

MSS

Paget disease and osteomyelitis

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Paget disease

- **Chronic** disease of bones with episodes of increased bone resorption (osteoclast activity, osteolytic phase)
followed by excessive ^{متزايد} **mixed** osteoblast and osteoclast activity (**mixed phase**)
leading to disordered, poorly formed bone with increased density (**osteosclerotic**
^{net =} **phase**) and increased likelihood of fractures . ^{fibrotic}
- It affects individual **over 40** with slight **male** predilection.
- Etiology : unknown, Occasionally hereditary influence is noted on chromosome **18q**.

Sites

- Patients can present as **polyostotic** (multiple bones) or **monostotic** (one bone)
- Most common sites:
 - **Spine and pelvis** (30 - 75%)
 - **Sacrum** (30 - 60%)
 - **Skull** (25 - 65%)
 - Femur (25 - 25%)
- Rare in hands / feet, ribs, fibula

Axial



So Paget disease

Accelerated bone turnover making disordered bone.

Bone turnover rate increased as much as 20 times normal.



PATHOPHYSIOLOGY

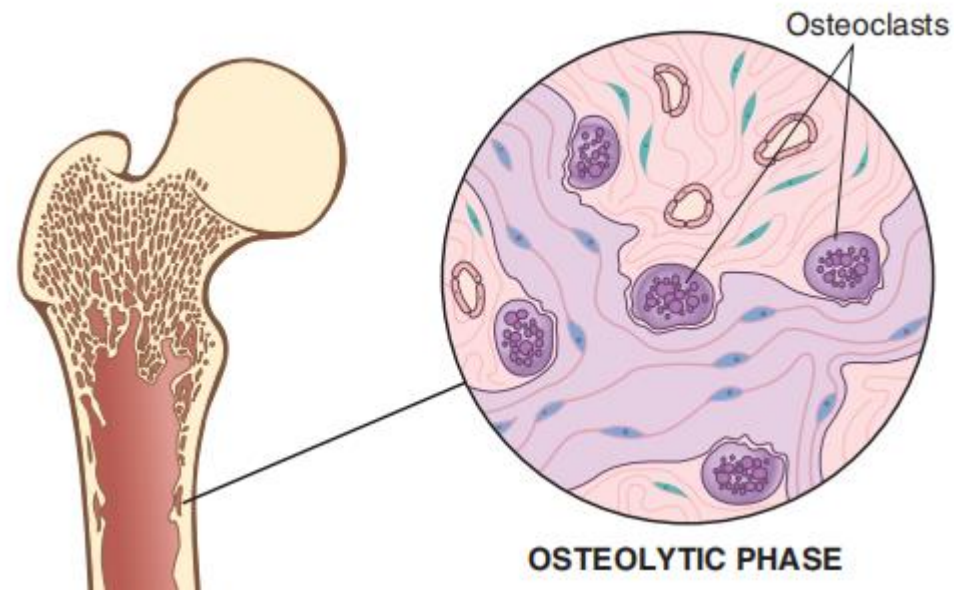
- Believed to be a disease of **osteoclasts**
- Genetic and environmental problems can lead to disruption in osteoclast differentiation and activation.



