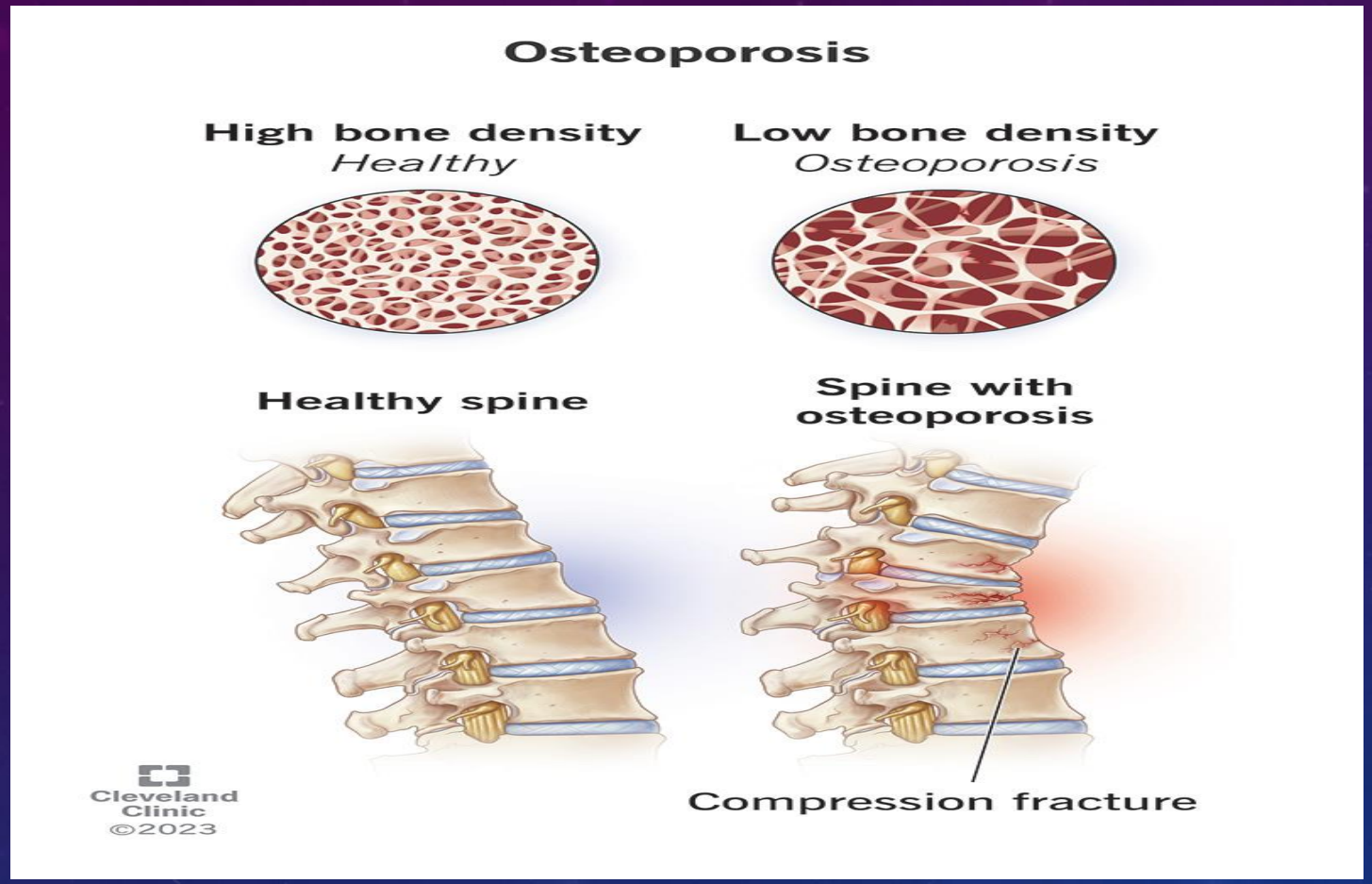
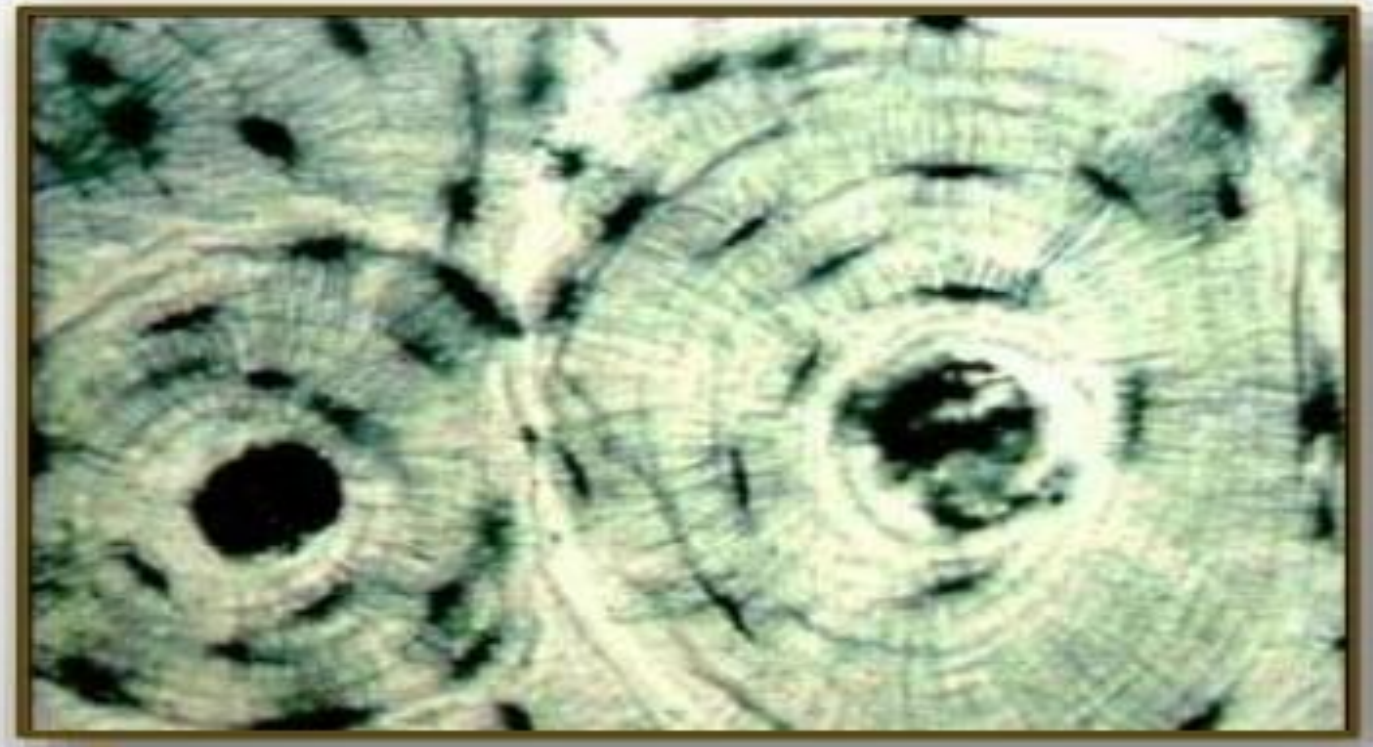
BONE DISEASE



CONSTITUTION OF BONE TISSUE

- Extracellular Bone Matrix;
- Bone Cells.



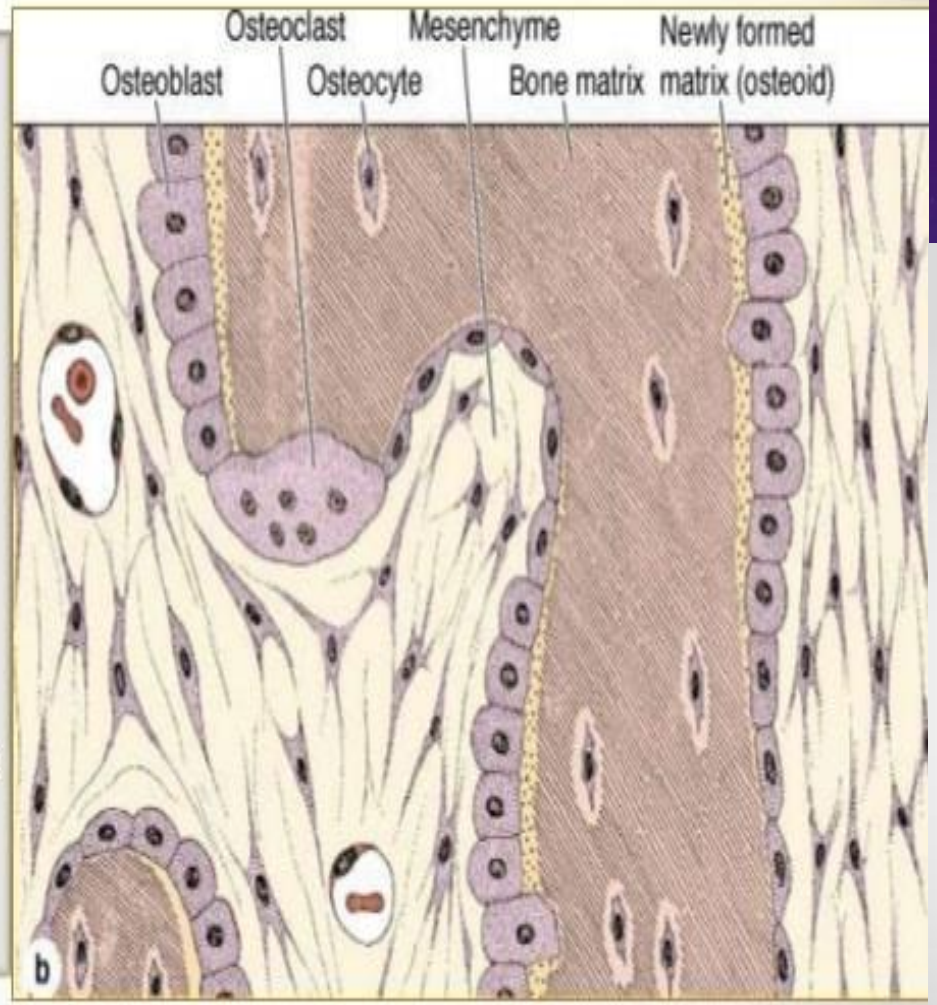
Functions:
1. Support;
2. Protection;
3. Movement;
4. Storage.

Bone Matrix

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

- **Inorganic Part:**

Calcium phosphate crystals called **hydroxyapatite:**



Bone Matrix

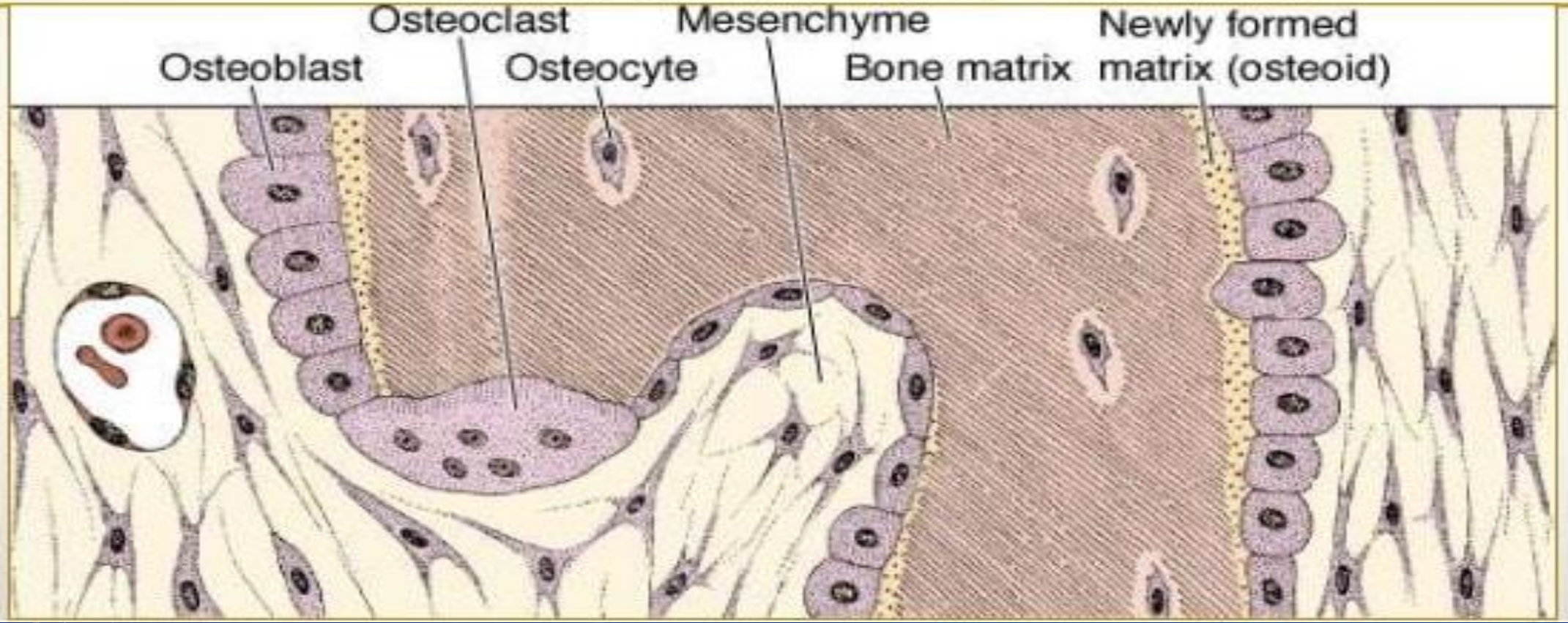
- The collagen and mineral components: Responsible for the major functional characteristics of bone.

Bone Cells

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

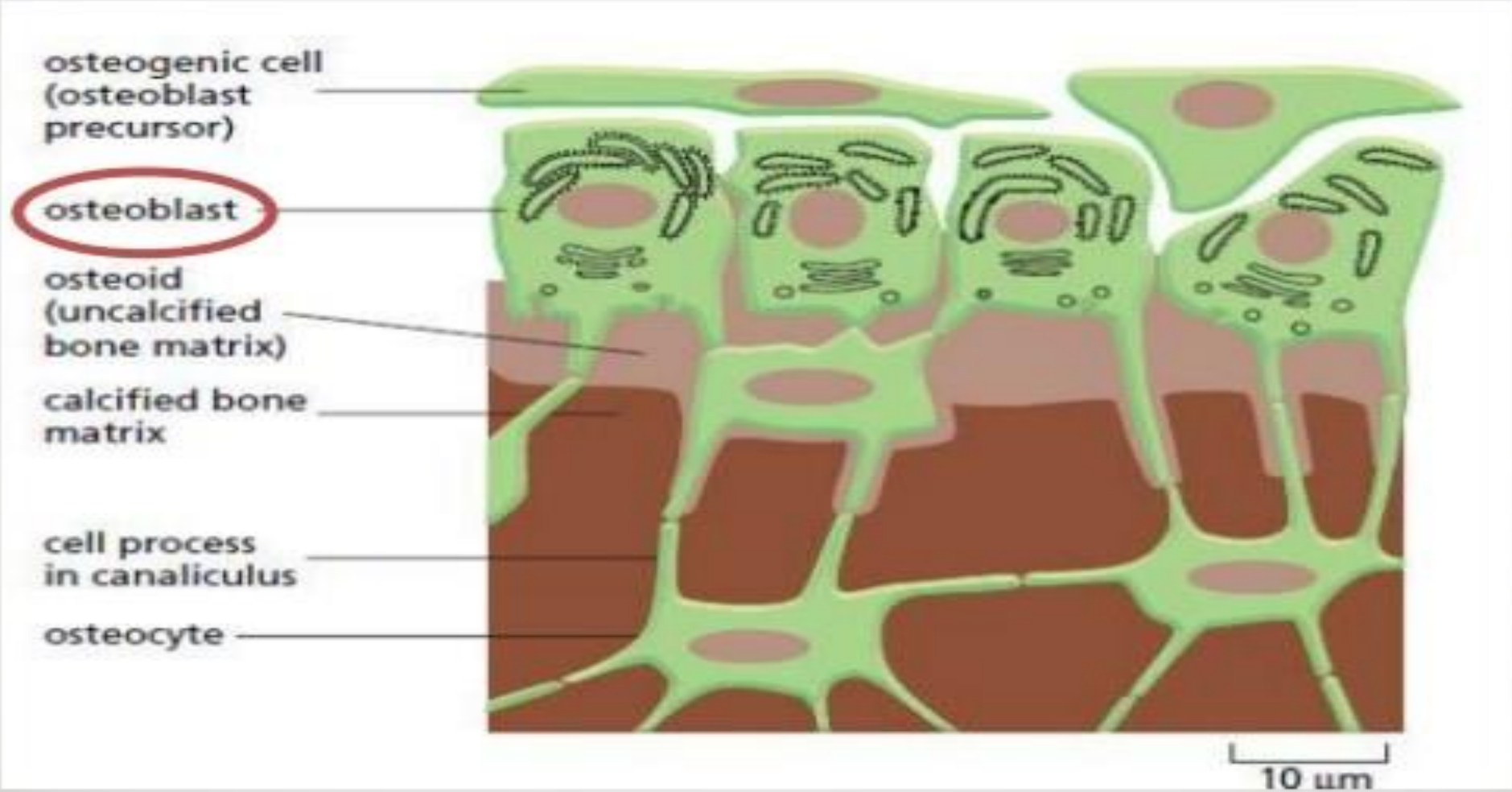
Bone cells are categorized as:

✓ **Osteoblasts; Osteocytes; Osteoclasts.**



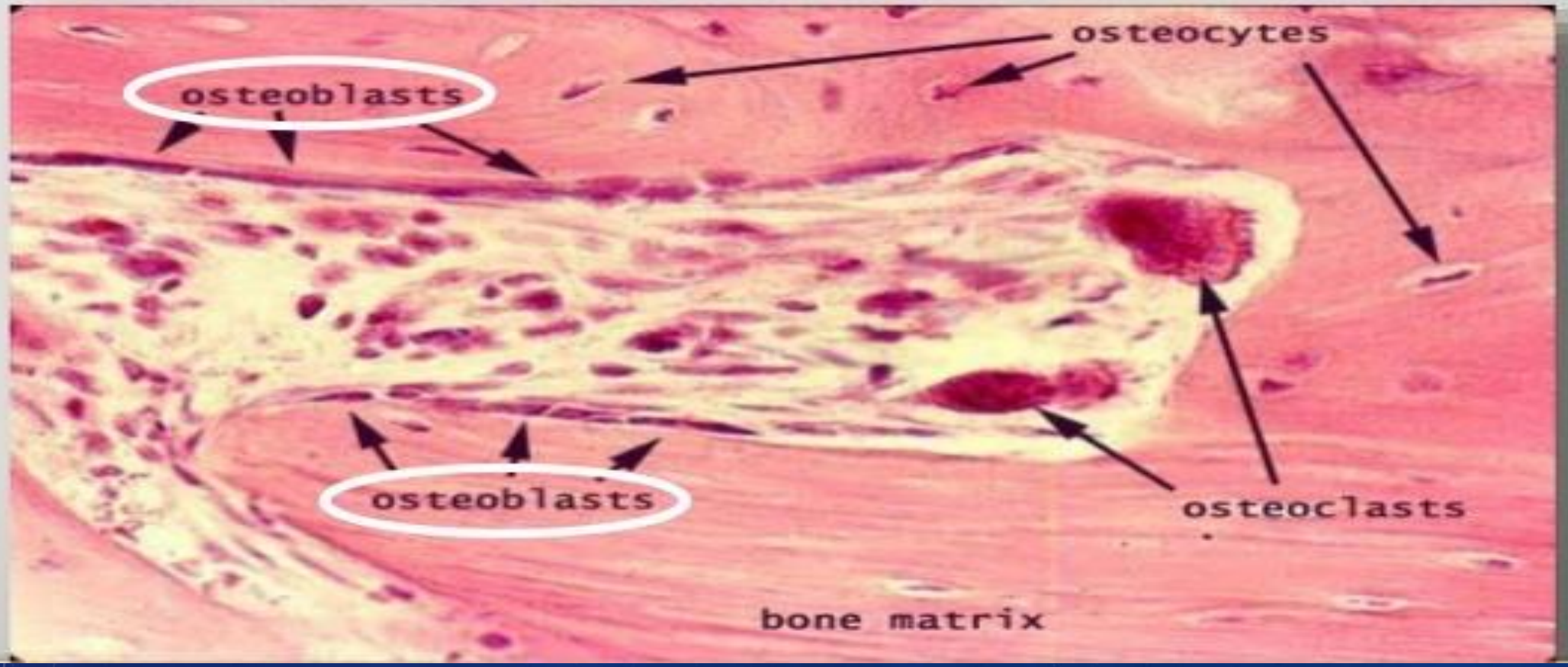
OSTEOBLASTS

- Extensive endoplasmic reticulum and numerous ribosomes;



