vital bone

ACUTE SUPPURATIVE OSTEOMYELITIS

CLINICAL FEATURES

EARLY :

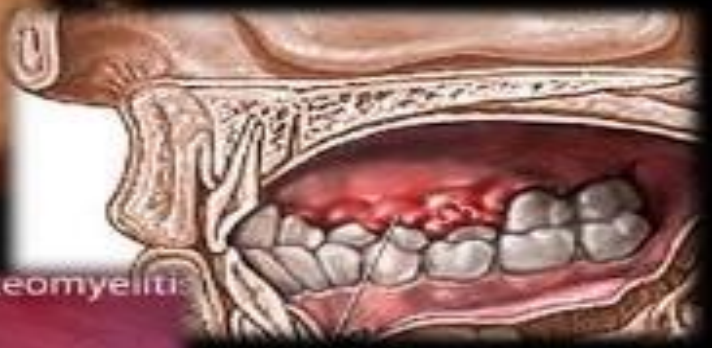
- Severe throbbing, deep- seated pain.
- Swelling due to inflammatory edema.
- Gingiva appears red, swollen & tender.

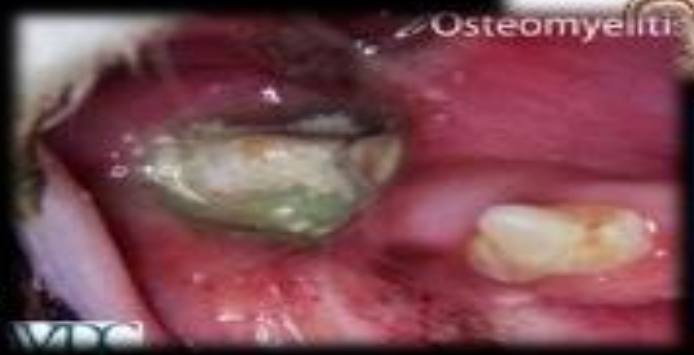
LATE :

- Distension of periosteum with pus.

FINAL:

- Subperiosteal bone formation cause swelling to become firm.





ACUTE SUPPURATIVE OSTEOMYELITIS

HISTOLOGY

- Submitted material for biopsy predominantly consists of necrotic bone & is diagnosed as sequestrum

- Bone shows:
  - Loss of osteocytes from lacunae.
  - Peripheral resorption.
  - Bacterial colonization.
  - Acute inflammatory infiltrate consisting of polymorphonuclear leukocytes in haversian canals & peripheral bone.

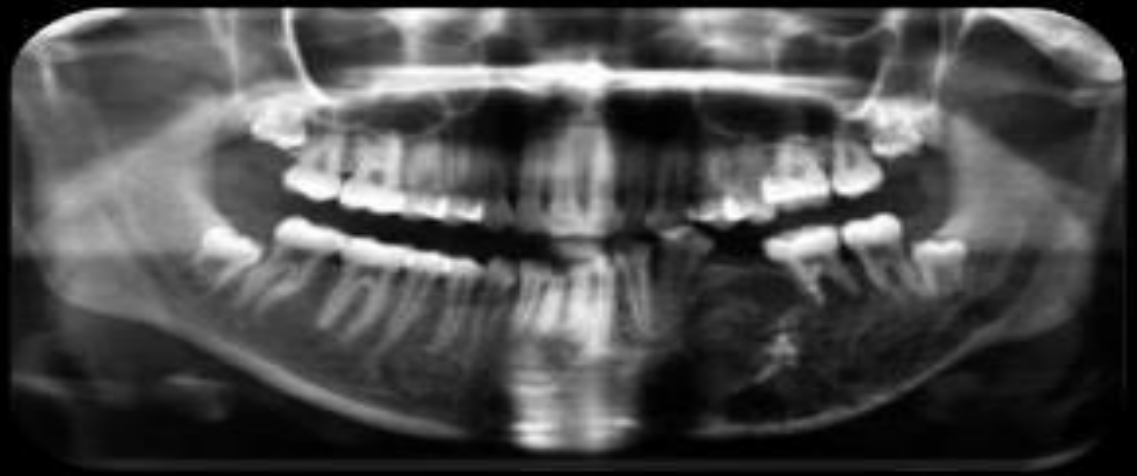
ACUTE SUPPURATIVE OSTEOMYELITIS

RADIOGRAPHIC FEATURES

- *May be normal in early stages of disease .*
- *Do not appear until after at least 10 days.*

Radiograph may demonstrate ill-defined radiolucency.