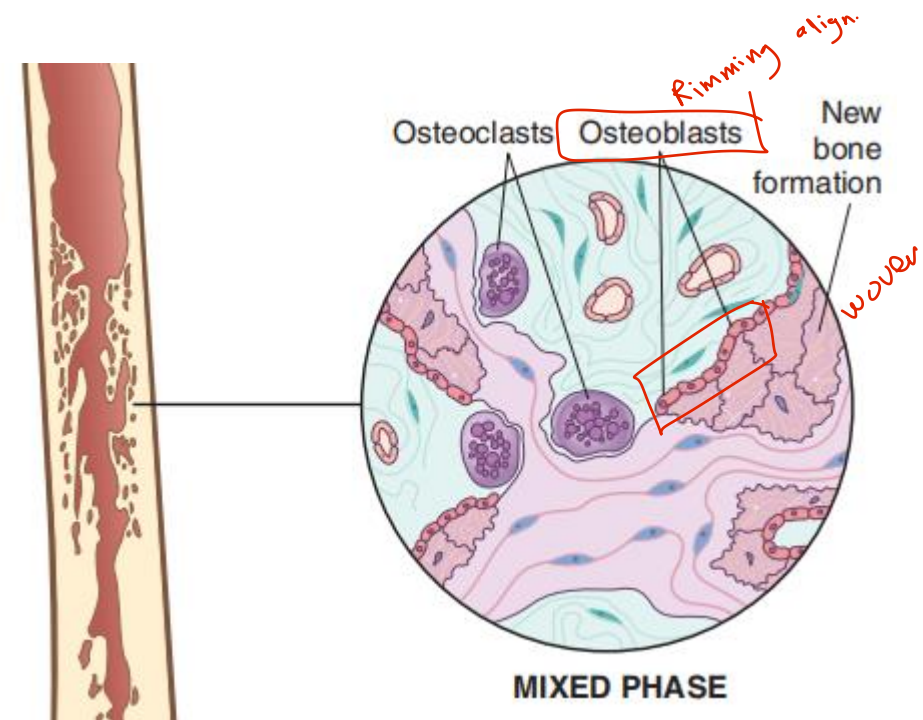
Phases

- Paget's disease histology progress through **three phases:**
- At a given time, **multiple stages** of disease may be demonstrated in **different skeletal regions** of same patient.



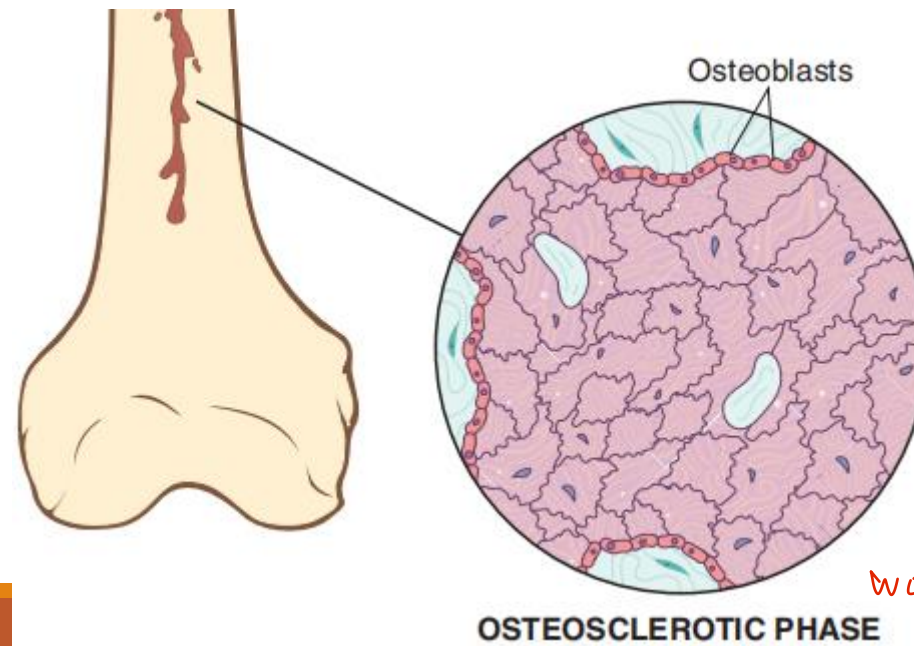


osteocytic (early, acute phase).



mixed osteolytic and osteoblastic (intermediate phase).