OSTEOBLASTS

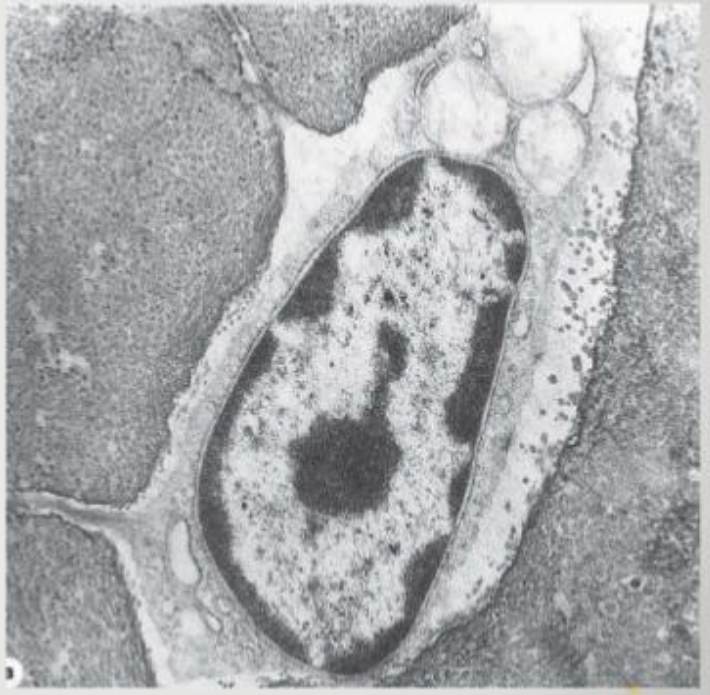
- Produce collagen and proteoglycans, which are packaged into vesicles by the Golgi apparatus and released from the cell by exocytosis;
- Form vesicles that accumulate calcium ions (Ca^{2+}), phosphate ions (PO^{2}_{4}), and various enzymes used to form hydroxyapatite crystals.



Osteocytes

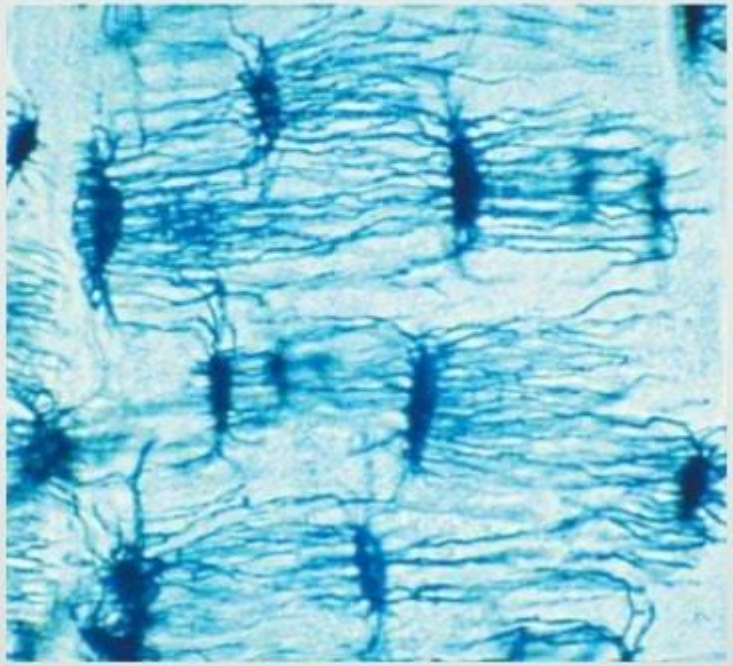
- They produce components needed to maintain the bone matrix;

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



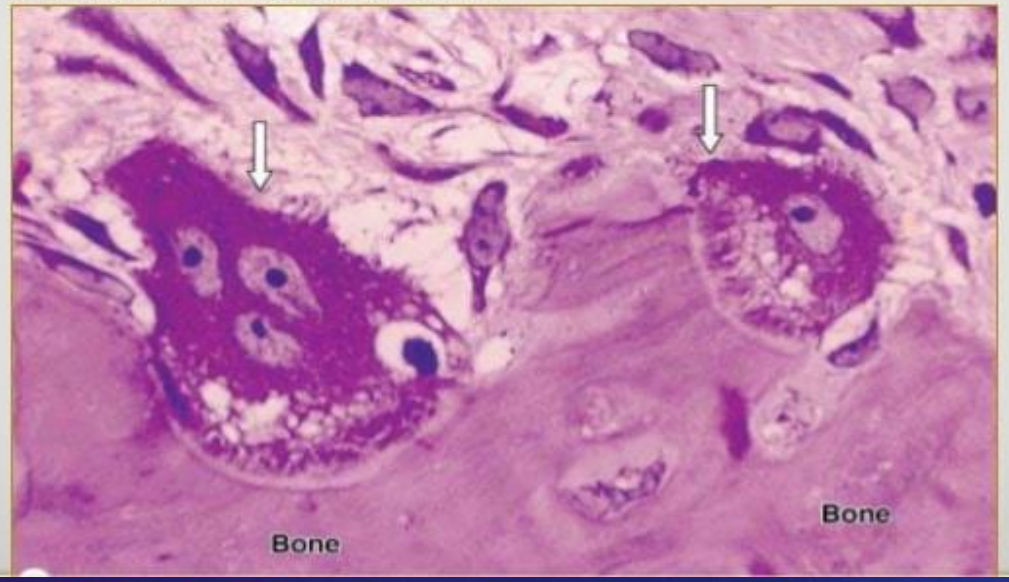
Osteocytes

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



Osteoclasts

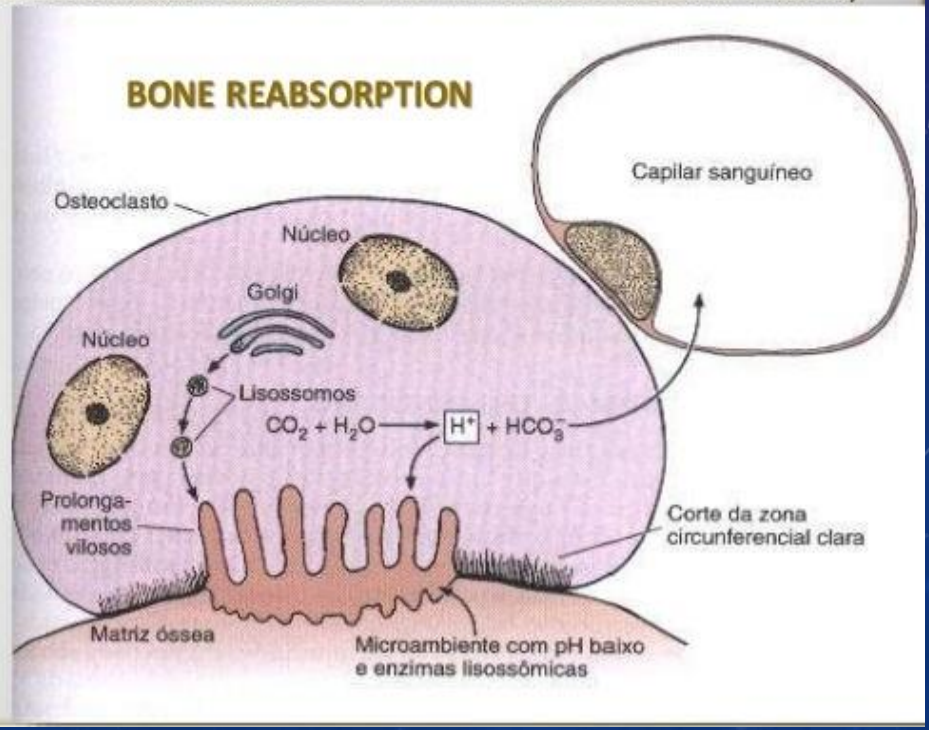
- Large cells with several nuclei;
- Responsible for the resorption, or breakdown of bone;
- **Ruffled border –** Projections where the plasma membrane of osteoclasts contacts bone matrix.



Osteoclasts

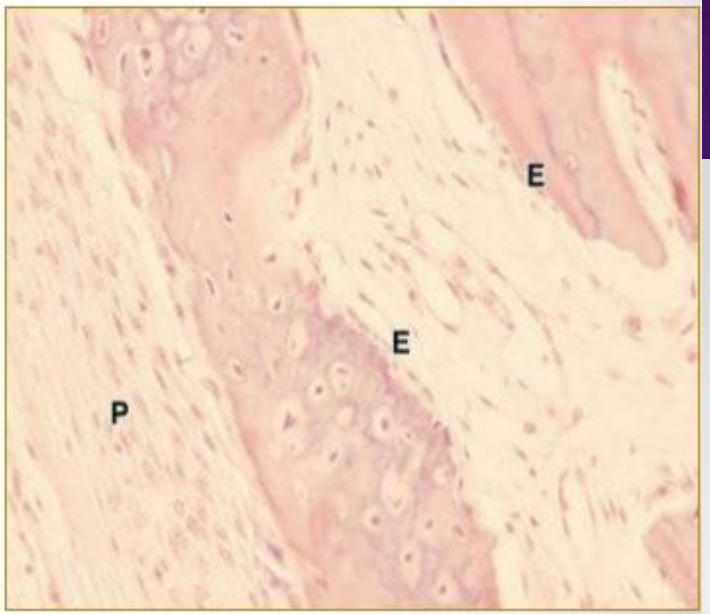
- Hydrogen ions are pumped across the ruffled border and produce an acid environment : Decalcification of the mineralized bone matrix;

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;**

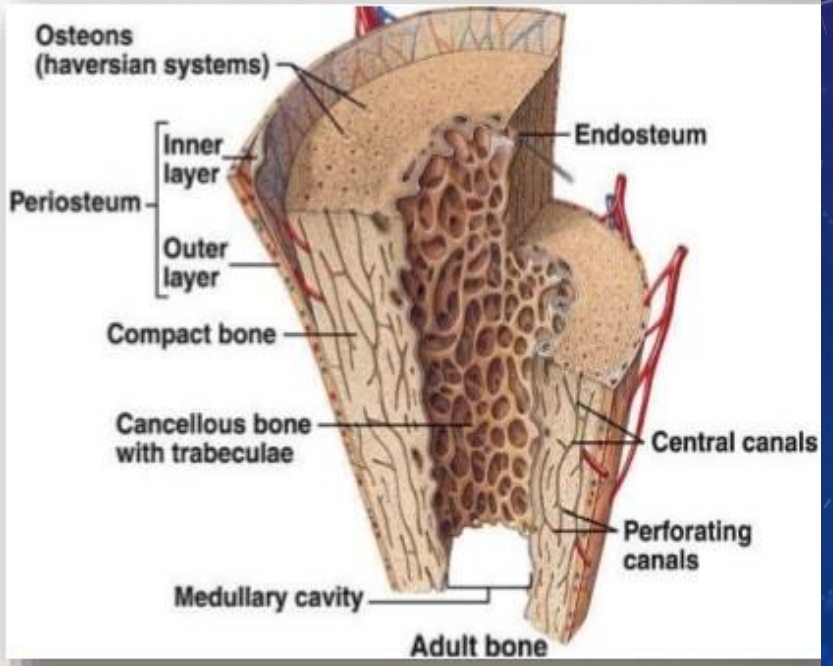
-**Collagen fibers** – Sharpey's fibers penetrate the bone and the periosteum hold firmly to the bone;

- **Fibroblasts.**

Periosteum e Endosteum

- Inner layer: **Endosteum**

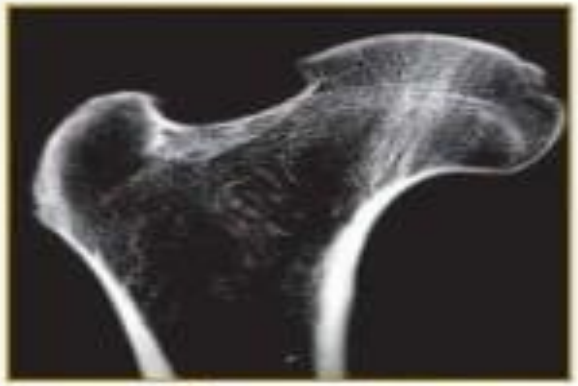
- **Osteogenic flattened cells** : Cover the trabecular bone cavity , the medullar channel, and Volkmann channel.



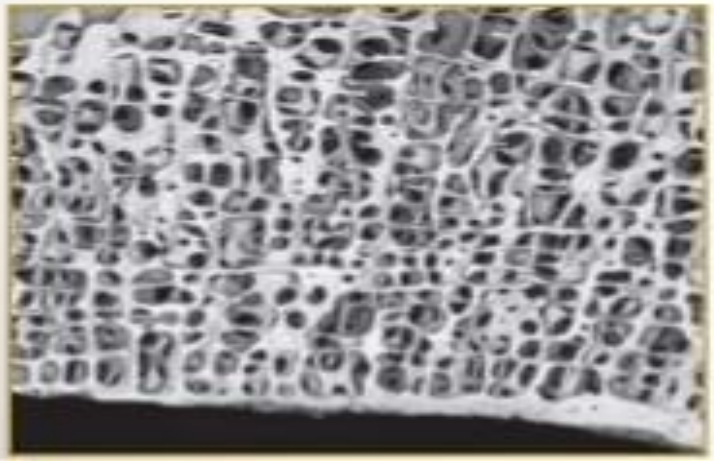
The aim of both layers is to promote the nutrition of bone tissue and provide new osteoblasts for bone growth and fracture repair.

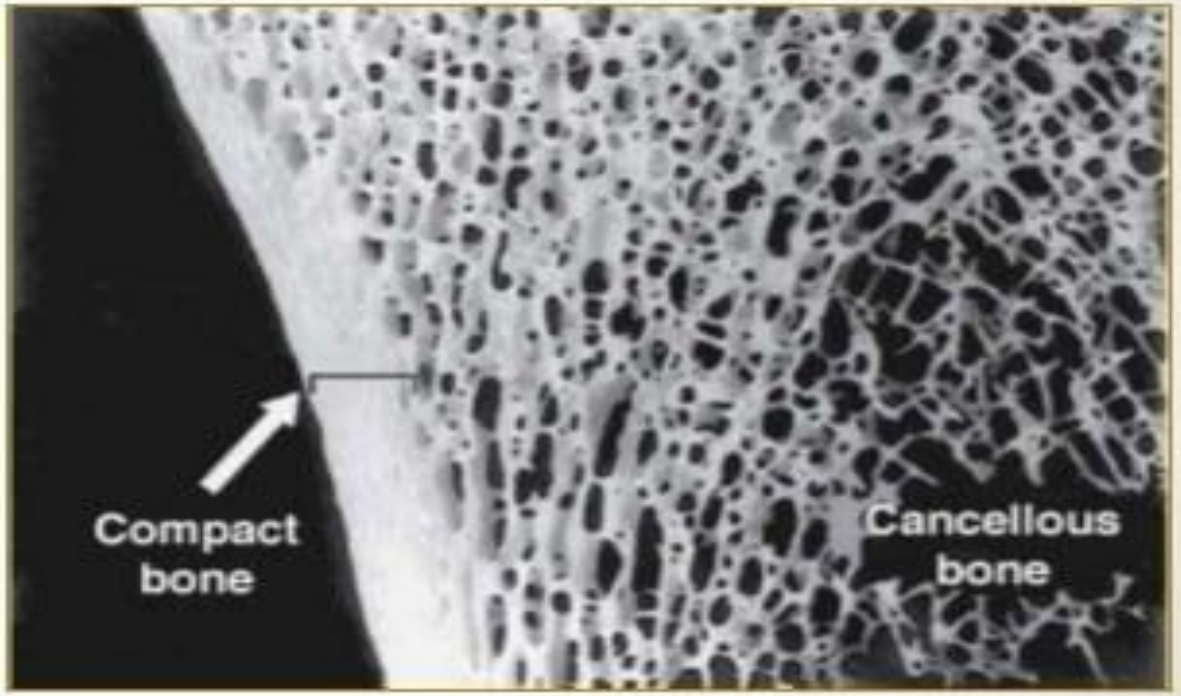
Types of Bone Tissue

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



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





A bone profile blood test measures minerals, proteins, and enzymes within the bones.
This test is crucial for monitoring bone health, ensuring proper repair and development.
As we age, maintaining healthy bones becomes increasingly important.
Bone-specific alkaline phosphatase (Bone ALP or BALP).
This is an estimate of the rate of bone formation over your entire skeleton. ...
Osteocalcin.
This is another marker of bone formation.
Urinary N-telopeptide of type I collagen.
Vitamin D levels.