After sufficient bone resorption irregular, mot-eaten areas of radiolucency may appear.



ACUTE SUPPURATIVE OSTEOMYELITIS

MANAGEMENT

ESSENTIAL MEASURES

- *Bacterial sampling & culture.*
- *Emperical antibiotic treatment.*
- *Drainage.*
- *Analgesics.*
- *Specific antibiotics based on culture & sensitivity.*
- *Debridement.*
- *Remove source of infection, if possible.*

ADJUNCTIVE TREATMENT

- *Sequestrectomy.*
- *Decortication (if necessary)*
- *Hyperbaric oxygen.*
- *Resection & reconstruction for extensive bone destruction.*

CHRONIC SUPPURATIVE OSTEOMYELITIS

- *Inadequate treatment of acute osteomyelitis*
- *Periodontal diseases*
- *Pulpal infections*
- *Extraction wounds*
- *Infected fractures*

↓

Infection in the medulllary spaces spread and form granulation tissue

↓

Granulation tissue forms dense scar to wall off the infected area

↓

Encircled dead space acts as a reserviour for bacteria & antibiotics have great difficulty reaching the site

CHRONIC SUPPURATIVE OSTEOMYELITIS

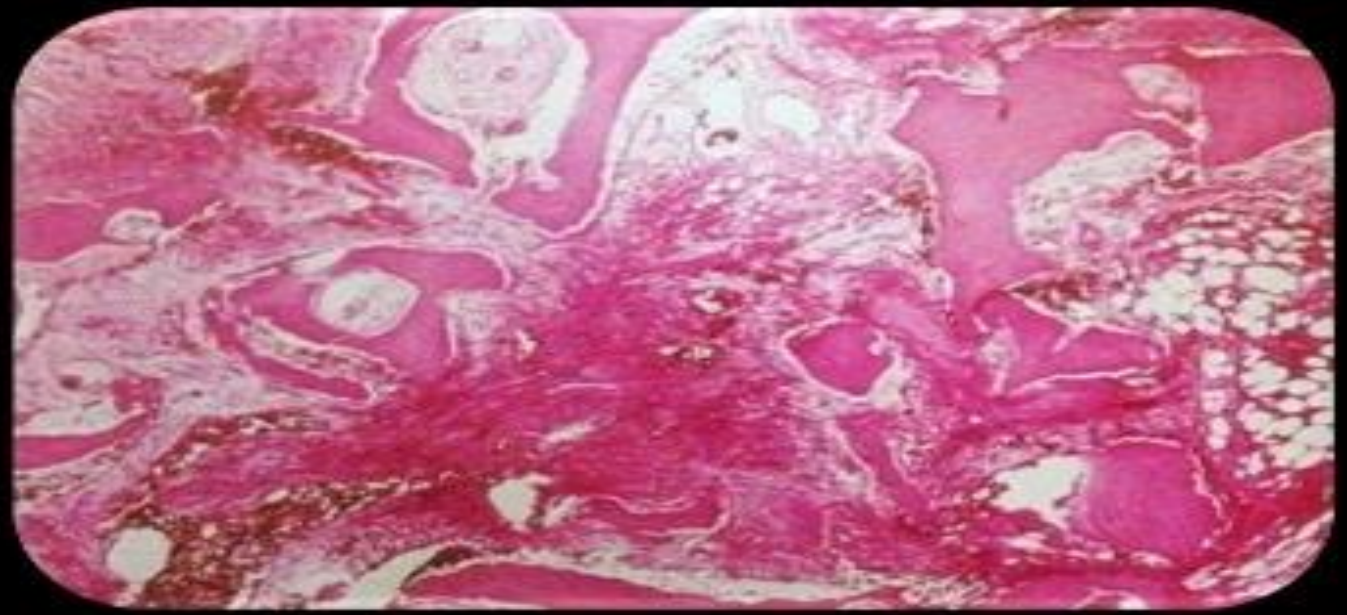
CLINICAL FEATURES

- *Swelling*
- *Pain*
- *Sinus formation*
- *Purulent discharge*
- *Sequestrum formation*
- *Tooth loss*
- *Pathologic fracture*