new bone: woven
mature: lamellar

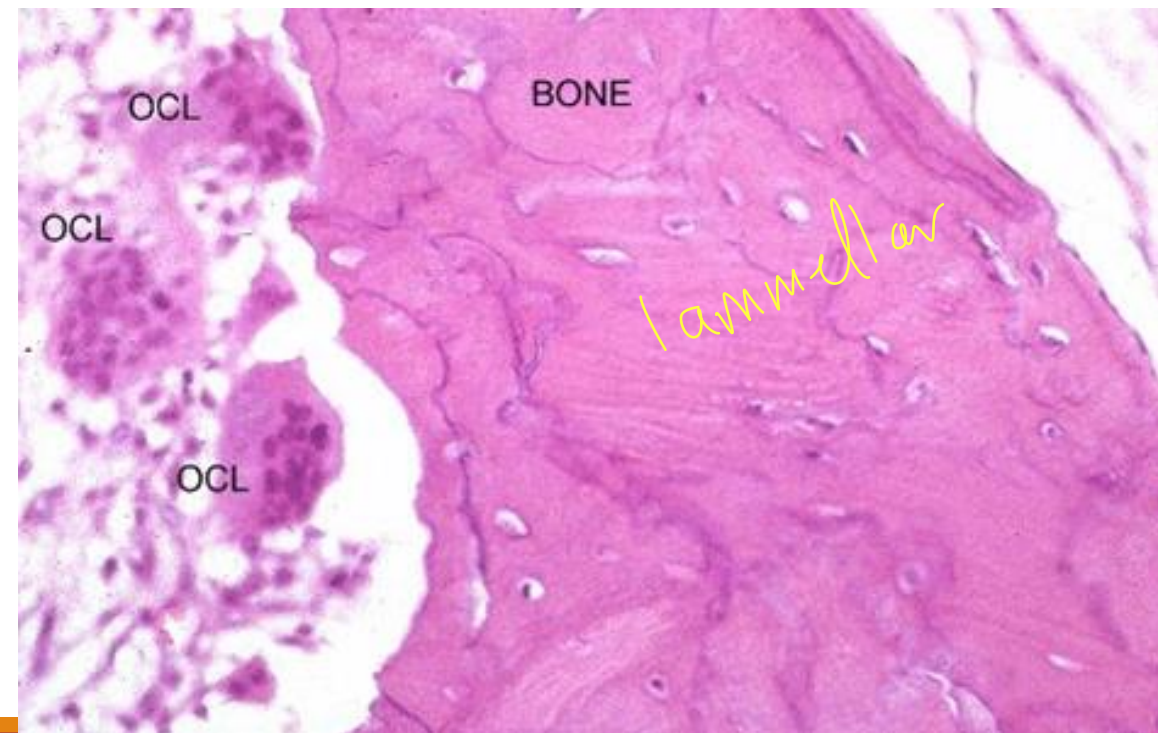


sclerotic phase (late, burnt-out).