Bone Profile Interpretation

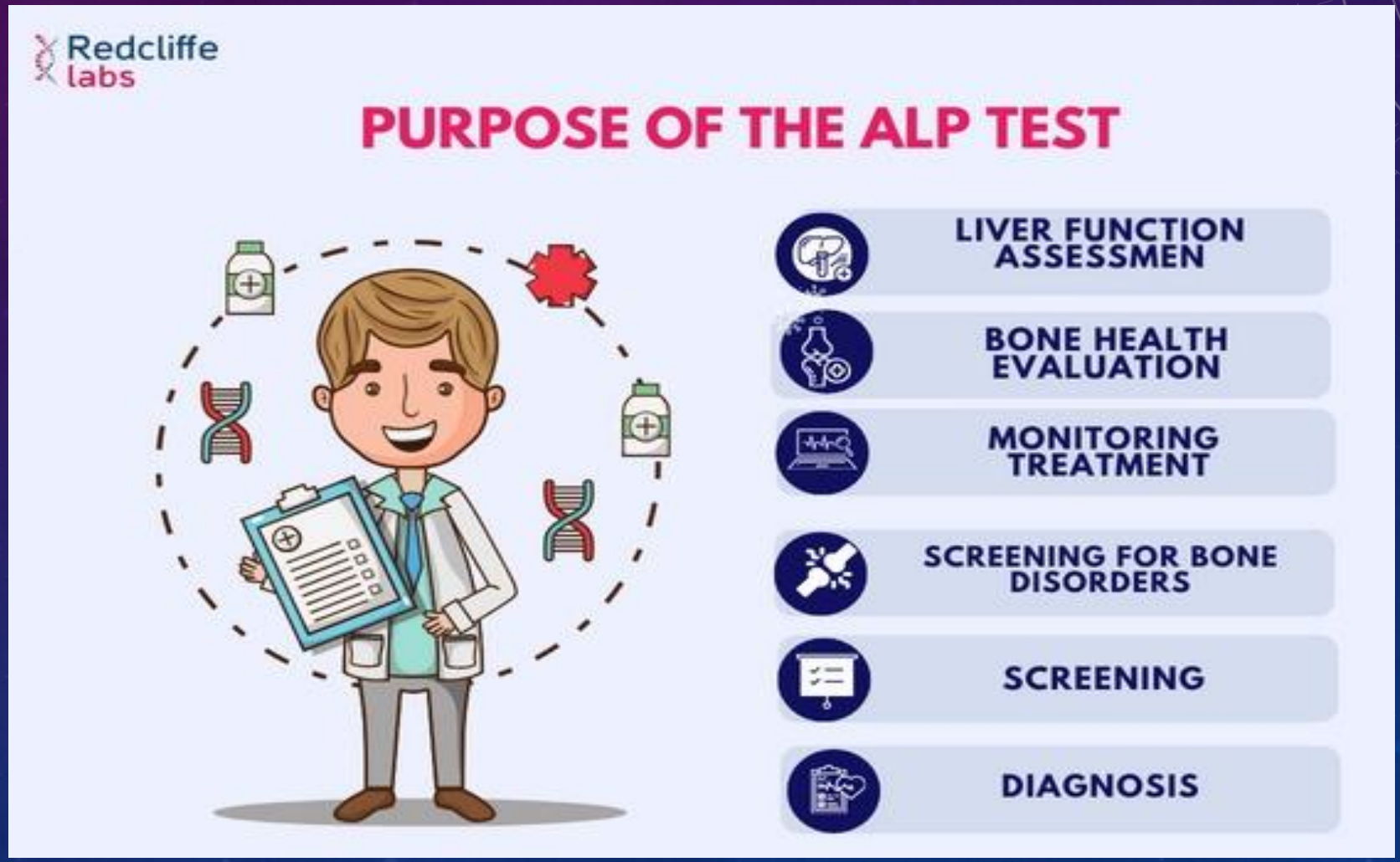
	Calcium	Phosphate	Alkaline Phosphatase (ALP)	Parathyroid Hormone (PTH)
Osteogenesis Imperfecta	Normal	Normal	Normal	Normal
Osteoporosis	Normal	Normal	Normal	Normal
Osteomalacia	↓	↓	↑	↑
Paget's disease of bone	Normal	Normal	↑ (Isolated raised ALP)	Normal
Primary hyperparathyroidism	↑	↓	↑	↑
Secondary hyperparathyroidism	↓	↑	↑	↑
Tertiary hyperparathyroidism	↑	↑	↑	↑
Hypoparathyroidism	↑	↑	↑	↓

Vitamin D Levels

30 ng/mL	Considered adequate and healthy
20 ng/mL - 30 ng/mL	Considered vitamin D insufficient
Less than 20 ng/mL	Vitamin D deficient
Greater than 80 ng/mL	Associated with toxic side effects

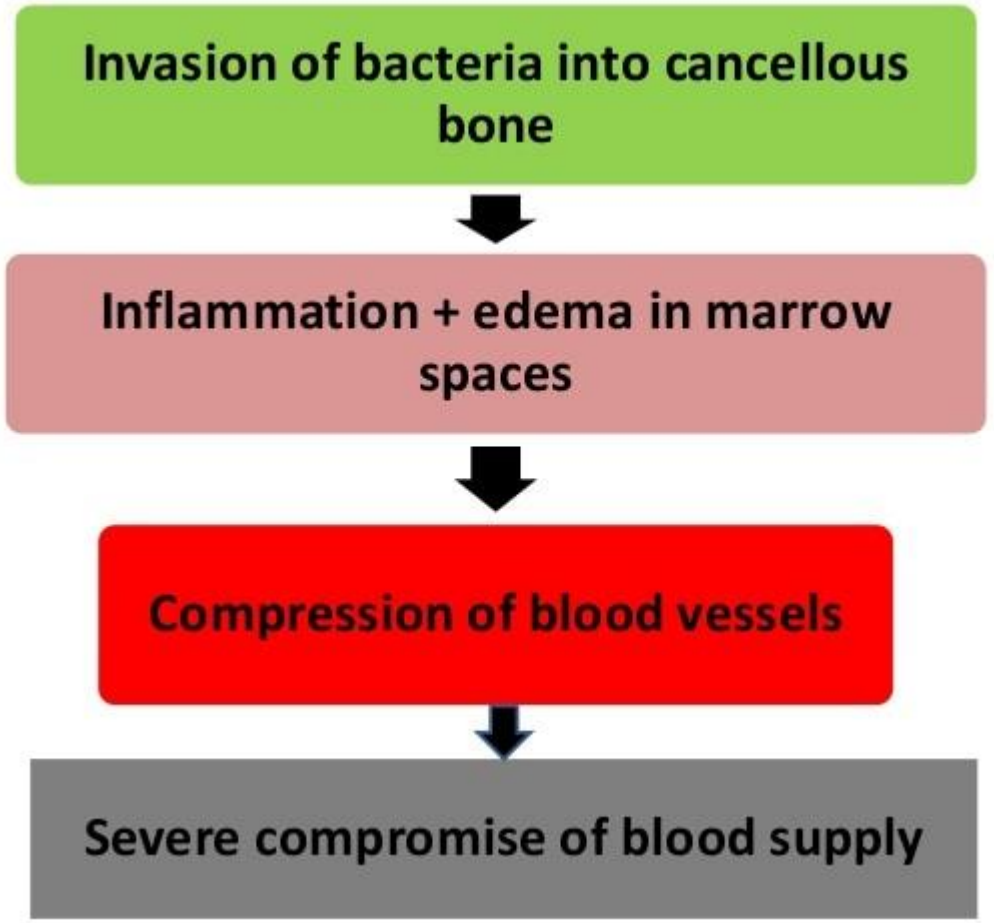
Normal Value

The normal range is 44 to 147 international units per liter (IU/L) or 0.73 to 2.45 microkatal per liter (µkat/L). Normal values may vary slightly from laboratory to laboratory. They also can vary with age and sex.

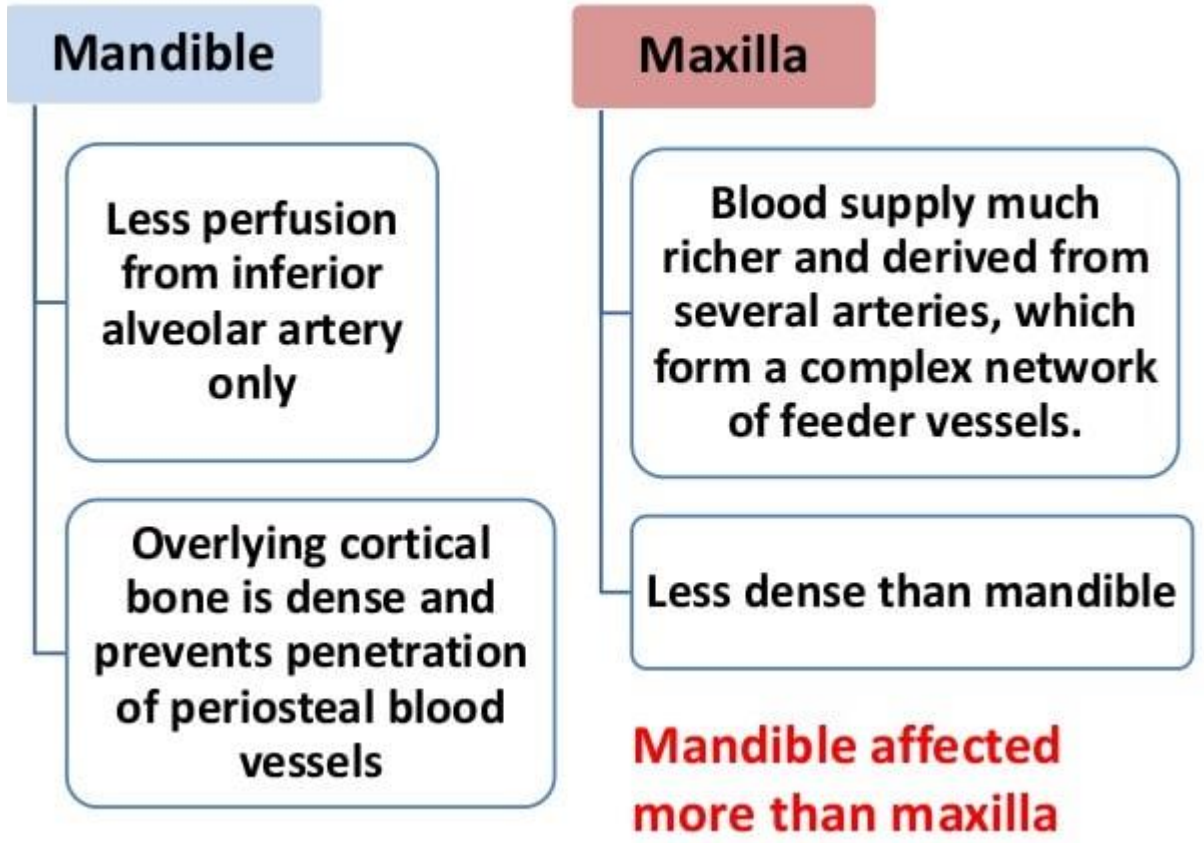


Definition

- 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.



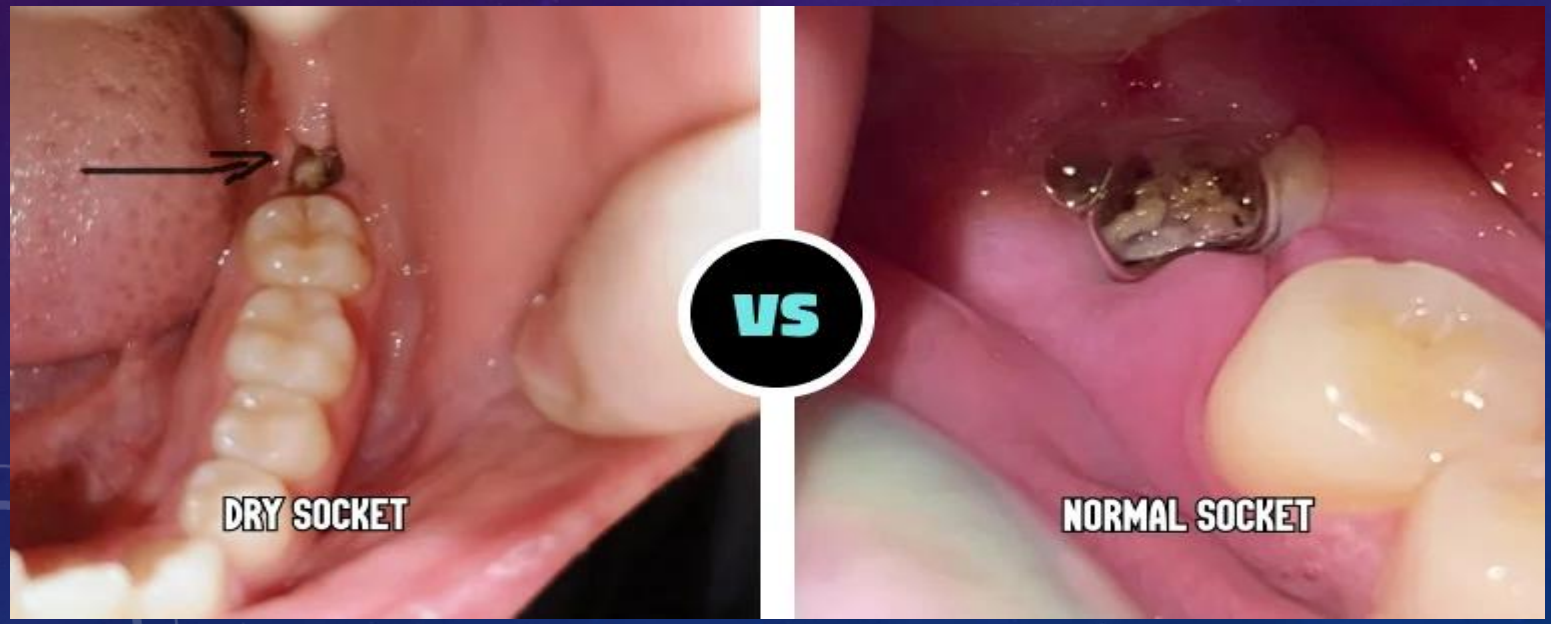
Bone diseases can make bones easy to break.
Different kinds of bone problems include:

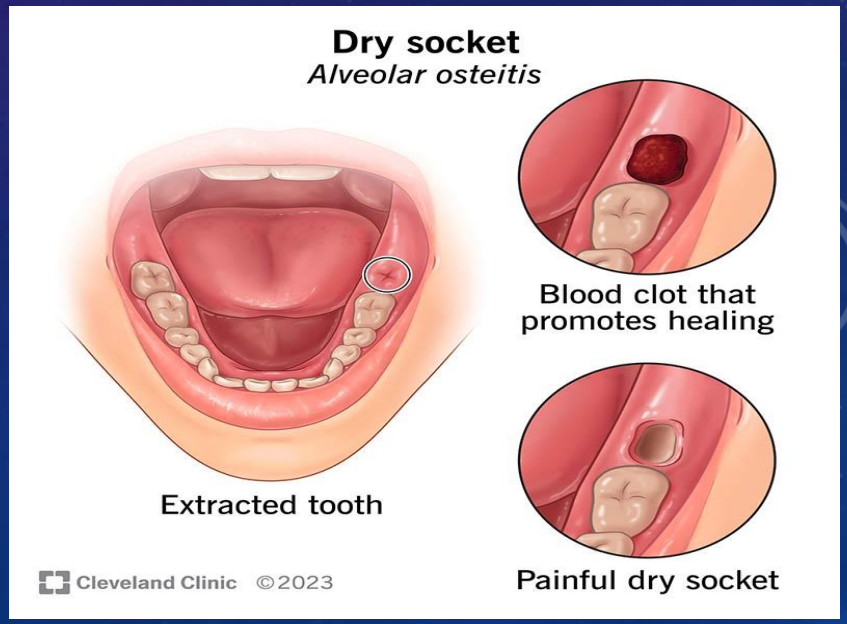
- Low bone density and osteoporosis, which make your bones weak and more likely to break
- Osteogenesis imperfecta makes your bones brittle
- Paget's disease of bone makes them weak
- Bones can also develop cancer and infections
- Other bone diseases, which are caused by poor nutrition, genetics, or problems with the rate of bone growth or rebuilding

-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**).

Dry socket is a **painful dental condition that sometimes happens after you have a tooth removed**. Having a tooth removed is called an extraction..





Symptoms of dry socket may include:

Severe pain within a few days after removing a tooth. Loss of part or all of the blood clot at the tooth removal site. The socket may look empty.

Treatment may include:

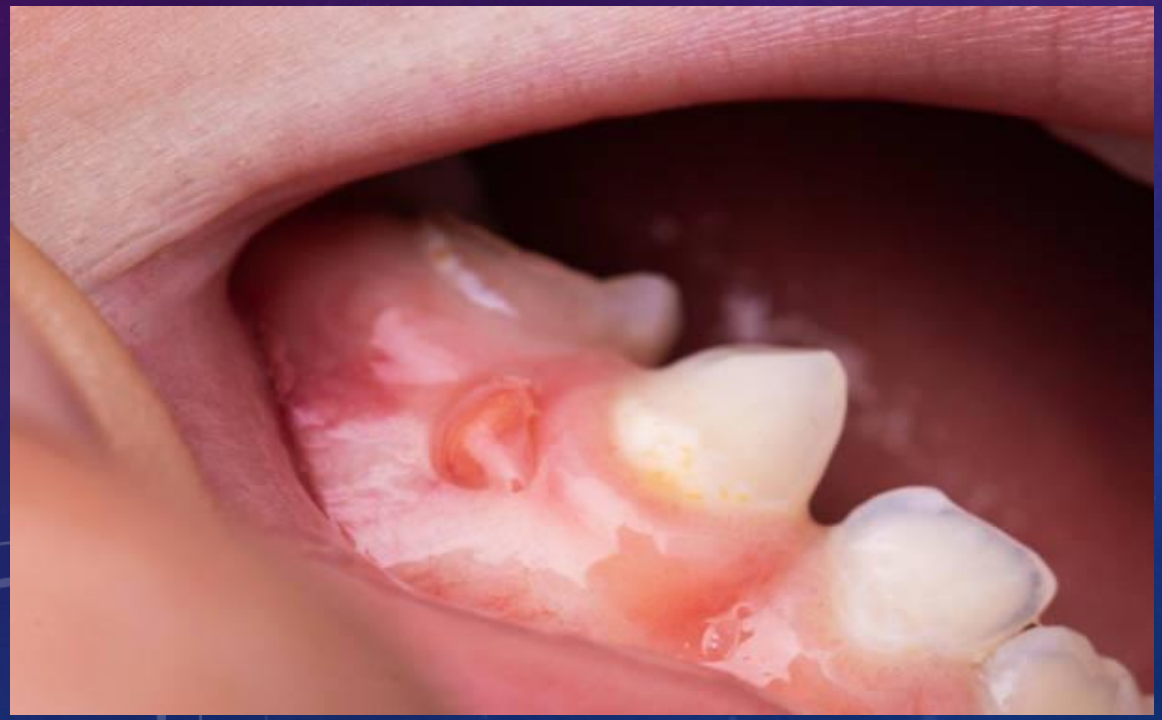
1.Flushing out the socket. flush out the socket to remove any food bits or other loose materials that may add to pain or possible infection.

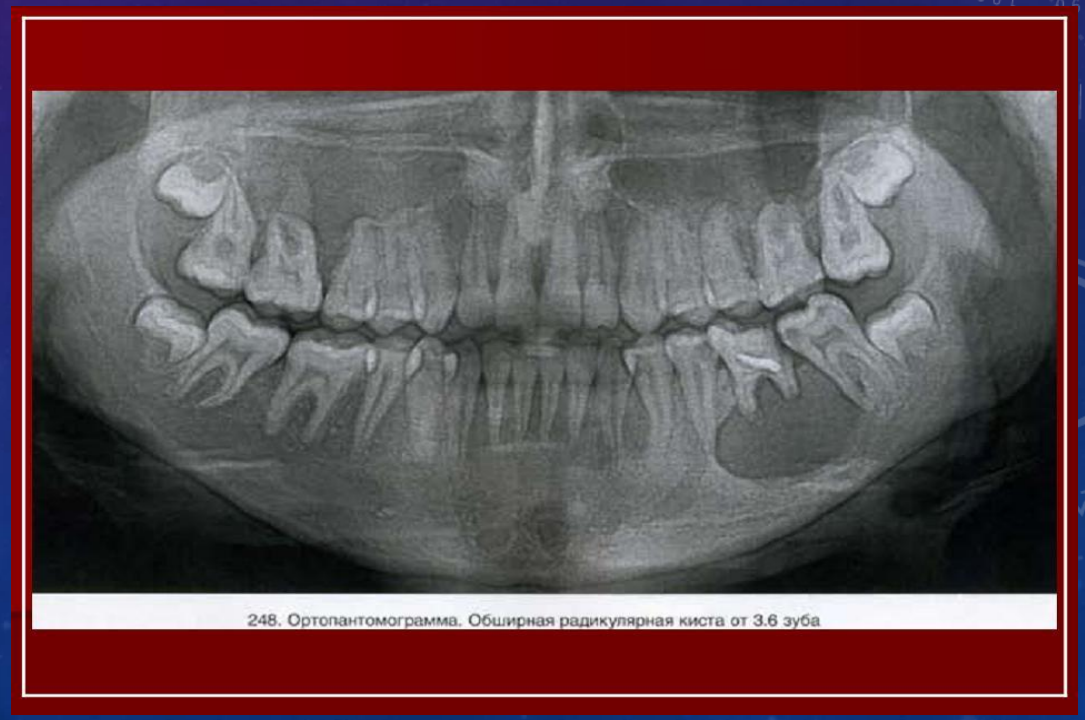
2.Dressing with medicine.