CHRONIC SUPPURATIVE OSTEOMYELITIS

HISTOLOGY

- *Inflammed connective tissue filling inter-trabecular areas of bone.*
- *Scattered sequestra.*
- *Pockets of abscess.*

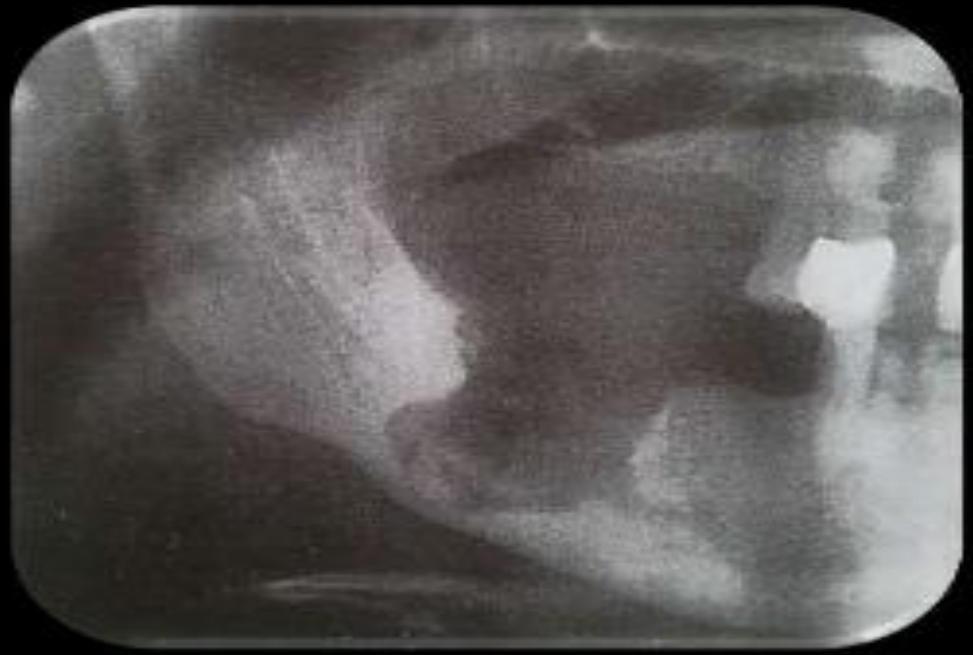


CHRONIC SUPPURATIVE OSTEOMYELITIS

RADIOLOGY

- *Patchy, ragged & ill defined radiolucency.*
- *Often contains radiopaque sequestra.*

- *Sequestra lying close to the peripheral sclerosis & lower border.*
- *New bone formation is evident below lower border.*



CHRONIC SUPPURATIVE OSTEOMYELITIS

MANAGEMENT

- *Difficult to manage medically.*
- *Surgical intervention is mandatory, depends on spread of process.*
- *Antibiotics are same as in acute condition but are given through IV in high doses.*

SMALL LESIONS

Curretage, removal of necrotic bone and decortication are sufficient.

EXTENSIVE OSTEOMYELITIS

Decortication combined with transplantation of cancellous bone chips.

PERSISTANT OSTEOMYELITIS

Resection of diseased bone followed by immediate reconstruction with an autologous graft is required. Weakened jawbones must be immobilized.

FOCAL SCLEROSING OSTEOMYELITIS

- *Also known as "Condensing osteitis".*
- *Localized areas of bone sclerosis.*
- *Bony reaction to low-grade peri-apical infection or unusually strong host defensive response.*
- *Association with an area of inflammation is critical.*

FOCAL SCLEROSING OSTEOMYELITIS

CLINICAL FEATURES

- *Children & young adults are affected.*
- *In mandible, premolar & molar regions are affected.*
- *Bone sclerosis is associated with non-vital or pulpitic tooth.*
- *No expansion of the jaw.*

HISTOLOGY

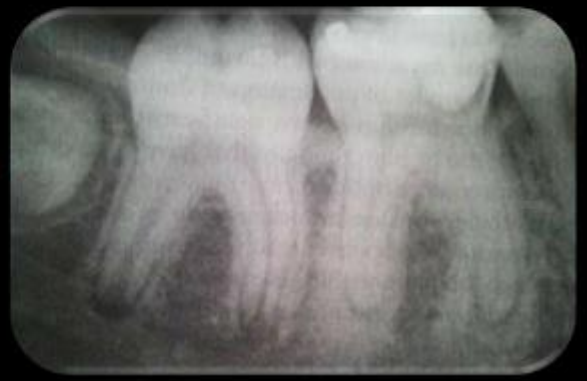
- *Dense sclerotic bone.*
- *Scanty connective tissue.*
- *Inflammatory cells.*

FOCAL SCLEROSING OSTEOMYELITIS

RADIOLOGY

- Localized but uniform increased radiodensity related to tooth.
- Widened periodontal ligament space or peri-apical area.
- Sometimes an adjacent radiolucent inflammatory lesion may be present.