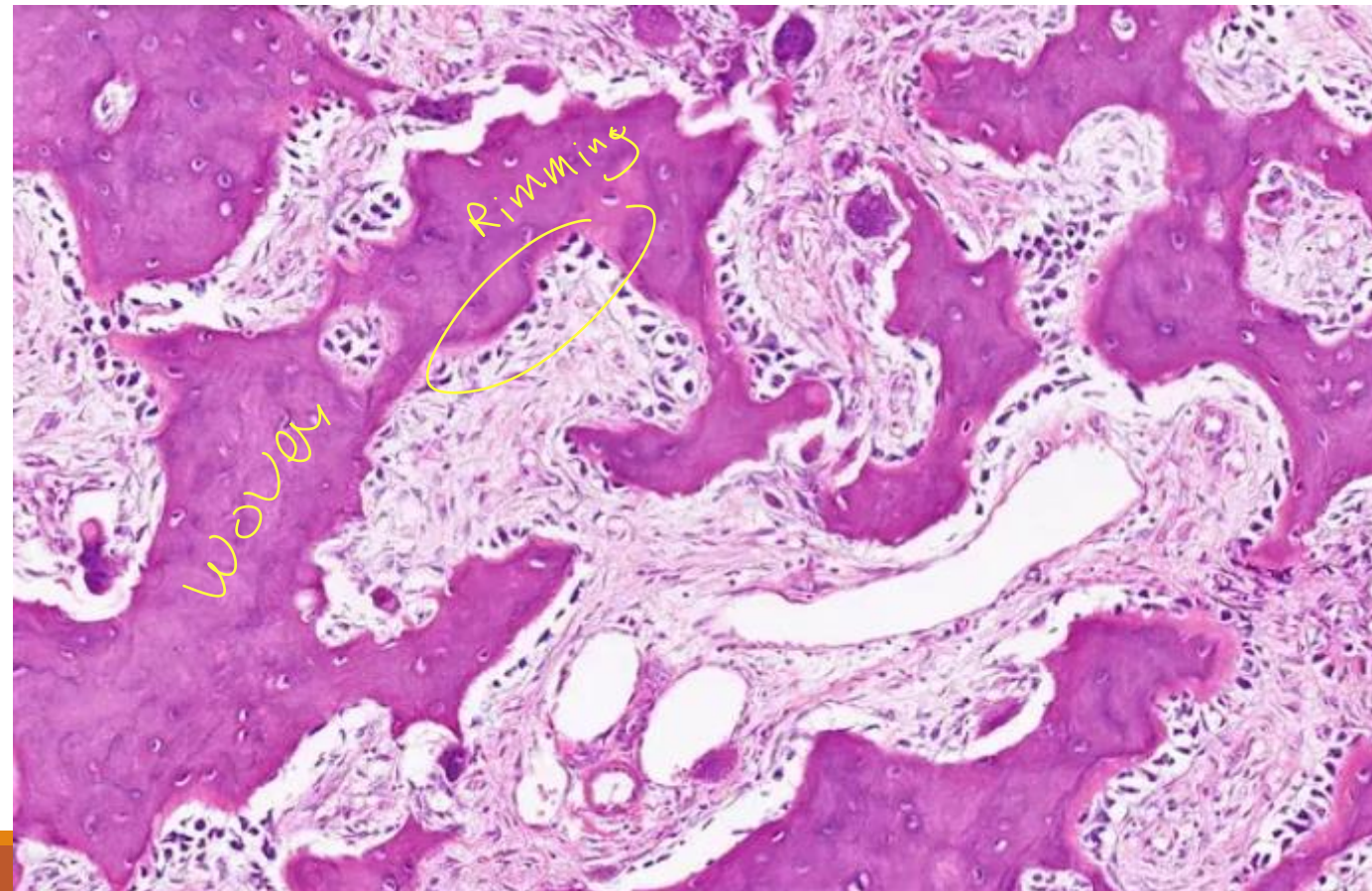
LYTIC PHASE

- Disease begins with lytic phase.
- The bone is resorbed by osteoclasts that are more numerous, larger and have **more nuclei** (up to 100).



Mixed Lytic and Blastic phase

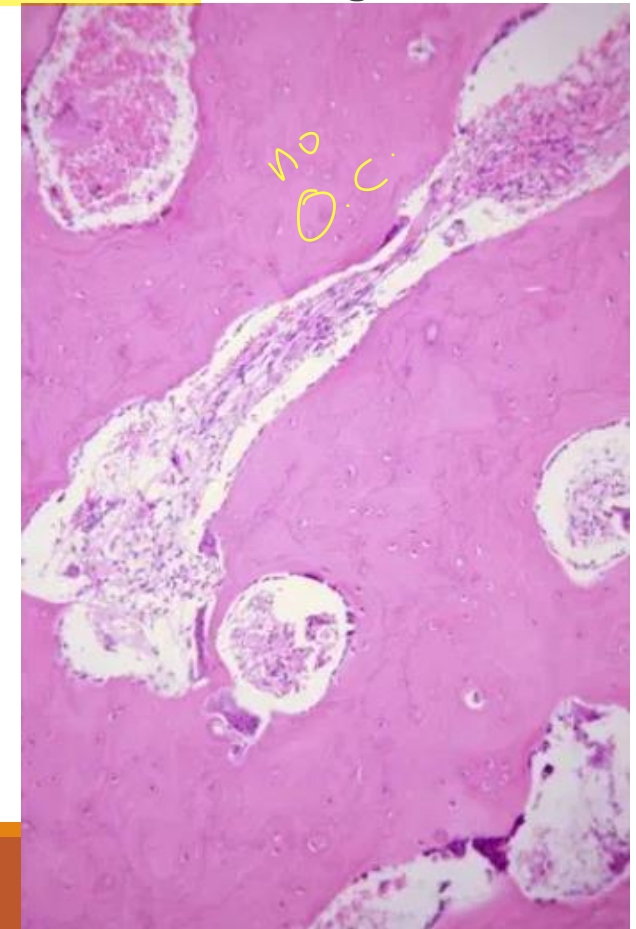
Rapid increase in bone formation from numerous osteoblasts.