3.Pain medicine.

4.Self-care.

-Periostitis: - inflammation of the periosteal spaces of the bone and may not be associated with osteomyelitis.
The distinction between osteomyelitis and periostitis cannot always be made in dry bones. However, in periostitis there will be no cloacae, involucrum, or changes in the marrow cavity.





Osteoarthritis

Most common arthritis and is prevalent in adults +45 years.

Pathophysiology

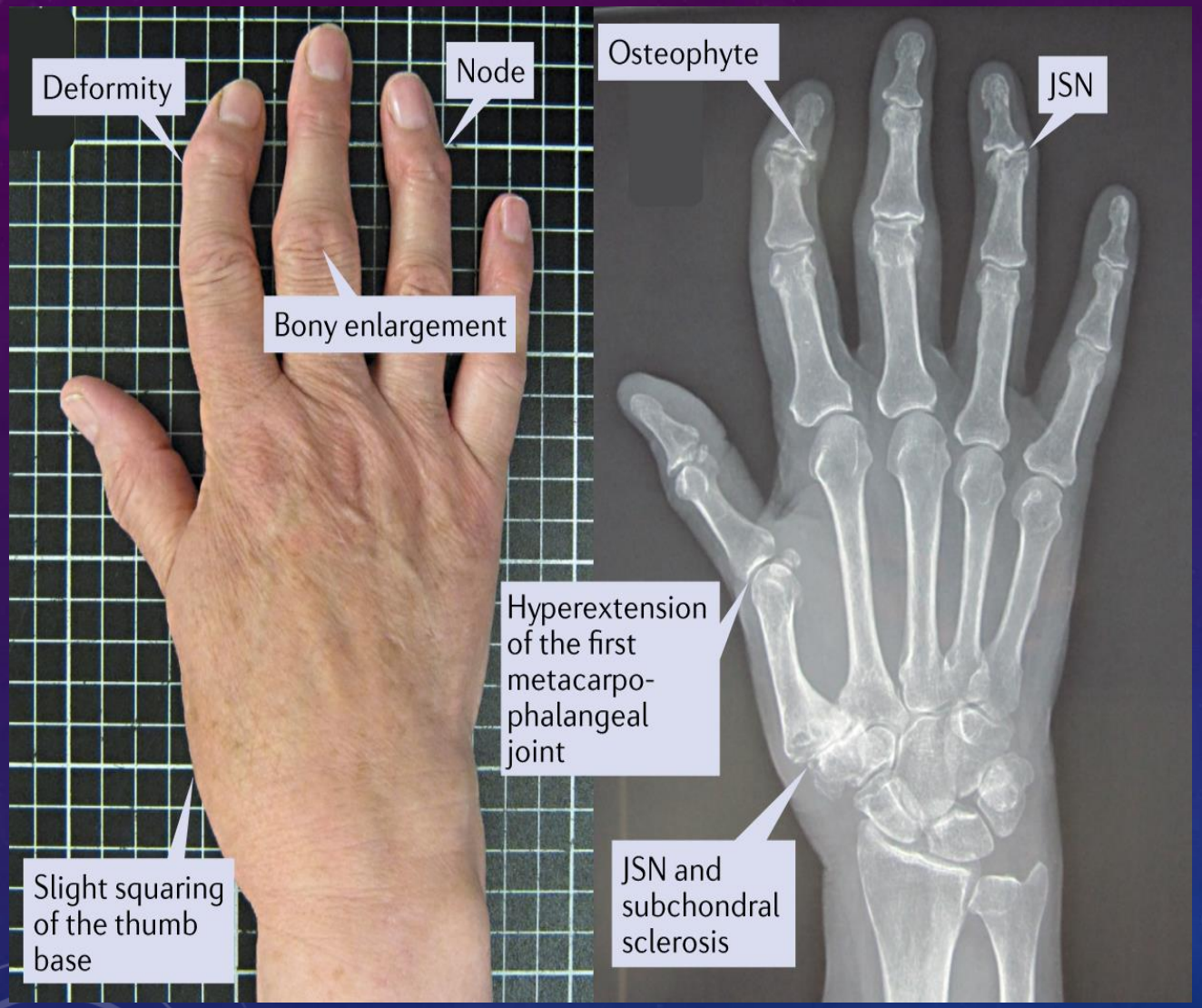
Osteoarthritis (OA) is where there is loss joint of space and the articular surface is worn down. This occurs due to release of enzymes that break down the collagen and proteoglycan causing destruction of the articular cartilage. The underlying subchondral bone becomes exposed and remodelling occurs causing formation of osteophyte.

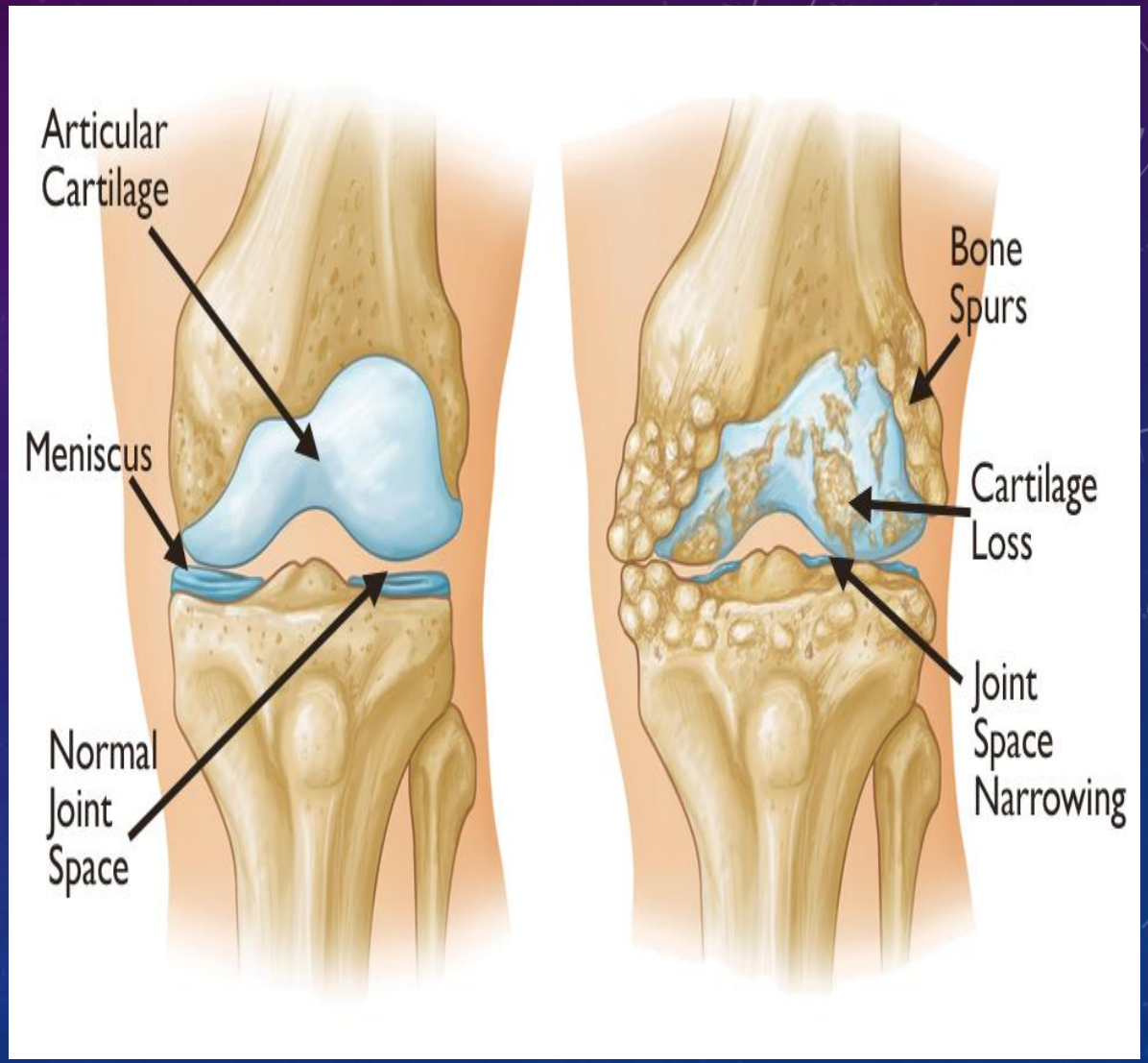
clinical features

Pain , Stiffness, Slow progression of loss of function. Deformity of knees, hips or larger joints ,Decrease in joint flexibility , Bony lumps on the fingers

Investigations

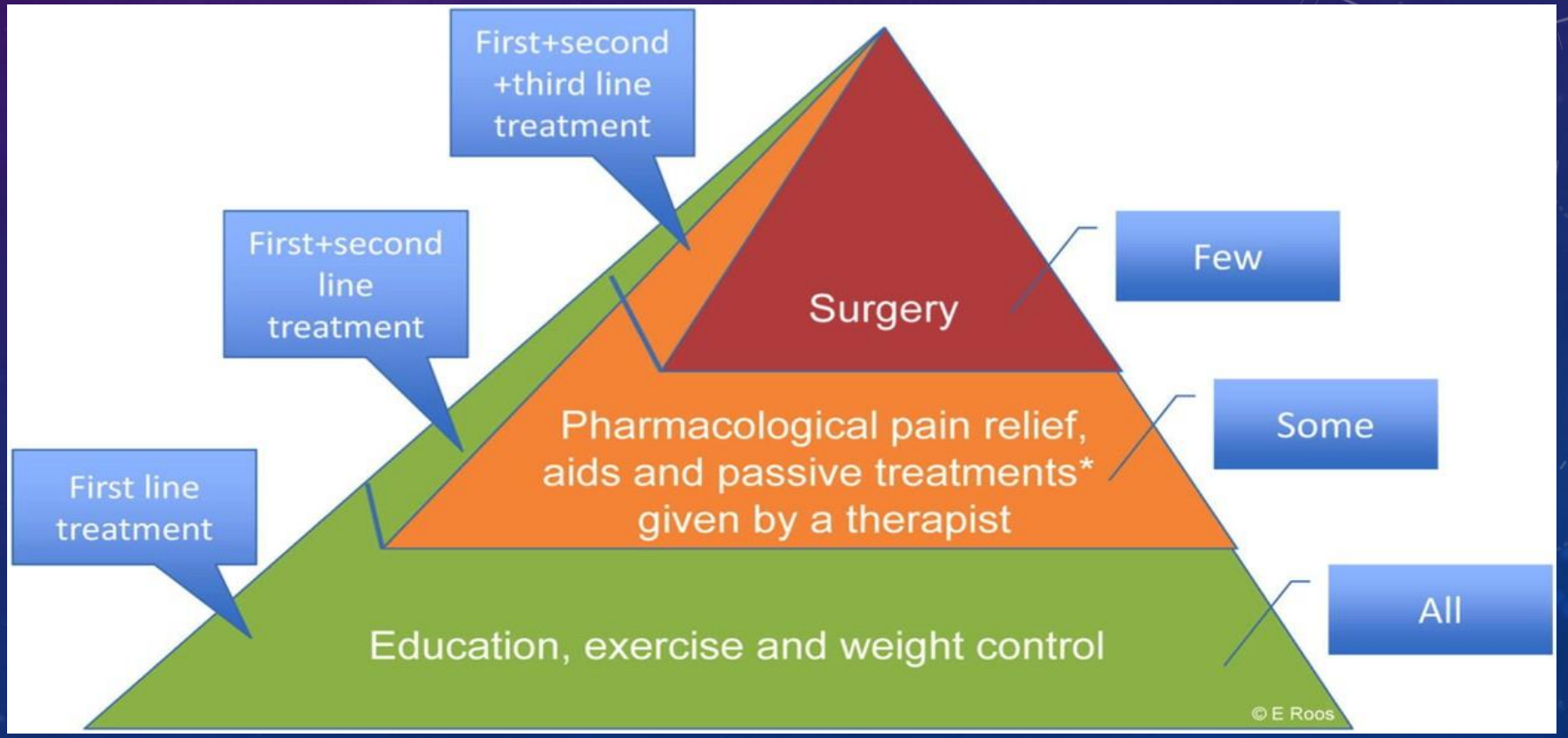
Radiographs: can show the reduced joint space, formation of osteophyte, erosion of the subchondral bone, bone cysts beneath the joint and deformities.
Blood tests may be carried out to rule out rheumatoid arthritis.


Deformity
Node
Bony enlargement
Slight squaring of the thumb base
Osteophyte
JSN
Hyperextension of the first metacarpo-phalangeal joint
JSN and subchondral sclerosis


Articular Cartilage
Meniscus
Normal Joint Space
Bone Spurs
Cartilage Loss
Joint Space Narrowing

Treatment:

Surgery
Physiotherapy
Exercise
Pharmaceutical drug
Nonsteroidal anti-inflammatory drug
Weight loss
Analgesics
Arthroplasty
Joint injections



Paget's disease (Osteitis deformans)

It is characterized by enlargement and thickening of bone, the internal architecture is abnormal and the bone is unusually brittle.

Causes:

The cause is unknown, although the discovery of inclusion bodies in the osteoclasts has suggested a viral infection (parvovirus).

Pathology:

The characteristic cellular change is a marked increase in osteoclastic and osteoblastic activity, bone turnover is accelerated, plasma alkaline phosphatase is raised (a sign of osteoblastic activity) and there is increases excretion of hydroxyproline in the urine (due to osteoclastic activity).

Stages:

--- Early osteoclastic stage.

--- Late osteoblastic stage.

Clinical features: the pelvis and tibia being the commonest sites. Most patients are asymptomatic. The disorder being discovered when an x-ray is taken for some unrelated conditions or after the incidental discovery of raised serum alkaline phosphatase. Pain and deformity may be present. The skull base may become flattened (platybasia), kyphosis and spinal stenosis also may occur, cranial nerve compression may lead to impaired vision, facial palsy, and deafness. Steal syndrome, in which blood is diverted from internal organs to the surrounding skeletal circulation.

X-ray: bone appears thick, sclerotic, and with coarse trabeculations.

Pathological fractures may be seen.

Investigations:

--- Serum calcium and phosphate are usually normal.

--- Serum alkaline phosphatase is increased.

--- 24 hr urinary excretion of pyridinoline cross-links is a good indicator of disease activity and bone resorption

The cotton wool appearance is a plain film sign of Paget disease and results from thickened, disorganized trabeculae which lead to areas of sclerosis in a previously lucent area of bone, typically the skull.

The presence of irregularly shaped bone particles appears like jigsaw-puzzle pieces and is the hallmark feature of Paget disease. As the disorder advances, the osteoblastic phase becomes dominant, resulting in excessive bone formation, which is fibrous and coarse.

Medications. Osteoporosis drugs (bisphosphonates) are the most common treatment for Paget's disease of bone. Bisphosphonates are typically given by injection into a vein, but they can also be taken by mouth. When taken orally, bisphosphonates are generally well tolerated but can irritate the stomach.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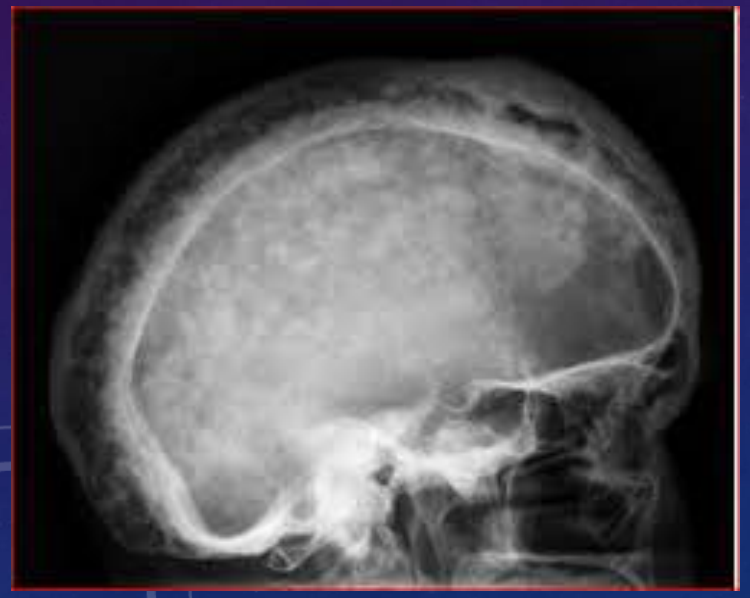
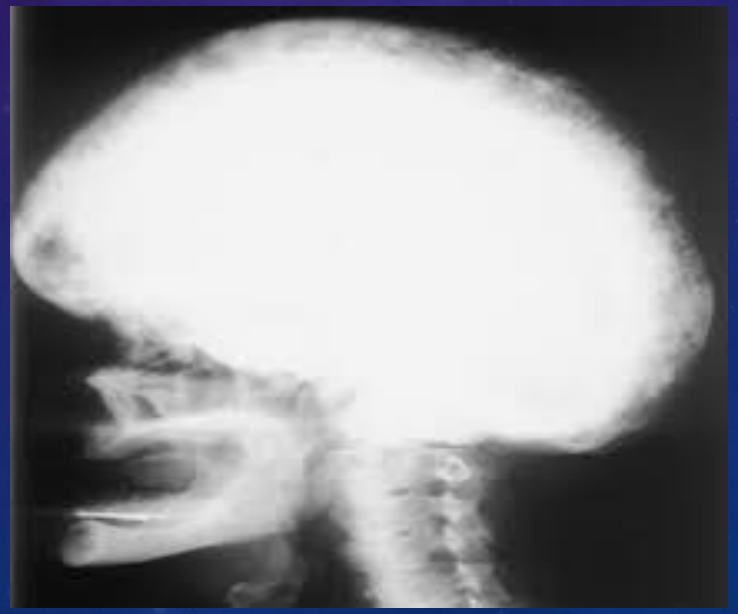
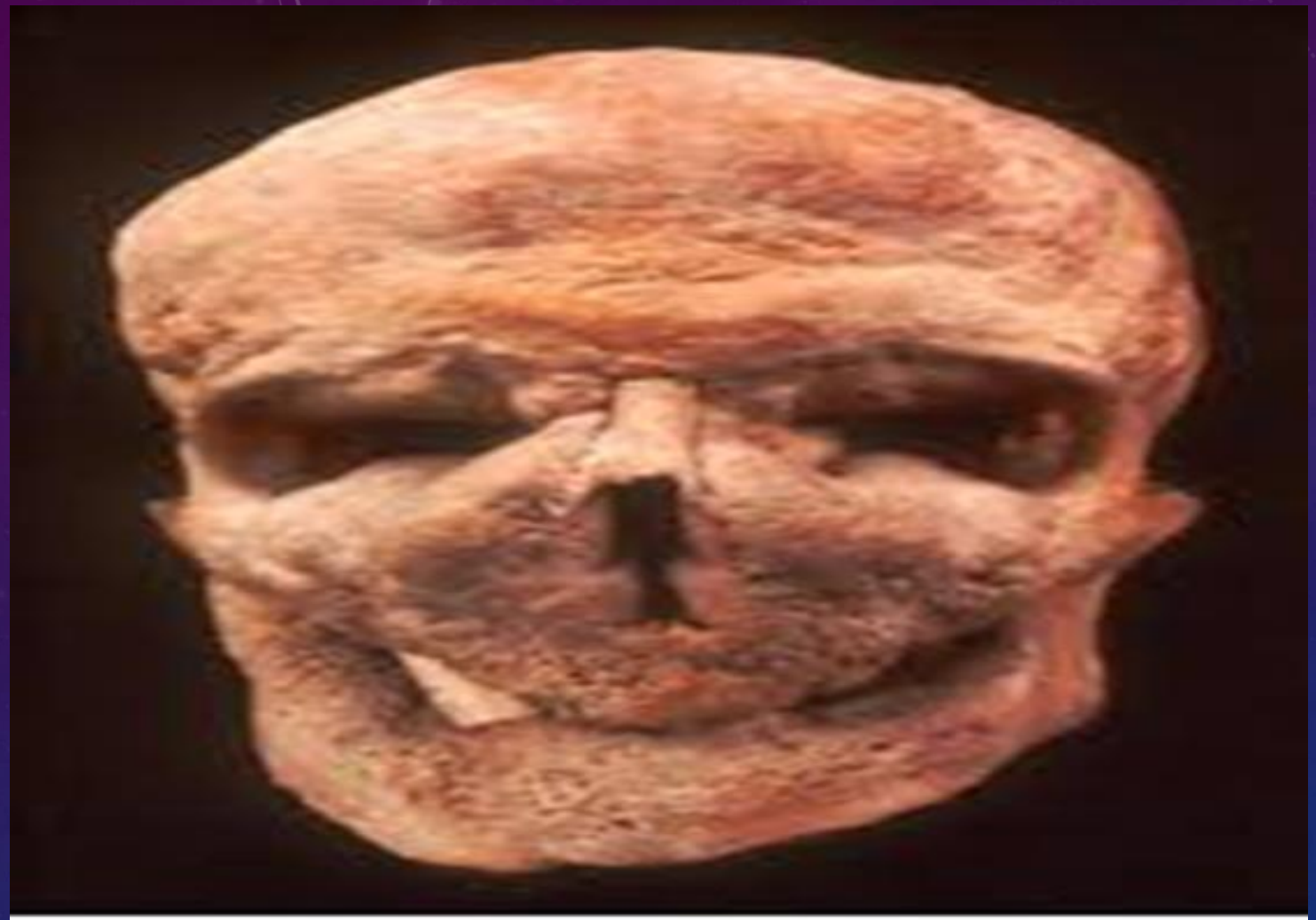





YASHODA
HOSPITALS
Paget's Disease



FACIAL BONE that is grossly thickened and symmetrically deformed may result from Paget's disease, a condition in which bone grows drastically out of control. The skull of the warrior Egil, a hero of the Icelandic sagas, may have looked similar to this one of a man who suffered from the disease several centuries ago.