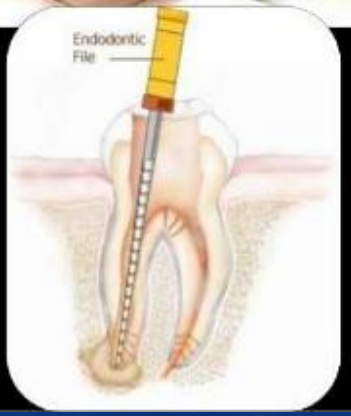
Increased areas of radiodensity surrounding apices of nonvital mandibular first molar



FOCAL SCLEROSING OSTEOMYELITIS

MANAGEMENT

- Elimination of the source of inflammation by extraction or endodontic treatment.
- If lesion persists and periodontal membrane remains wide, reevaluation of endodontic therapy is considered.
- After resolution of lesion, inflammatory focus is termed as bone scar.



DIFFUSE SCLEROSING OSTEOMYELITIS

- *It is an ill-defined, highly controversial, evolving area of dental medicine.*
- *Exact etiology is unknown.*
- *Chronic intraosseous bacterial infection creates a smoldering mass of chronically inflammed granulation tissue.*

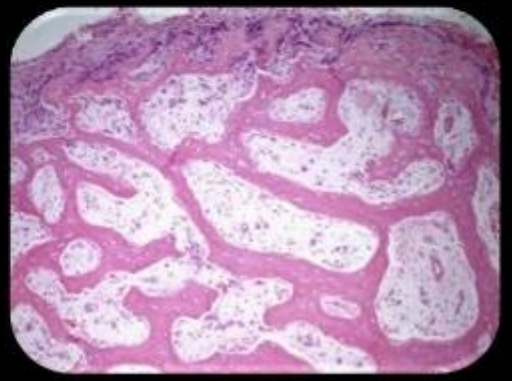
DIFFUSE SCLEROSING OSTEOMYELITIS

CLINICAL FEATURES

- *Arises exclusively in adult-hood with no sex pre-dominance.*
- *Primarily occurs in mandible.*
- *No pain.*
- *No swelling.*

HISTOLOGY

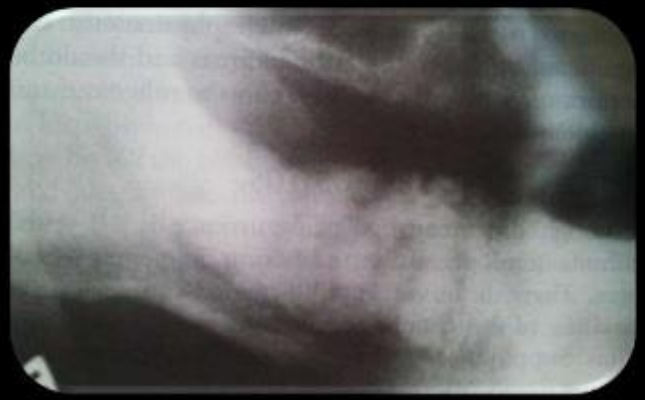
- *Bone sclerosis and remodling.*
- *Scanty marrow spaces.*
- *Necrotic bone separates from vital bone & become surrounded by granulation tissue.*
- *Secondary bacterial colonization often is visible.*



DIFFUSE SCLEROSING OSTEOMYELITIS

RADIOLOGY

Increased radiodensity may be seen surrounding areas of lesion.