Sclerotic Phase

The bone formation dominates and has a disorganized woven pattern and is weaker than normal bone. Woven pattern allows the bone marrow to be infiltrated by blood vessels leading to hyper vascular bone state.

BI سبب
= ↑ heart burden
= H.F.



Clinical presentation

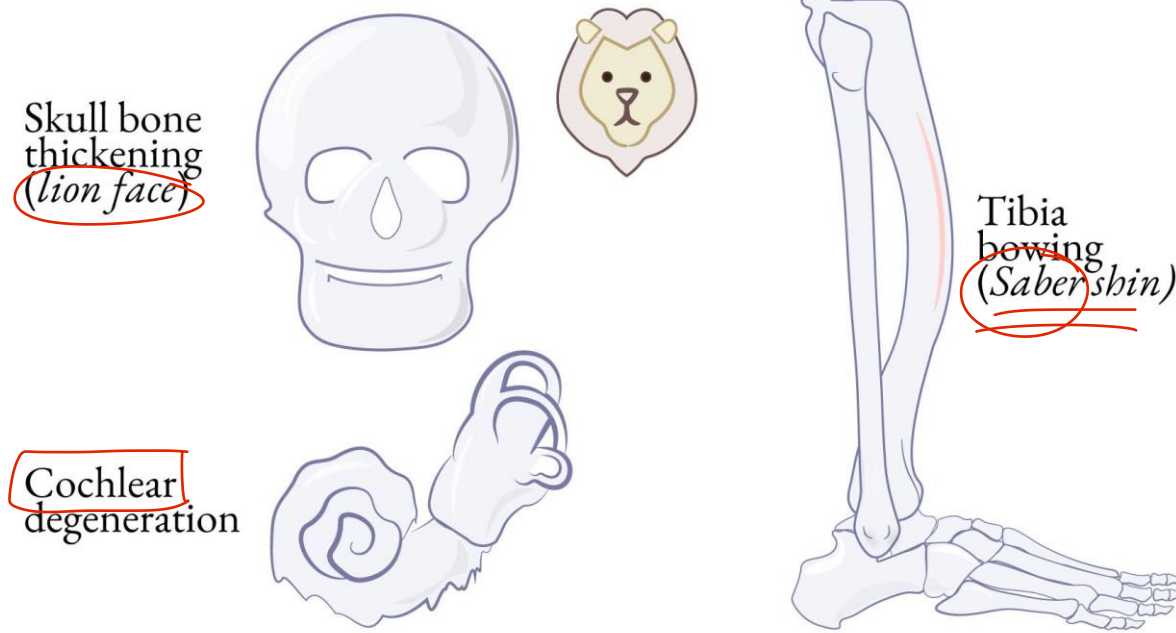
- Initial presentation is commonly after **pathologic fracture** or Incidental finding after imaging studies or **serum alkaline phosphatase** for other reasons
(liver investigation)
- Bone pain and **deformities** *تشوي*
- Bone overgrowth or deformity can cause osteoarthritis.

(Bowel d)



Pain (nerve compression)

Paget's Disease (Osteitis Deformans)



BIG LIONS Have Heart failure

- Bone pain
- Increased bone density
- Giant osteoclasts

- Lion face
- Increased Alk phos.
- Osteolytic/sclerotic X-Ray
- Nerve compression
- Saber shin

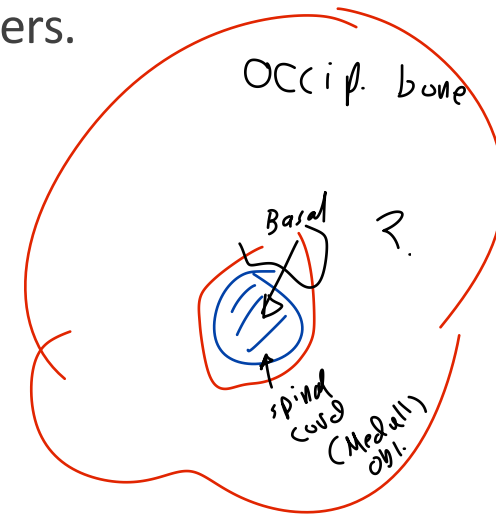
- Hearing loss
- Heart failure (high-output)



Complications

- Neurological complications – nerve root compression.
- Skull involvement- **deafness** and **basilar invagination** cranial nerve disorders.
- Sarcomatous degeneration – **Osteosarcoma**.
- Increased **bone vascularity** – high output cardiac failure.