Osteomalacia and Rickets

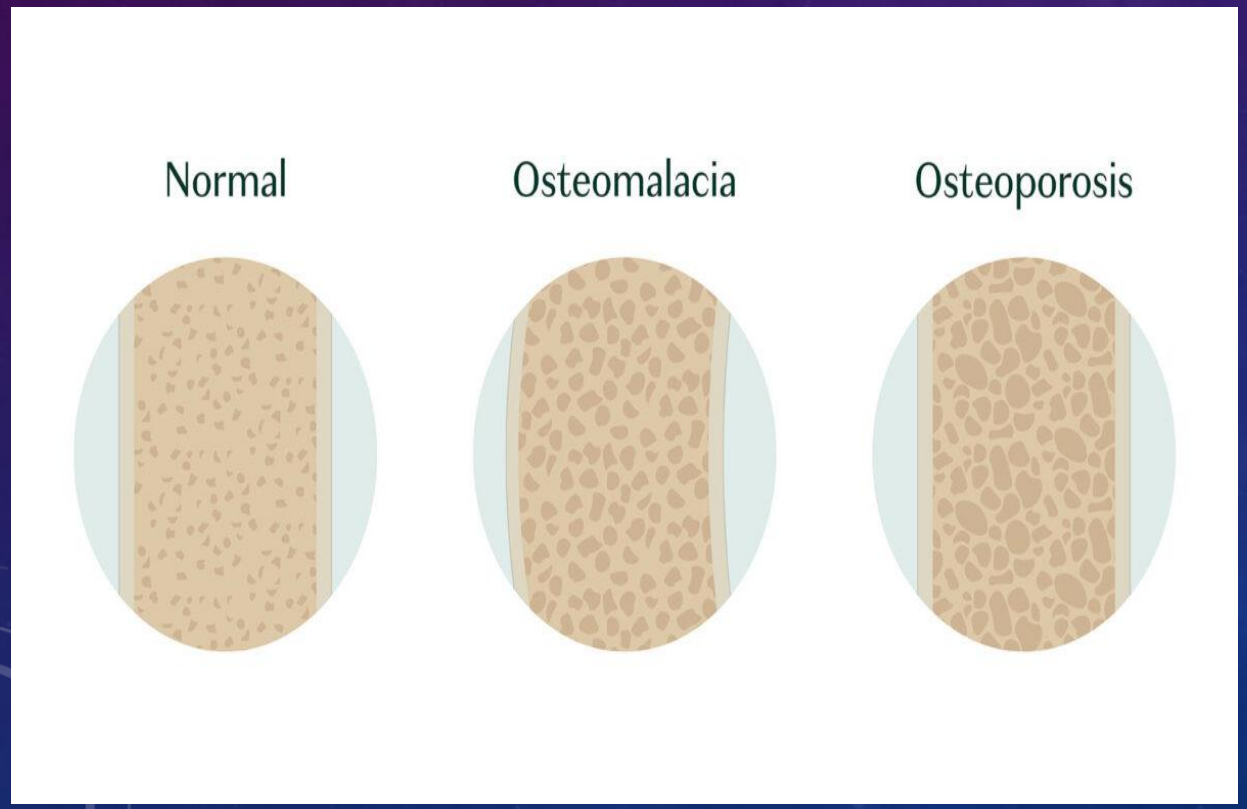
Rickets present in childhood and it is where the bone is unmineralized.

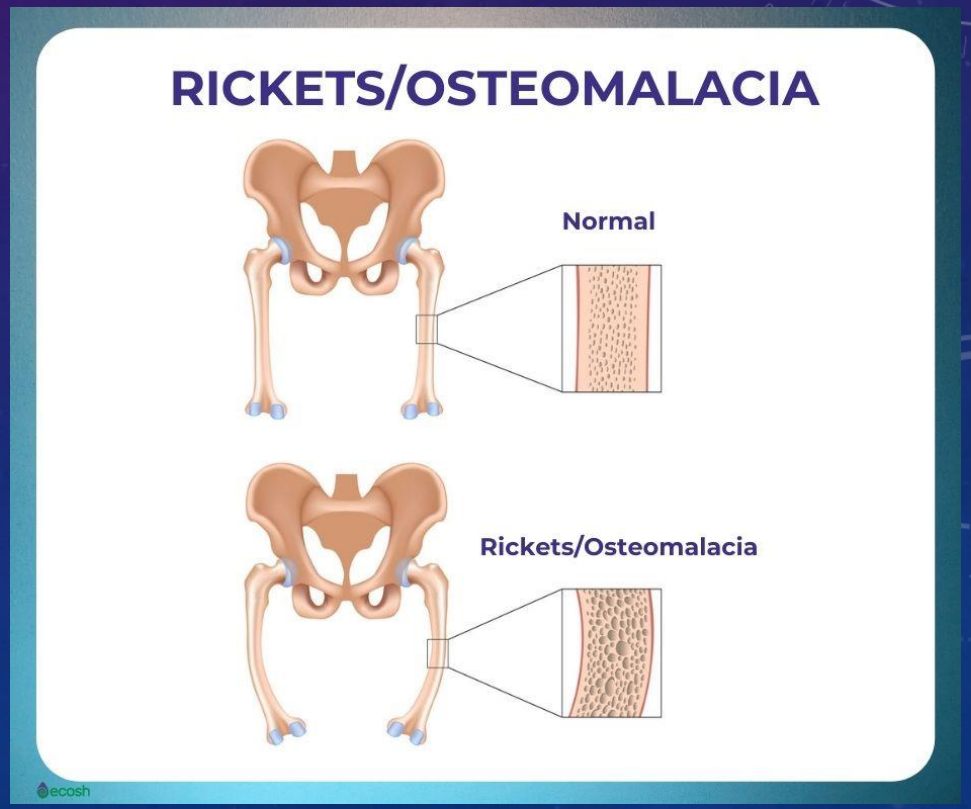
The difference between Osteomalacia and rickets is that rickets is seen in children where there growth plates are open so prone to disorders whereas, adults growth plates have fused.

Both conditions are characterised by low ratio of mineralised: unmineralized of the bone.

Pathophysiology

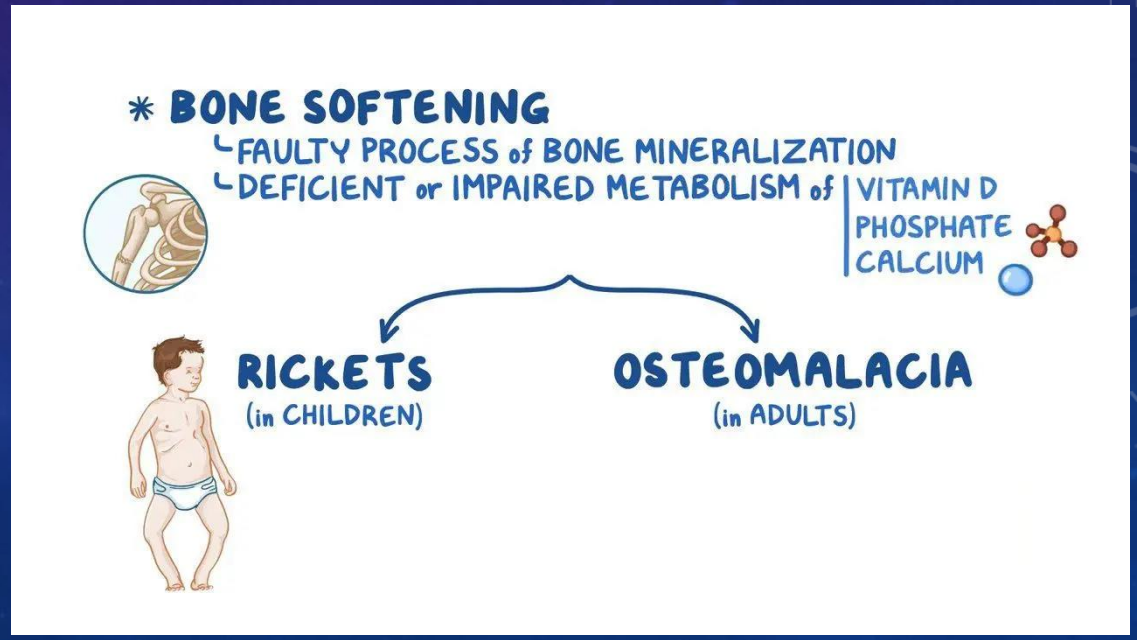
Osteomalacia and rickets are caused by vitamin D deficiency.





Osteomalacia causes soft bones due to a lack of vitamin D. It is a disorder of decreased mineralization, which results in bone breaking down faster than it can re-form. In osteoporosis, bone mass decreases over time, leading to weakened and brittle bones that are susceptible to fracture.

The adult form of rickets that causes soft bones – treatment with supplements will usually cure the condition. However, it may be several months before any bone pain and muscle weakness is relieved. continue taking vitamin D supplements regularly to prevent the condition returning.



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.

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

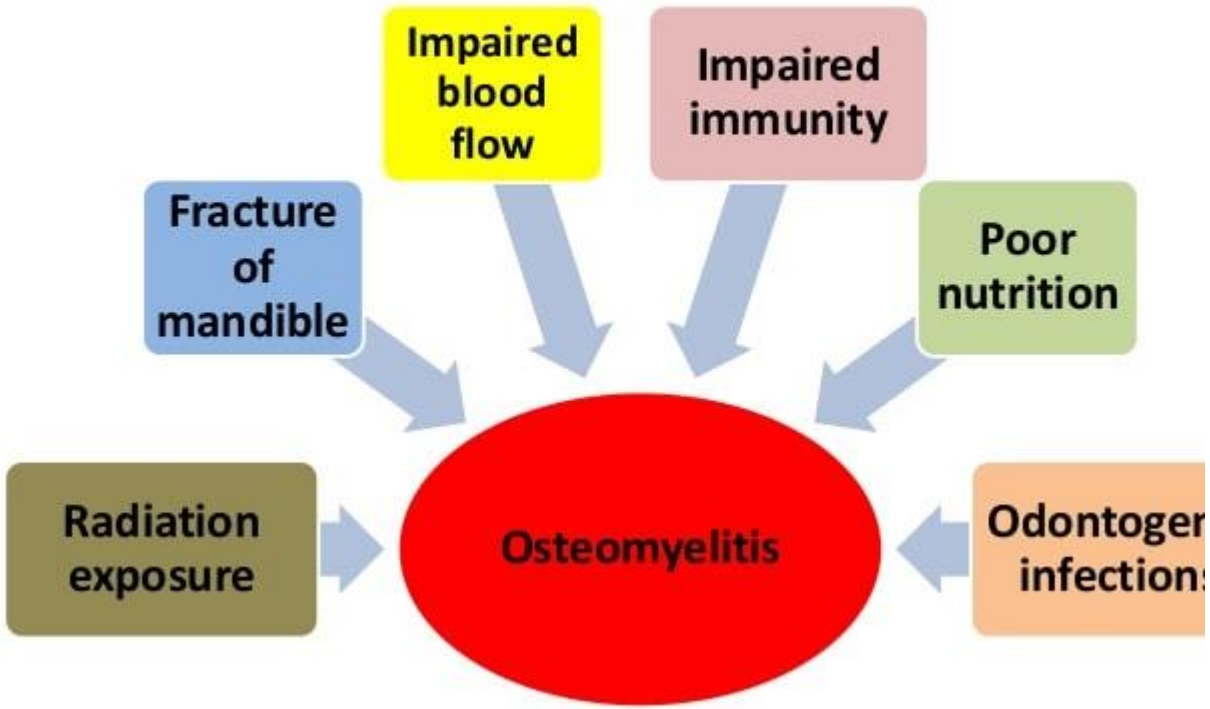
↓

Local factors decreases the vitality of bone

↓

Systemic conditions comprises the defense system of the host

Predisposing factors

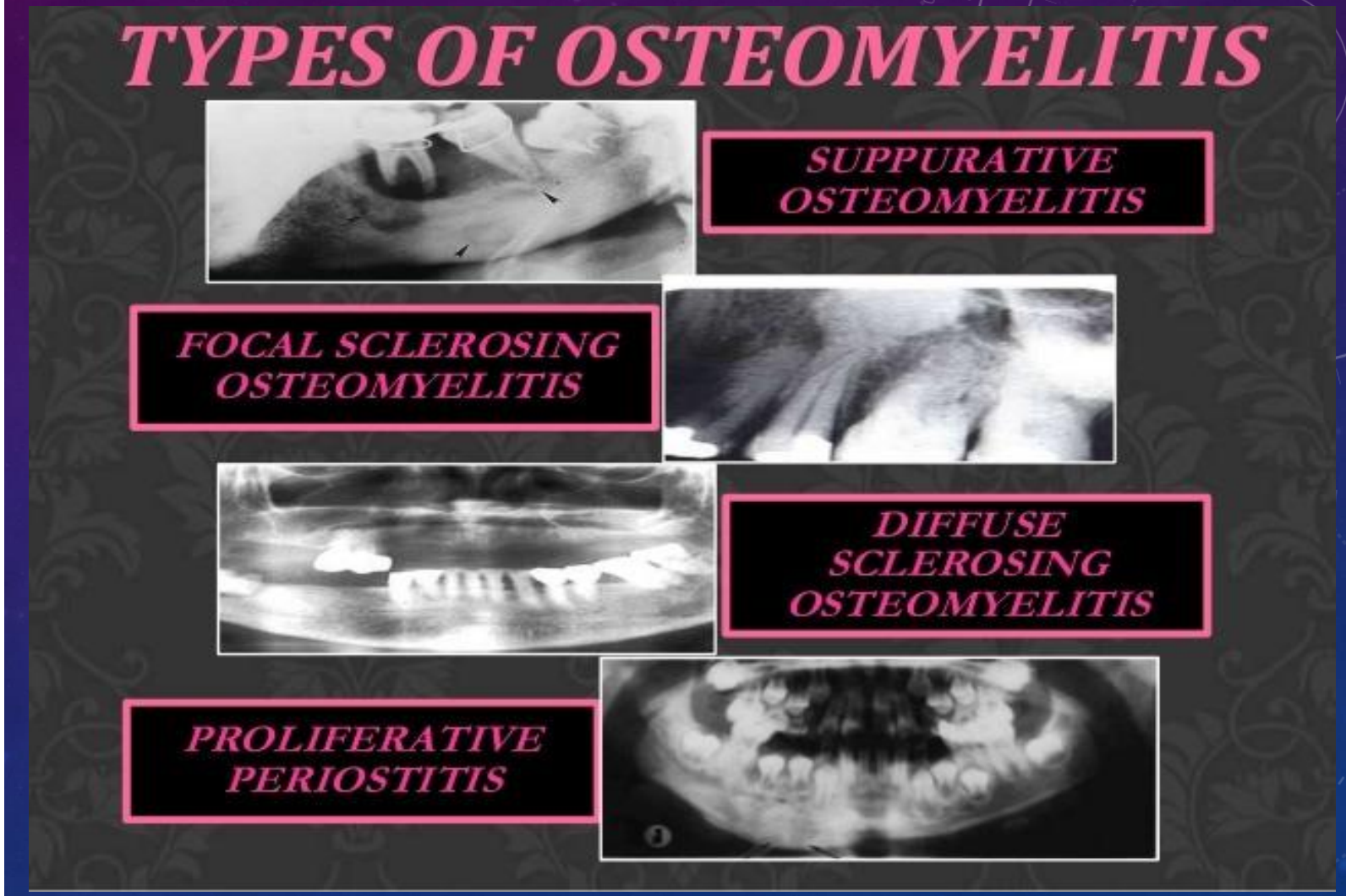


Microbiology

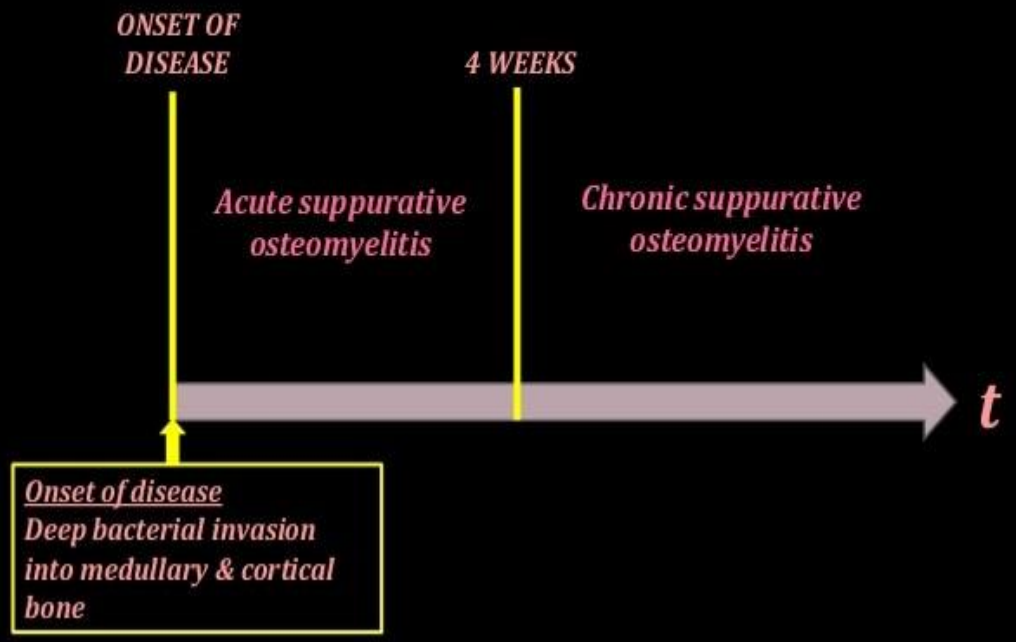
- Similar to those of odontogenic infections
  - Viridan streptococci
  - Strict anaerobes:
    - Bacteroides
    - Prevotella
    - Fusobacterium
    - Peptostreptococci species

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



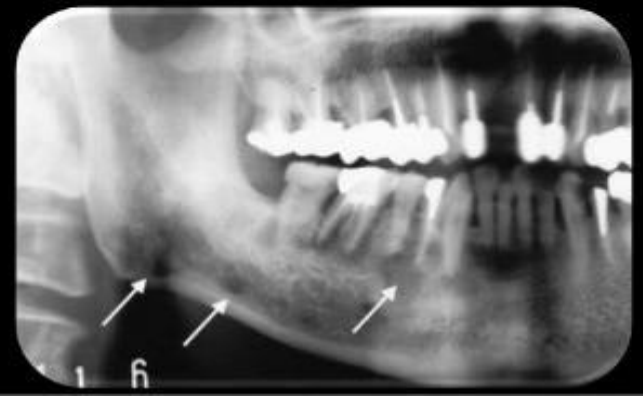
SUPPURATIVE OSTEOMYELITIS



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 :

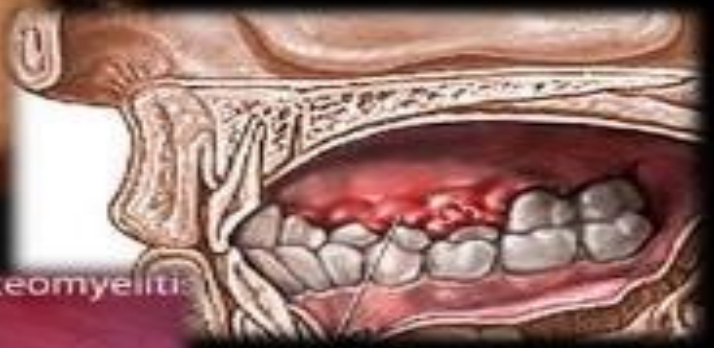
- Severe throbbing, deep- seated pain.
- Swelling due to inflammatory edema.
- Gingiva appears red, swollen & tender.