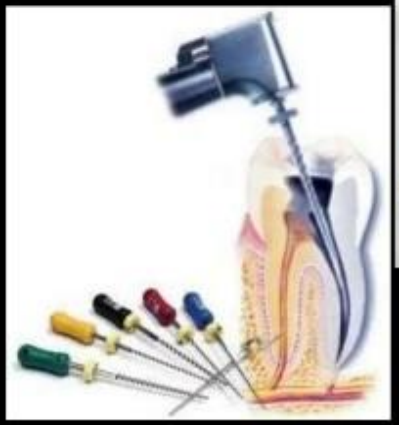


Diffuse area of increased radiodensity of Rt. Side of mandible

DIFFUSE SCLEROSING OSTEOMYELITIS

MANAGEMENT

- *Elimination of originating sources of inflammation via extraction & endodontic treatment.*
- *Sclerotic area remain radiographically.*



PROLIFERATIVE PERIOSTITIS

- *Also known as " Periostitis ossificans" & "Garee's osteomyelitis".*
- *It represents a periosteal reaction to the presence of inflammation.*
- *Affected periosteum forms several rows of reactive vital bone that parallel each other & expand surface of altered bone.*

PATHOGENESIS

The spread of low-grade, chronic apical inflammation through cortical bone

↓

Periosteal reaction occurs

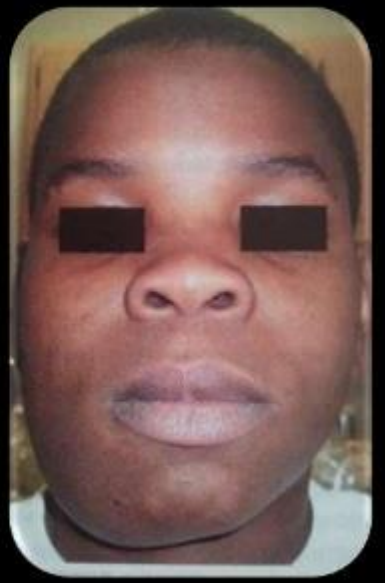
↓

Stimulates proliferative reaction of periosteum

PROLIFERATIVE PERIOSTITIS

CLINICAL FEATURES

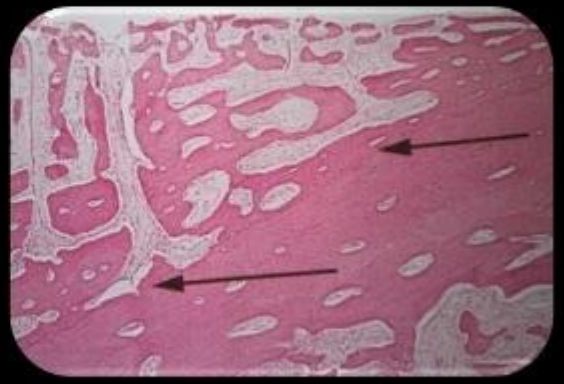
- *Affected patients are primarily children & young adults.*
- *Incidence is mean age of 13 years.*
- *No sex predominance is noted.*
- *Most cases arise in the premolar & molar area of mandible.*
- *Hyperplasia is located most commonly along lower border of mandible.*
- *Most cases are uni-focal, multiple quadrants may be affected.*



PROLIFERATIVE PERIOSTITIS

HISTOLOGY

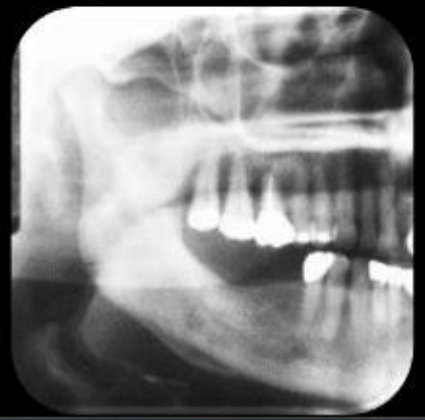
- Parallel rows of highly cellular & reactive woven bone .
- Trabeculae are frequently oriented perpendicular to surface.
- Trabeculae sometimes form an interconnecting meshwork of bone.
- Between trabeculae, uninflammed fibrous tissue is evident.