Went 2x
BYs UN.



Investigations

- Serum Alkaline phosphatase will be increased.
- Serum calcium and phosphate levels will be normal.
- X-RAYS: Long bones (bowing thickening of cortex).



TREATMENT

- At this time there is **no cure** for Paget's disease, therefore treatment is designed to control the **symptoms** and prevent complications.

- Goals of treatment:
 - Suppression of Active disease.
 - Relief of Pain Prevention of Deformity and fractures.
 - High output cardiac dysfunction.
 - Reducing the Sarcomatous transformation

Osteomyelitis

severe persistent infection of bone and bone marrow.

Types of osteomyelitis: *أسباب مختلفة لعدوى العظام*

1. **Post traumatic** osteomyelitis: (47% cases) *↓ immune*
2. Osteomyelitis due to **vascular insufficiency**: (34% cases)
3. Osteomyelitis due to **hematogenous spread**: (19%) *Septicemia*
4. Osteomyelitis post infection of prosthetic joints



Classification

Acute osteomyelitis:	Childhood osteomyelitis: long bones of the legs and upper arms. <i>* Highly Vascul.</i> <i>* Friable</i>	Pyogenic osteomyelitis <i>Staph. aureus</i>
Chronic osteomyelitis:	Adults osteomyelitis: bones of the vertebrae.	Tuberculous osteomyelitis



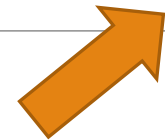
Pathogenesis

Bacteria form a **biofilm** in the **metaphysis (primary focus)**

↓ immunity



Biofilms protect bacteria from host immune response



Abscess in metaphysis



Sub periosteal abscess

حالت



dead bone
Sequestrum formation (bone death)



Involucrum formation (New brittle bone formation)



Pus perforates periosteum and forms **abscess in soft** tissues

Draining sinus (fistula)



Factors affecting pathogenesis

1. ↑ Virulence of the infecting . *Organism*
2. Underlying disease. *(DM)*
3. ↓ Immune status of the host.
4. Type, location and vascularity of the bone. *child → long bones*
Adult → Vertebrae
5. **Factors that compromise bone integrity:** • Trauma • Surgery • Presence of foreign bodies • Placement of prostheses Leads to the onset of bone infection

Chronic osteomyelitis

- **Longstanding** infection of bone lasting months to **years**; characterized by low grade inflammation and presence of dead bone or fistulous tract. (Abscess) سقر!
- The infected foci within the bone are surrounded by sclerotic, relatively avascular bone covered by a thickened periosteum and scarred muscle and subcutaneous tissue. fibrous/fatty replaced
- This avascular envelope of scar tissue leaves systemic antibiotics essentially ineffective. (IV) So Antibiotic can't reach