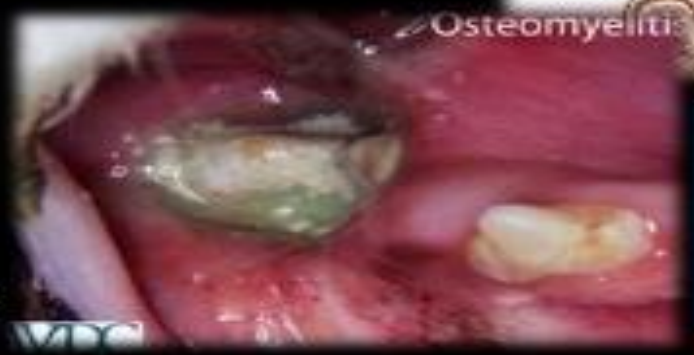
LATE :

- 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.

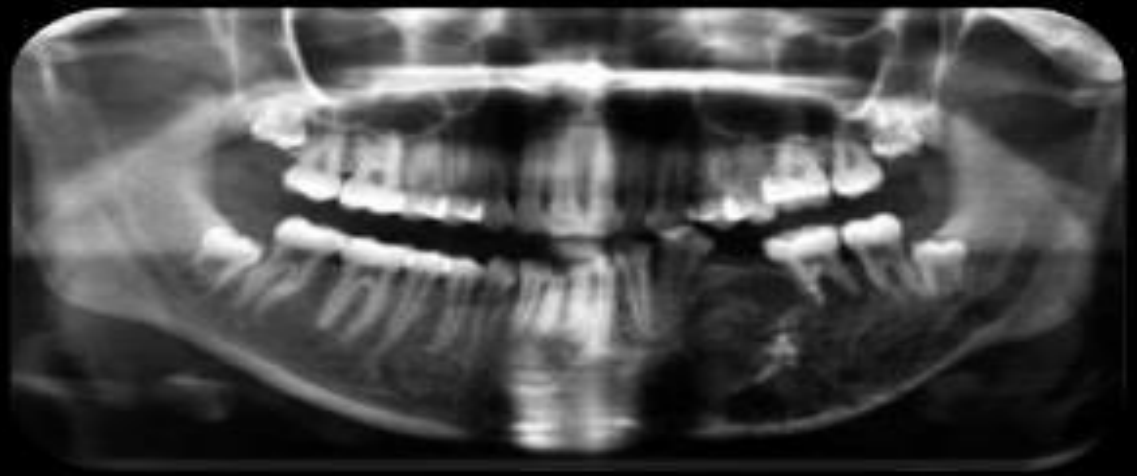
ACUTE SUPPURATIVE OSTEOMYELITIS

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

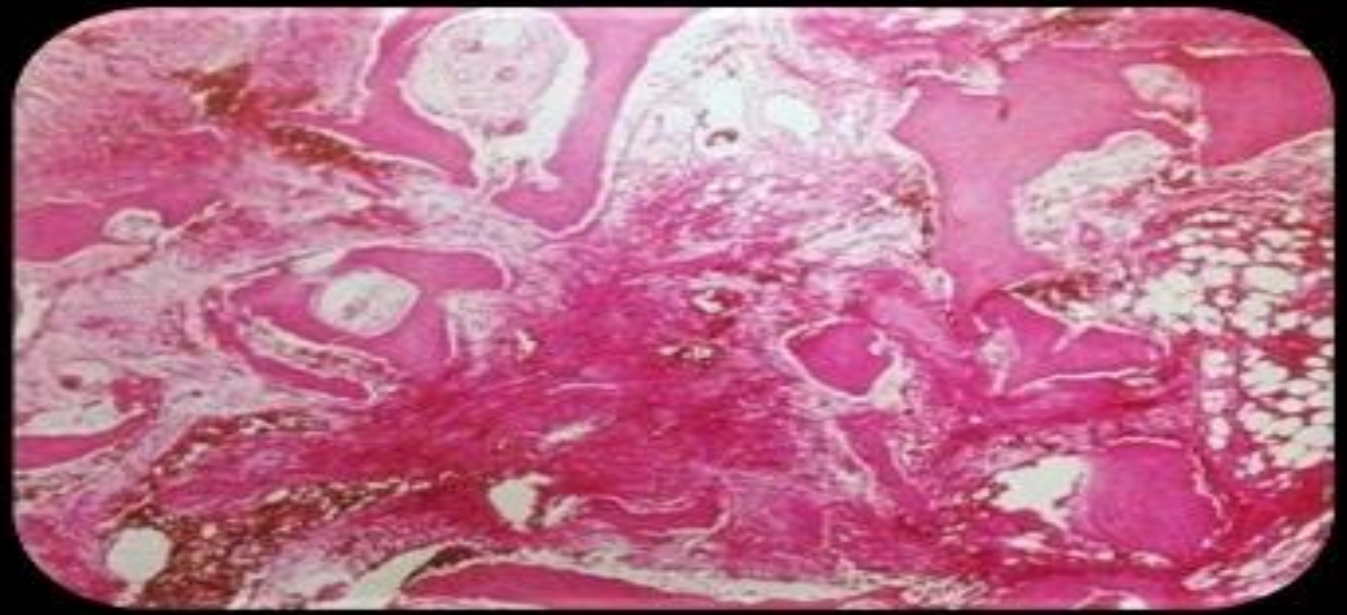
CLINICAL FEATURES

- *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.*

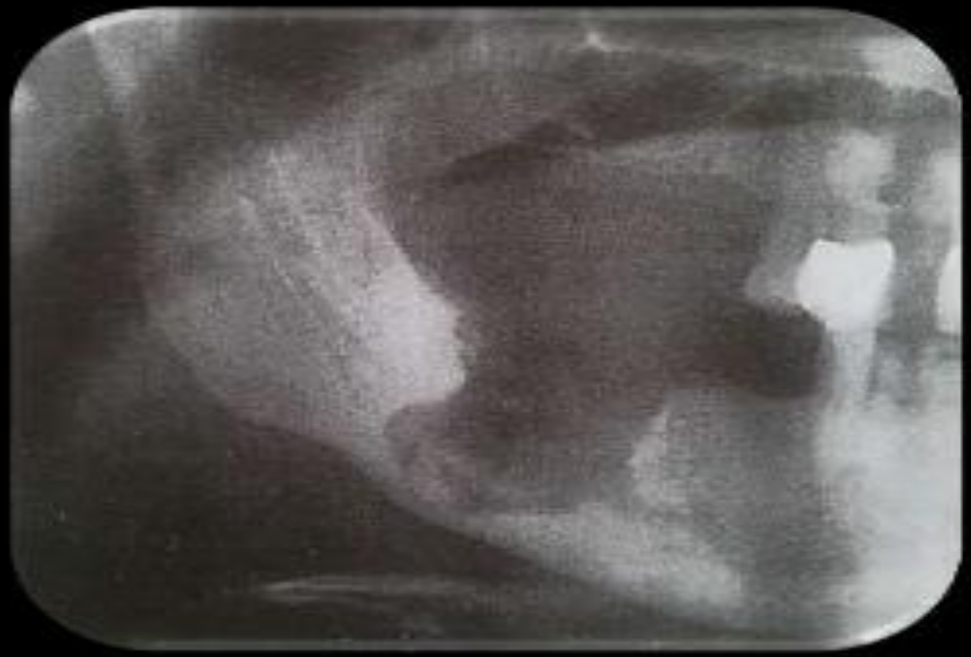


CHRONIC SUPPURATIVE OSTEOMYELITIS

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

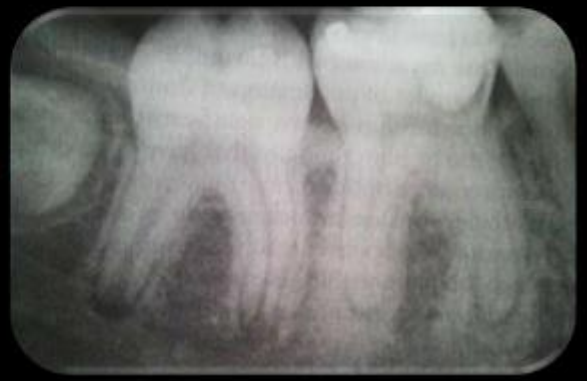
- *Dense sclerotic bone.*
- *Scanty connective tissue.*
- *Inflammatory cells.*

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.

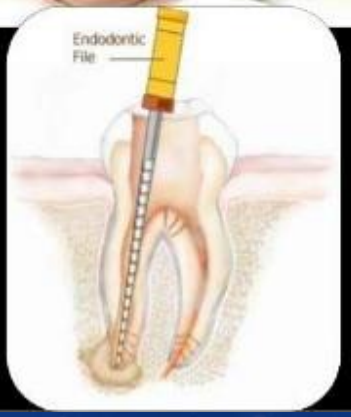
Increased areas of radiodensity surrounding apices of nonvital mandibular first molar



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.*

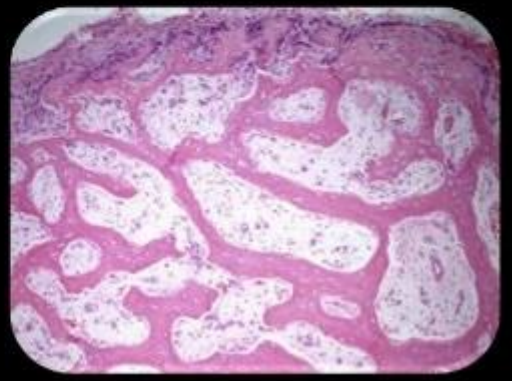
DIFFUSE SCLEROSING OSTEOMYELITIS

CLINICAL FEATURES

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

HISTOLOGY

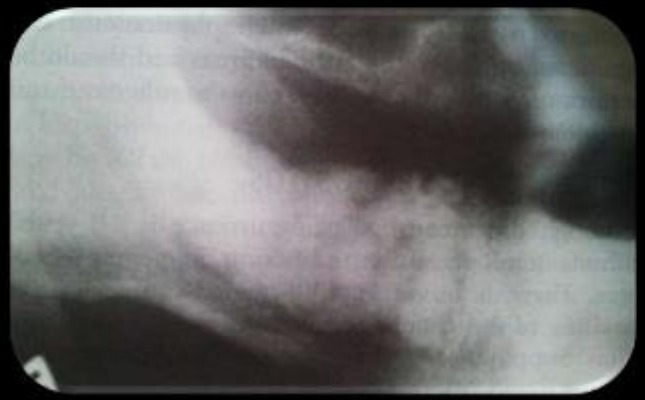
- *Bone sclerosis and remodling.*
- *Scanty marrow spaces.*
- *Necrotic bone separates from vital bone & become surrounded by granulation tissue.*
- *Secondary bacterial colonization often is visible.*



DIFFUSE SCLEROSING OSTEOMYELITIS

RADIOLOGY

Increased radiodensity may be seen surrounding areas of lesion.

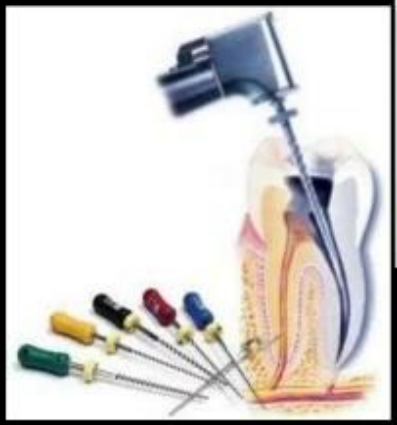


Diffuse area of increased radiodensity of Rt. Side of mandible

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

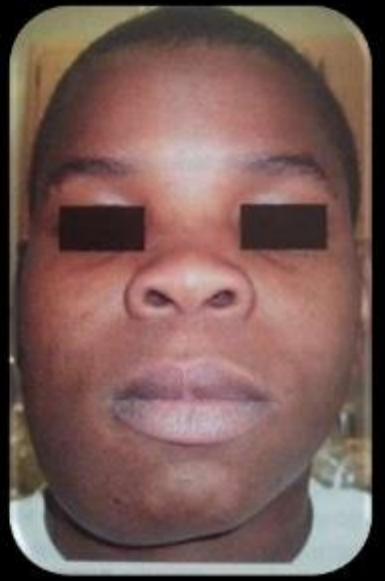
↓

Stimulates proliferative reaction of periosteum

PROLIFERATIVE PERIOSTITIS

CLINICAL FEATURES

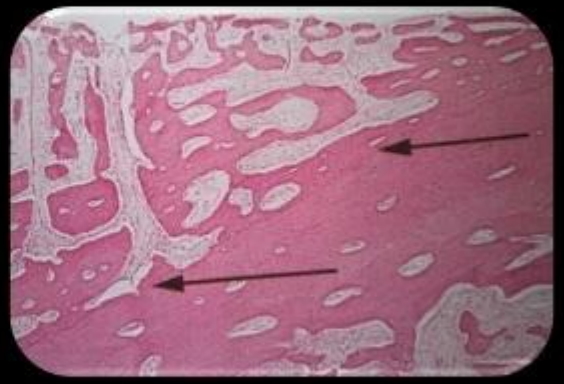
- *Affected patients are primarily children & young adults.*
- *Incidence is mean age of 13 years.*
- *No sex predominance is noted.*
- *Most cases arise in the premolar & molar area of mandible.*
- *Hyperplasia is located most commonly along lower border of mandible.*
- *Most cases are uni-focal, multiple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



PROLIFERATIVE PERIOSTITIS

HISTOLOGY

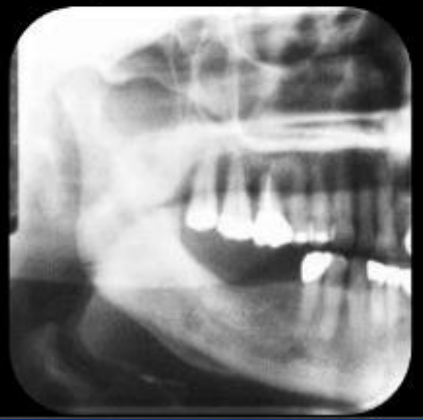
- Parallel rows of highly cellular & reactive woven bone .
- Trabeculae are frequently oriented perpendicular to surface.
- Trabeculae sometimes form an interconnecting meshwork of bone.
- Between trabeculae, uninflammed fibrous tissue is evident.



PROLIFERATIVE PERIOSTITIS

RADIOLOGY

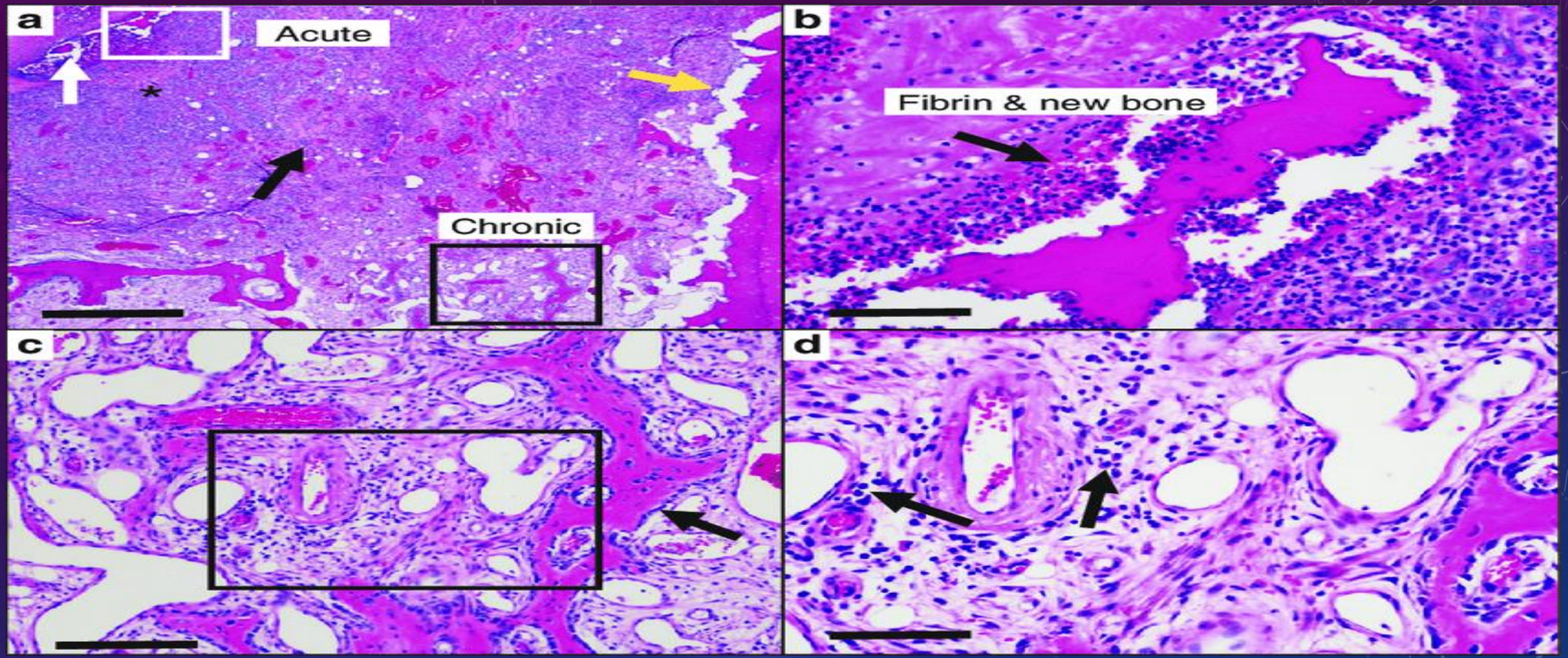
- Radiopaque laminations of bone roughly parallel each other & underlying cortical surface.
- Laminations may vary from 1-12 in number.
- Radiolucent separations often are present between new bone & original cortex.