PROLIFERATIVE PERIOSTITIS

RADIOLOGY

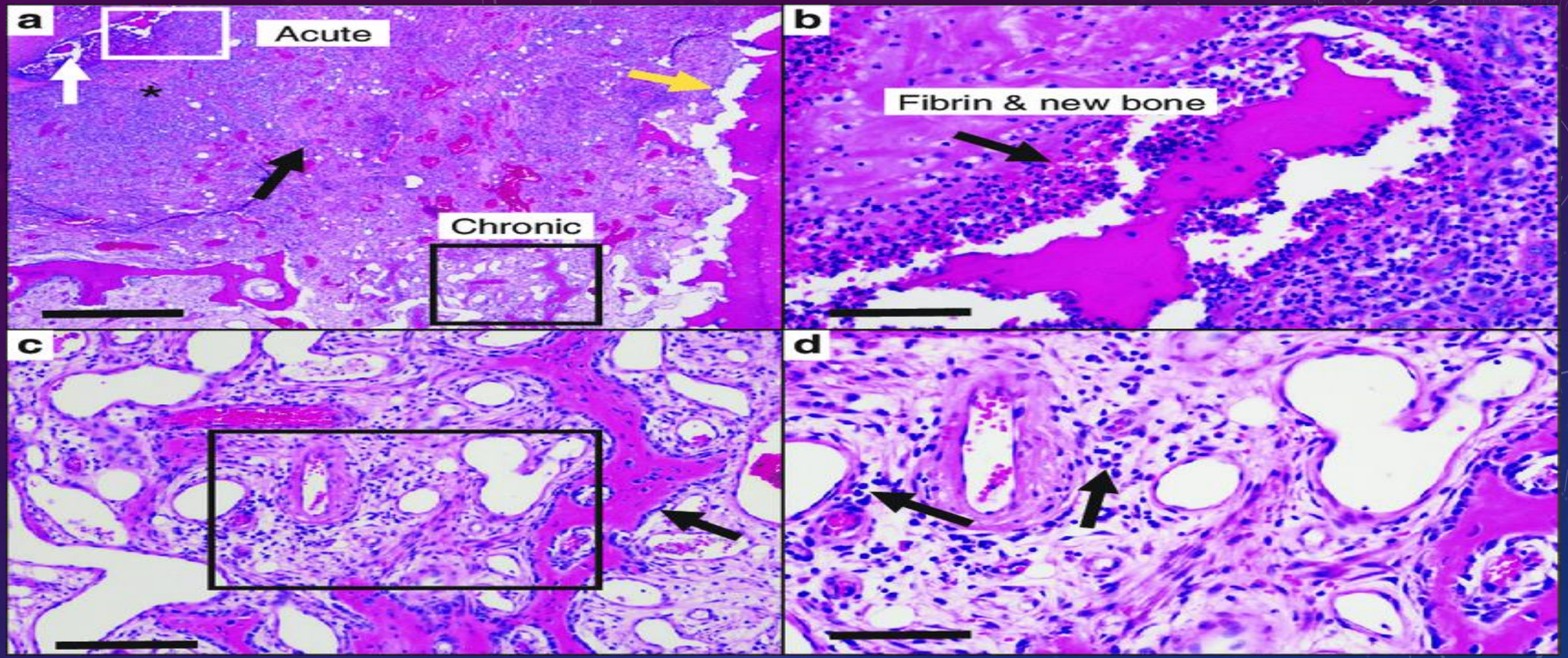
- Radiopaque laminations of bone roughly parallel each other & underlying cortical surface.
- Laminations may vary from 1-12 in number.
- Radiolucent separations often are present between new bone & original cortex.



PROLIFERATIVE PERIOSTITIS

MANAGEMENT

- Removal of infection.
- After infection has resolved, layers of bone will consolidate in 6-12 months .



Histologic features of "acute" and "chronic" osteomyelitis exist in the same lesion. the trabecular bone destroyed and replaced by an acute inflammatory reaction, consisting of neutrophils and fibrovascular granulation tissue (black arrow) . The inflammation extends to the bone beneath the articular cartilage (yellow arrow) and has destroyed much of the cortical bone (white arrow). Reactive new bone has formed in the lower part of the image, along with a chronic inflammatory and fibrovascular reaction. b A region of interest of acute inflammation (white box in a) is shown highlighting a fragment of dead cortical bone surround by neutrophils (black arrow), with an associated fibrinous exudate, which are hallmarks of acute osteomyelitis . c A region of interest of chronic inflammation (black box in a) showing new bone formation (black arrow), and replacement of normal bone marrow with fibrovascular inflammatory tissue (boxed region) . d This region of interest (boxed area in c) is presented at high power, showing blood vessels, osteoblasts rimming newly formed woven bone (bottom right), and collections of lymphocytes and plasma cells (arrows), which are characteristic of chronic osteomyelitis


Thank
You