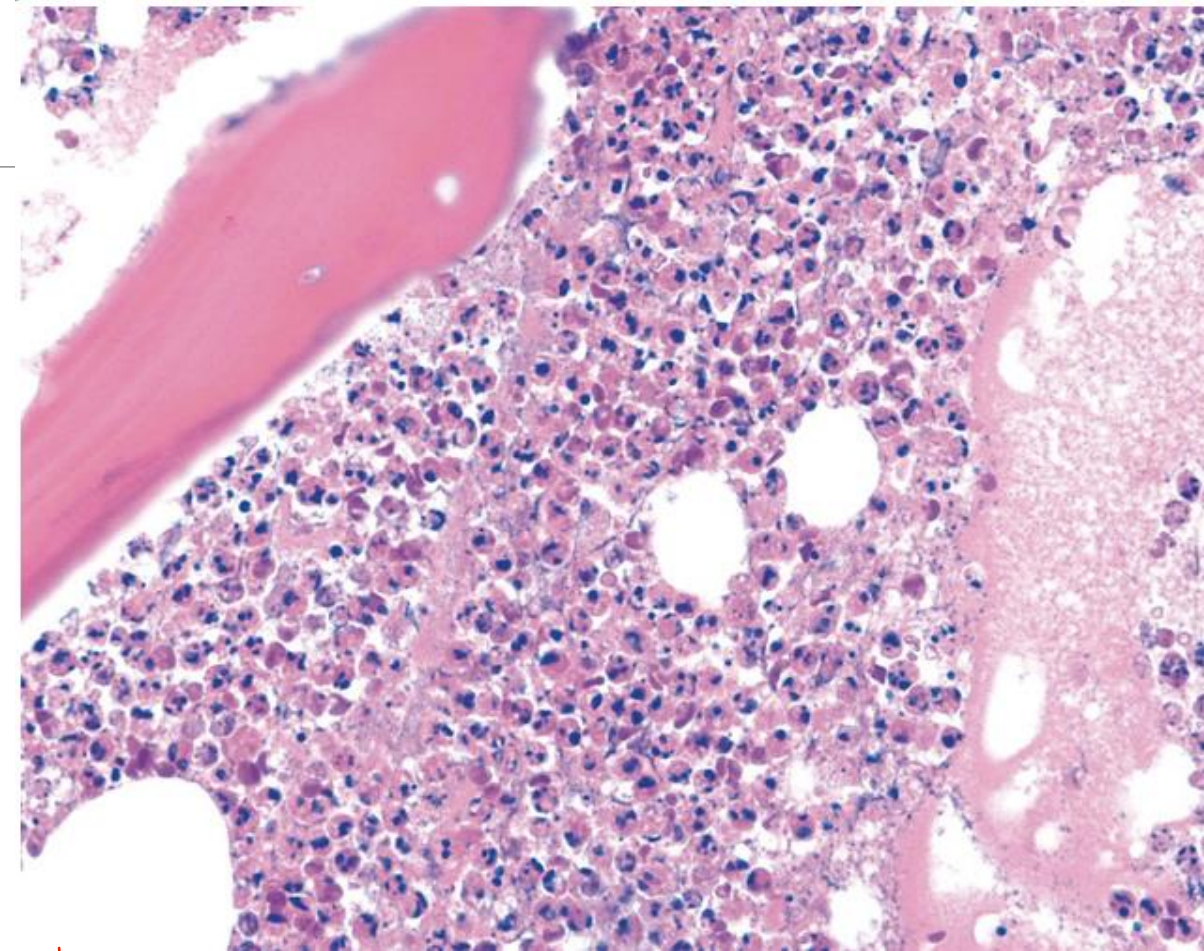
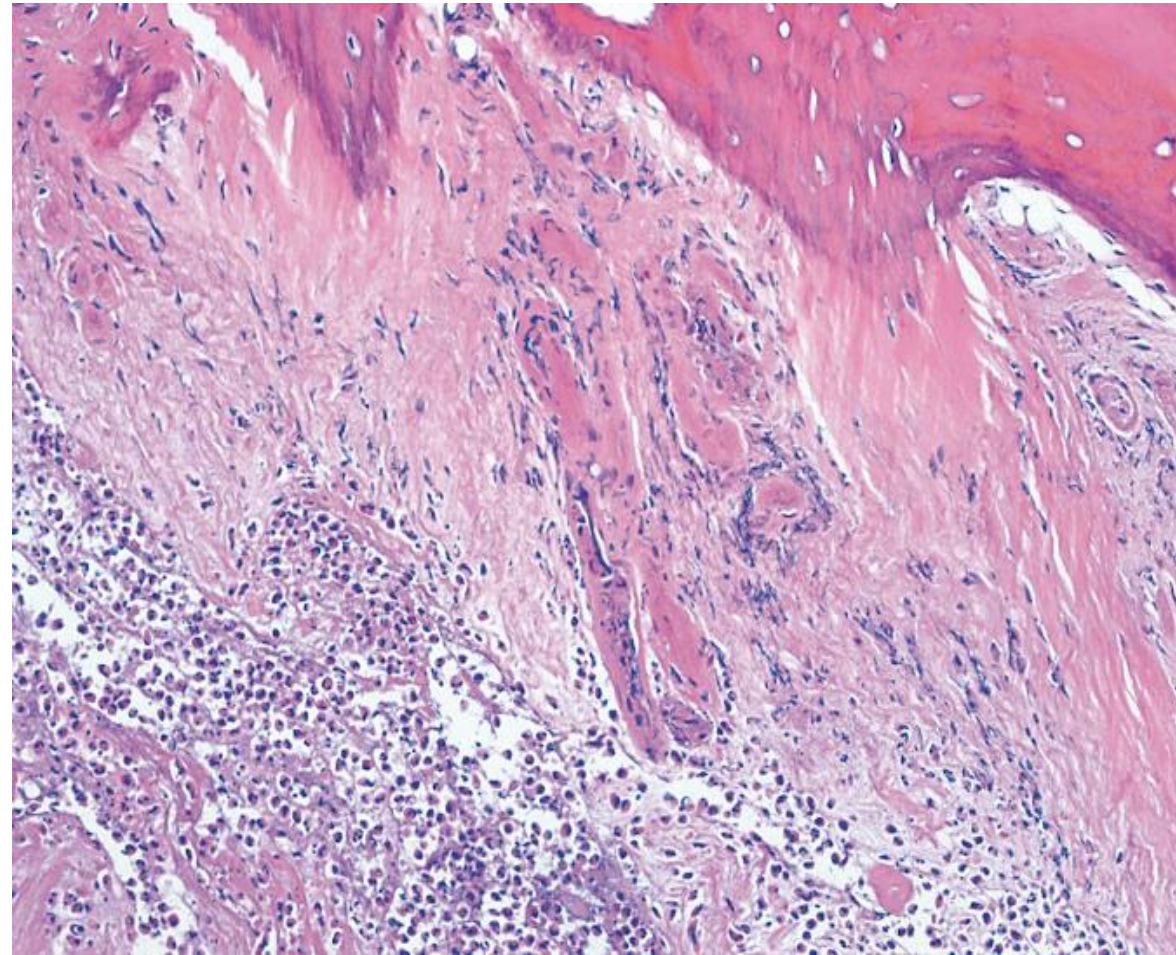
Factors leading to chronic osteomyelitis