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

Malignancy

Multiple Myeloma

Multiple myeloma is a disseminated and malignant disease that affects the plasma cells, causing bone lesions.

Pathophysiology

The mutated plasma cells produce faulty immunoglobins and osteoclast activating factors, this leads to bone resorption and patients experiencing pain.

Clinical features

Increase in plasma viscosity .Increased bleeding or clotting tendency .Renal failure .Prone to infections. Pathological fractures. Weakness .Paraesthesia

Investigations

Increase in ESR .Leukopenia .Thrombocytopenia .Normochromic anaemia .Hypergammaglobulinemia. Hypercalcemia

Dental impacts

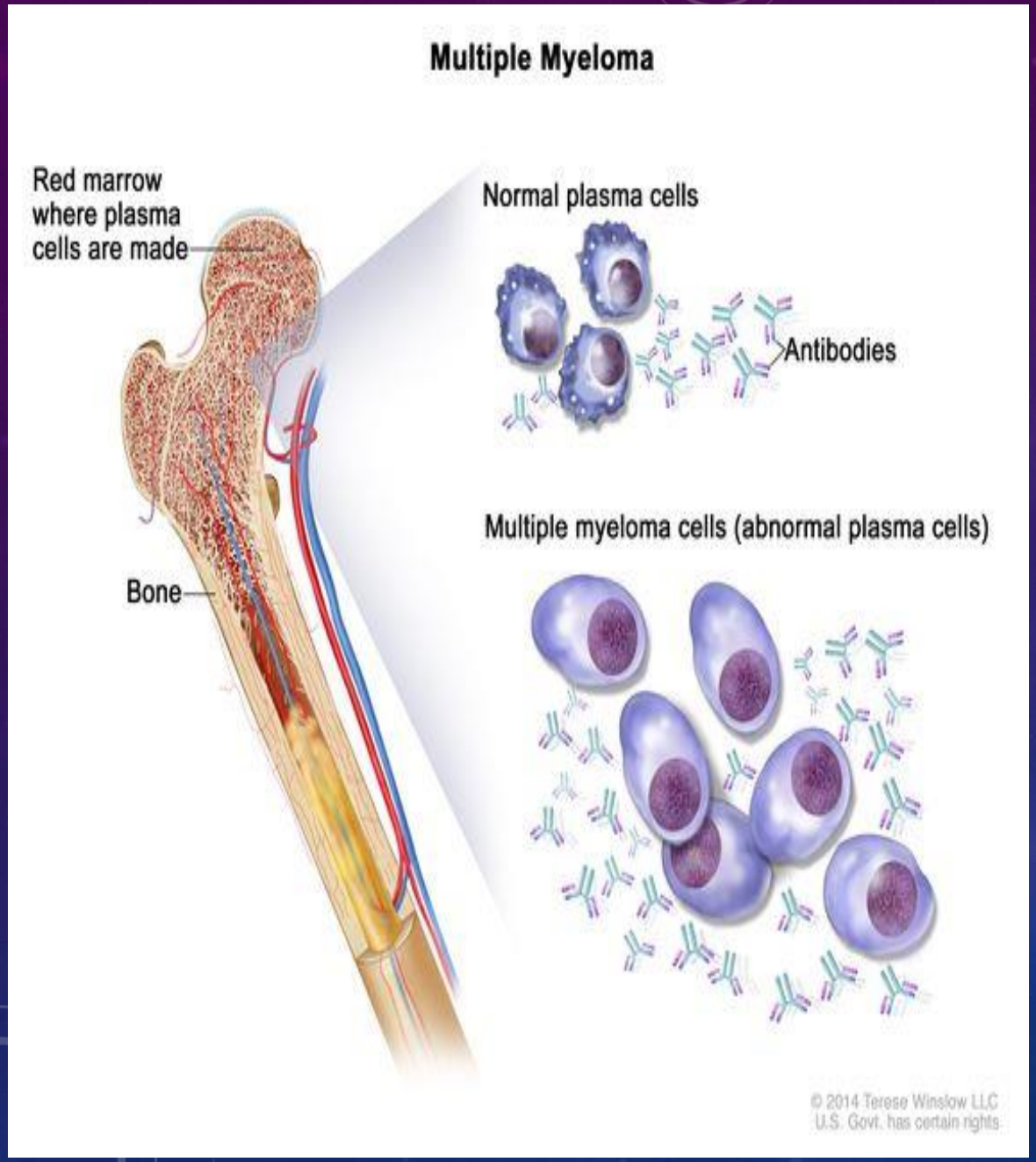
Jaw lesion can be potential first sign, small, punched-out, osteolytic lesion involves the posterior mandible are common. However, this is rare.

Symptoms of mental anaesthesia, root resorption, loosening of the tooth, pathological fractures can be impacted.

Patients may present with gingival bleeding, oral petechiae, cranial nerve palsies and herpes simplex or zoster infections. Abnormal protein that interferes with organs normal function (amyloid) can be found in the oral soft tissues this can lead to local or widespread swellings for example macroglossia.

Complications: Melphalan is a type of chemotherapy drug and has been associated with mucositis.

Bisphosphonates can increase the risk of MRONJ.


Multiple Myeloma
Red marrow where plasma cells are made
Bone
Normal plasma cells
Antibodies
Multiple myeloma cells (abnormal plasma cells)
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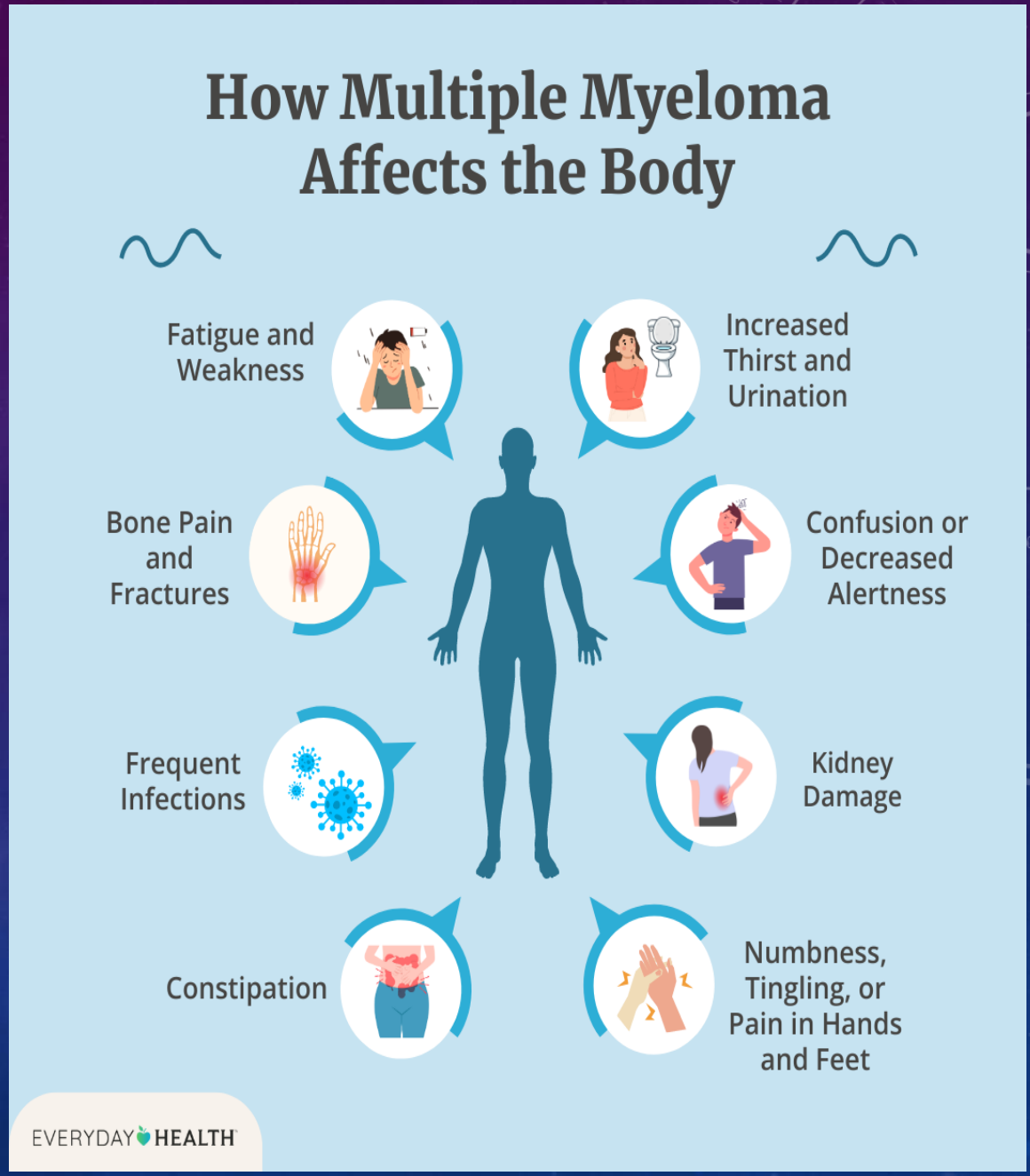
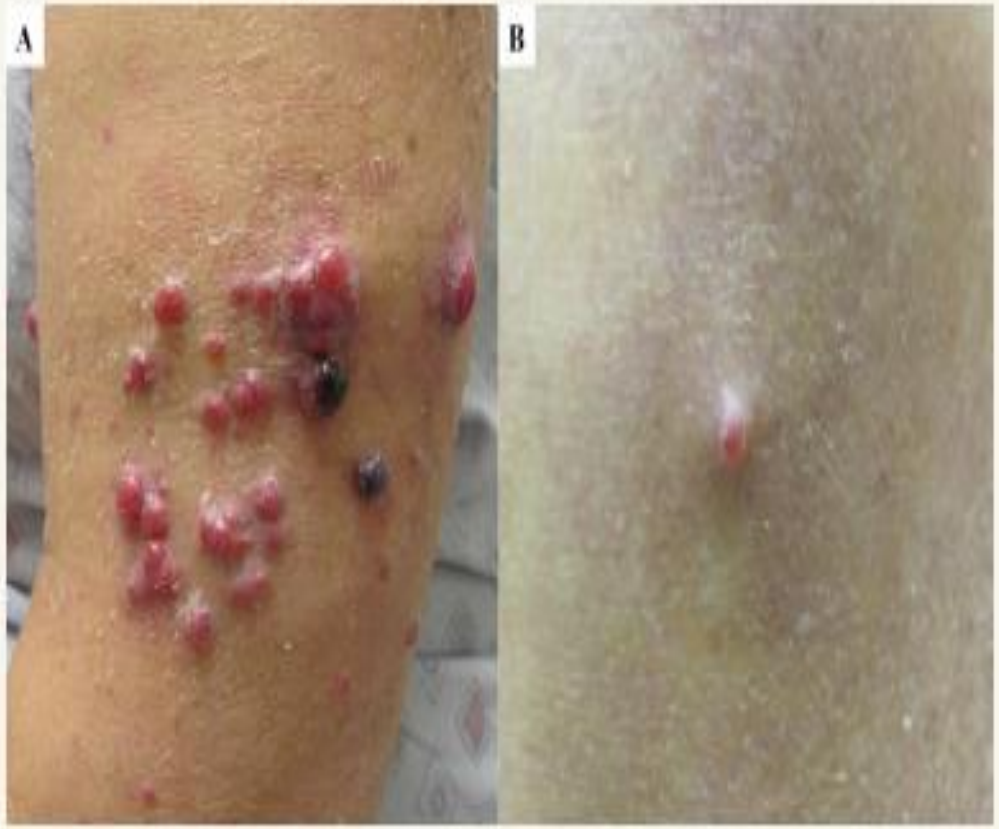
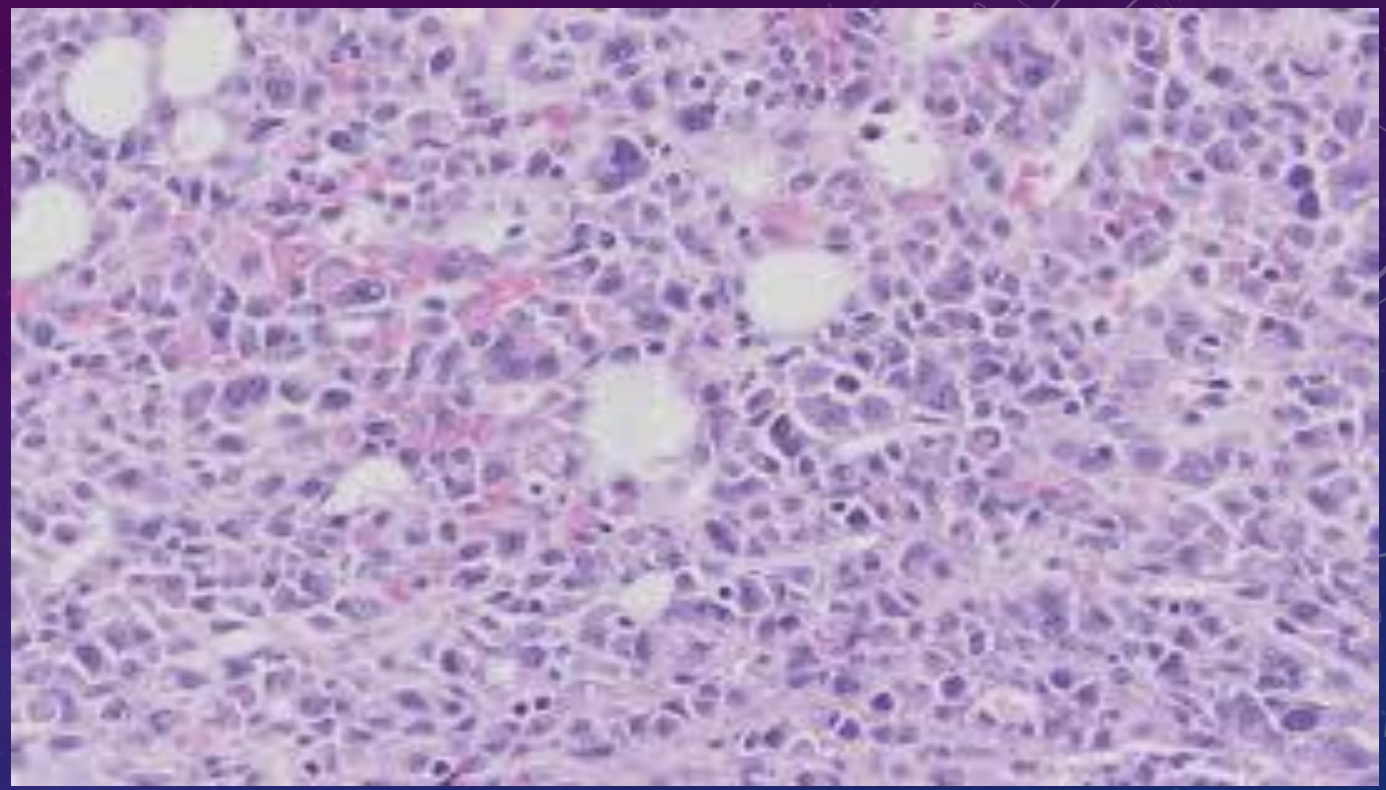

How Multiple Myeloma Affects the Body
Fatigue and Weakness
Increased Thirst and Urination
Bone Pain and Fractures
Confusion or Decreased Alertness
Frequent Infections
Kidney Damage
Constipation
Numbness, Tingling, or Pain in Hands and Feet
EVERYDAY HEALTH

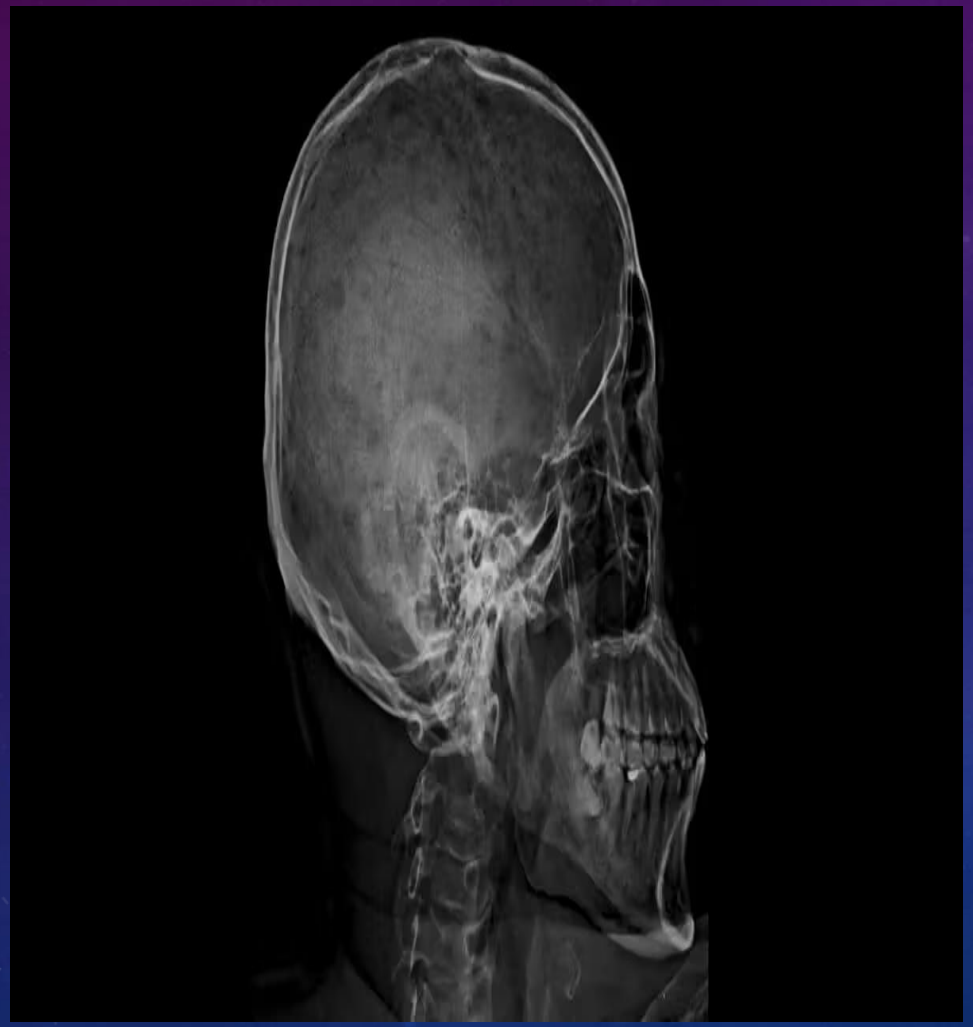
FIGURE 1. Metastatic multiple myeloma presenting as red-to-violaceous, smooth topped papules coalescing into plaques on the left arm (A), and a solitary red papule on the right leg (B).