- Trauma
- Diabetes *P*
- Prosthetic orthopaedic device *T*
- Peripheral vascular disease (*Poor removal*)
- Chronic joint pain
- i/v drug abuse
- Immunosuppression
- Alcoholism

Initiation

P₁ → P₂ → P₃ → P₄ → P₅ → P₆ → P₇ → P₈ → P₉ → P₁₀

Microscopic (histologic) description



- Inflammatory infiltrate rich in **plasma cells** ★ Diagnostic
- Fibrosis, variable ★
- **Granulomas**, in cases of tuberculosis or fungal infection

TB

Etiology

- Penetrating wound, open fracture: *Staphylococcus aureus*
- In dwelling prosthetic device: *Staphylococcus epidermidis* (normal flora on skin)
- Intravenous drug users: *Pseudomonas* infections.
- Gastrointestinal or genitourinary infections: *Escherichia coli* & others
- Tooth abscess, gingival disease, dental extraction: *Streptococcus viridans*
- Mycobacterium tuberculosis: **Bone tuberculosis**
- Sickle cell disease: *Salmonella* species in the West
Staphylococcus aureus in Middle East & Africa

Clinical presentation

- gn) Inf
- Fever, chills, irritability, fatigue.
 - Tenderness, redness, and warmth in the area of the infection.
 - Swelling around the affected bone.
 - Lost range of motion. (no motion)
 - The symptoms for acute and chronic osteomyelitis are very similar
- Site
- acute = chronic



Osteomyelitis complications

1. **Bone death (osteonecrosis):** An infection can impede blood circulation within the bone, leading to bone death.
2. **Septic arthritis:** In some cases, infection within bones can spread into a nearby joint.
3. **Impaired growth:** In children, the most common location for osteomyelitis is in the softer areas, called growth plates, at either end of the long bones of the arms and legs. Normal growth may be interrupted in infected bones.
4. **Skin cancer:** If osteomyelitis has resulted in an open sore that is draining pus, the surrounding skin is at higher risk of developing squamous cell cancer

Pistula → irritation

SCC

Treatment

Surgery to remove dead bone (sequestrum) → Debridment

Antibiotics.

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