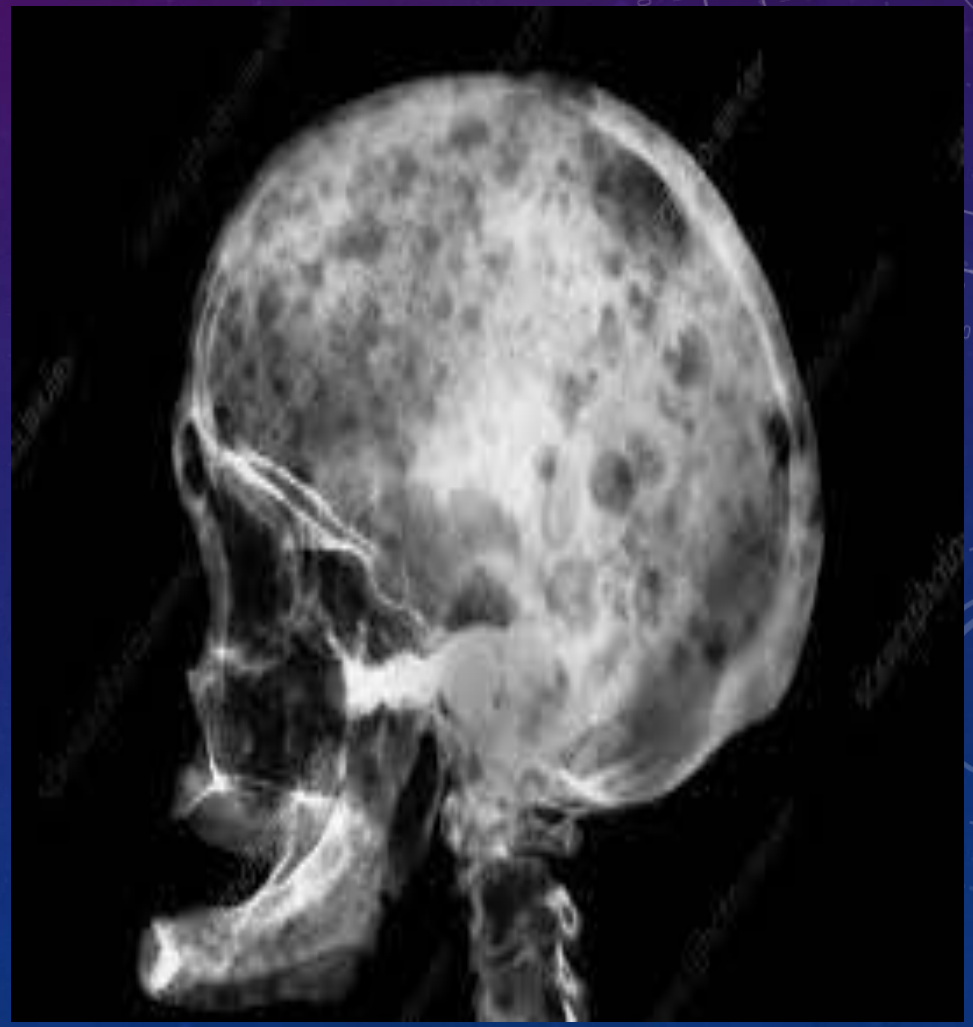




The atypical features of the plasma cells are better seen at higher magnification (40x lens, H&E).

X-rays can show lytic lesions in bones, lytic lesions usually appear as holes or punched-out areas in flat bone, such as in the skull or pelvis.





Osteosarcoma

The most common primary malignant bone tumour that presents in children and young adults. It is described as a very aggressive form of cancer.

Pathophysiology

Genetic mutation that causes the tumour to become malignant and is involved in the direct formation of bone or osteoid.

Clinical features

Bone pain or tenderness
An increased weight is felt through the skin
Swelling and redness at the location of the tumour
Pain when moving the joints around
Restricted movement
Fracture from minor trauma or simple movement

Investigations

Core needle biopsy

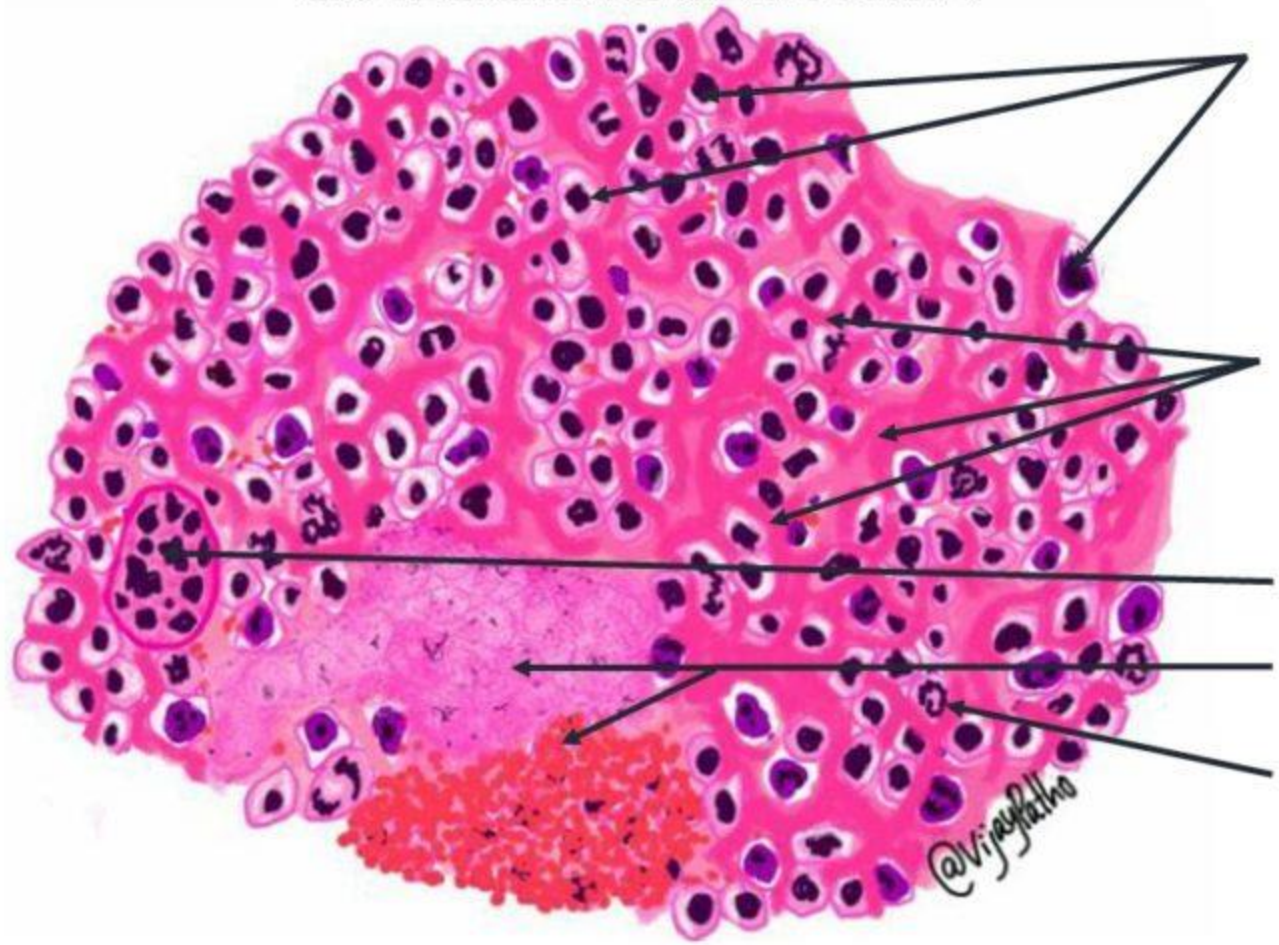
Radiographs followed by MRI scan to check the extent.14

Biochemical markers – serum alkaline phosphate (ALP) and lactase dehydrogenases (LDH) can aid with diagnosing the condition and the prognosis. The increase in ALP is caused by increase osteoblastic activity. The higher the level, the more progressed the cancer is and poorer prognosis. If the patient remains with high levels after treatment this indicates residual disease or potential metastasis. 16

Treatment

Chemotherapy and surgery are routinely carried out. The drugs used in chemotherapy is doxorubicin, cisplatin, and methotrexate

OSTEOGENIC SARCOMA



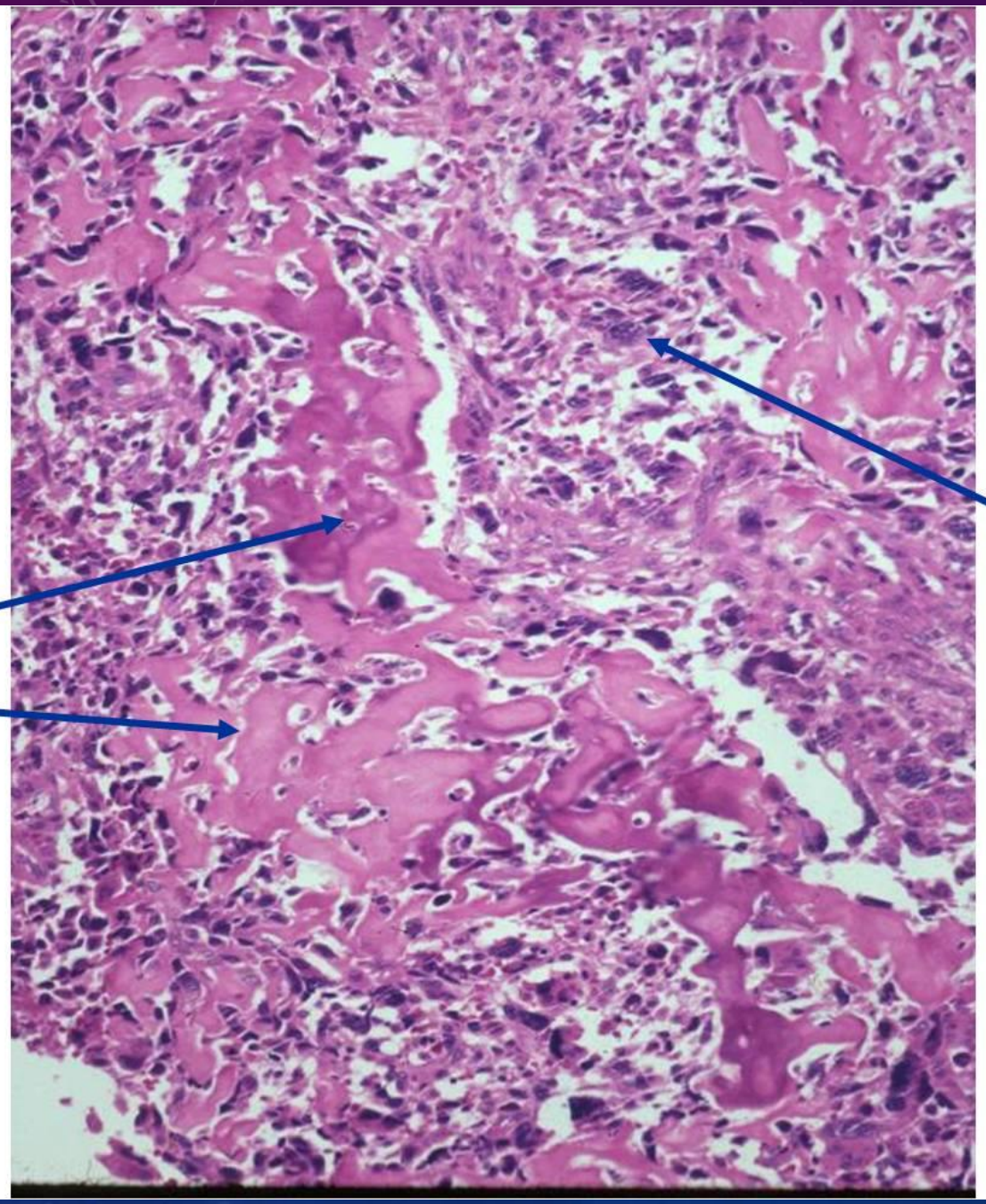
Tumor cells with varied morphology with pleomorphic & hyperchromatic nuclei

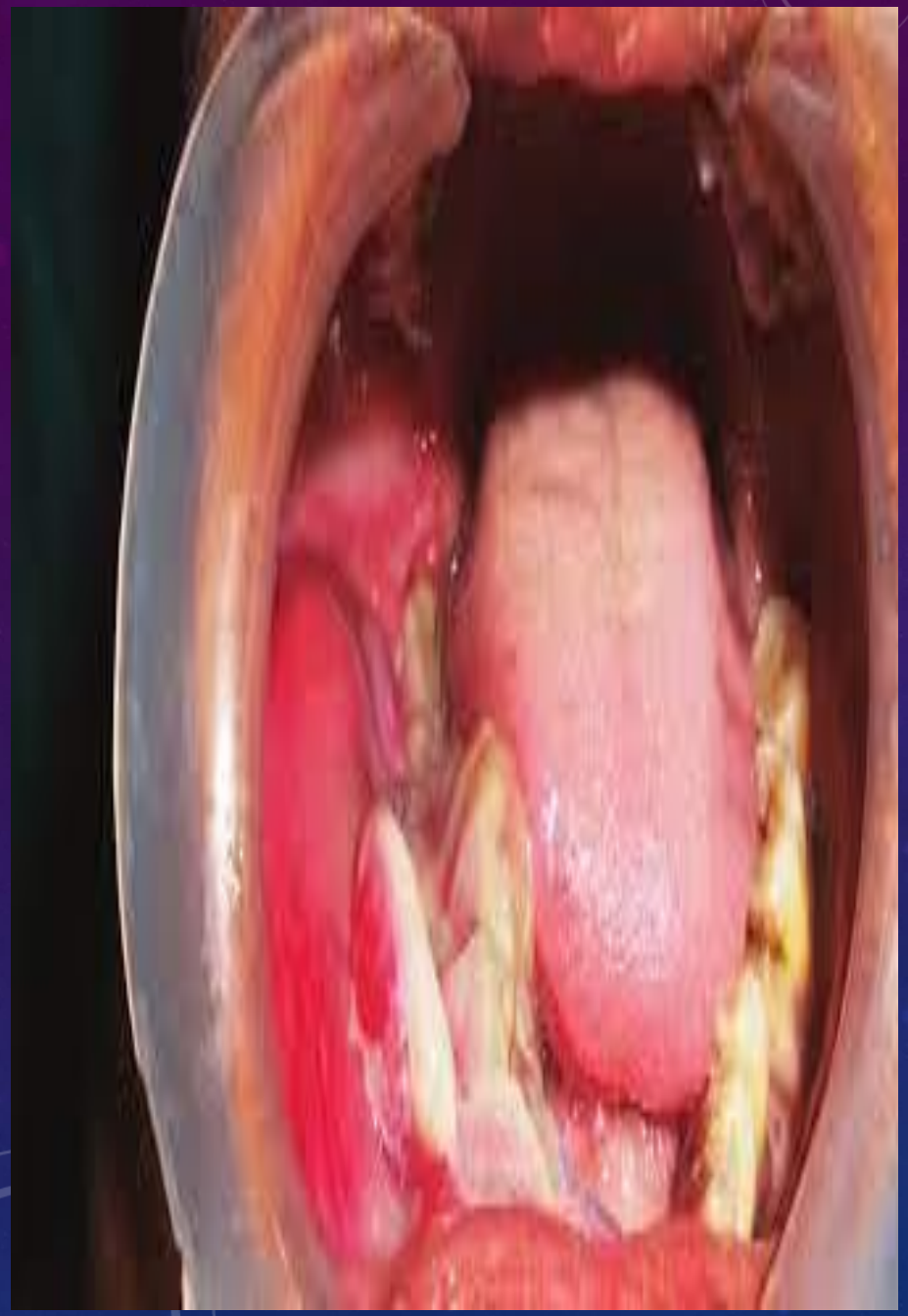
eosinophilic, homogenous, glassy appearing lacelike material - **OSTEOID**

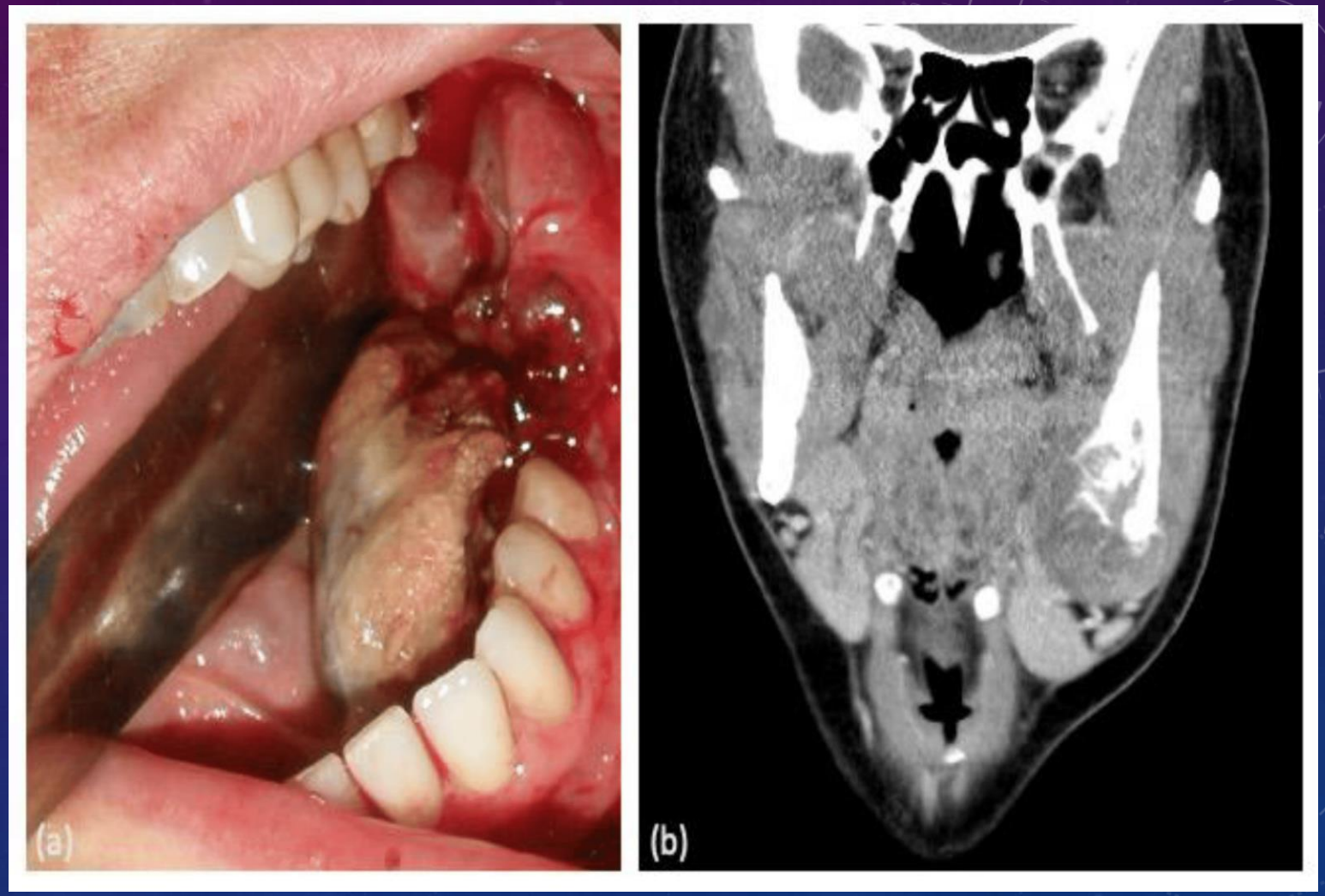
Tumor giant cell

Necrosis & hemorrhage

Atypical mitosis


Osteoid Production
High Grade Anaplastic Spindle Cells with Large Hyperchromatic Nuclei




(a)
(b)


A
B


No cut


Thank
You