Bone and Joint Infections

Types

1. Septic Arthritis
2. Osteomyelitis

A. Septic Arthritis

- Infection of the joints resulting in inflammation of the synovial membrane with purulent effusion into the joint capsule.
- usually bacterial,
- 50% of cases in children <3 years
- Hip joint In <3years, while knee joint in older children
- Usually acute painful arthritis, (may be subacute or chronic)
- **AKA** : pyogenic arthritis, infective arthritis or suppurative arthritis.
- Surgical Emergency - > Permanent joint dysfunction with subchondral bone loss

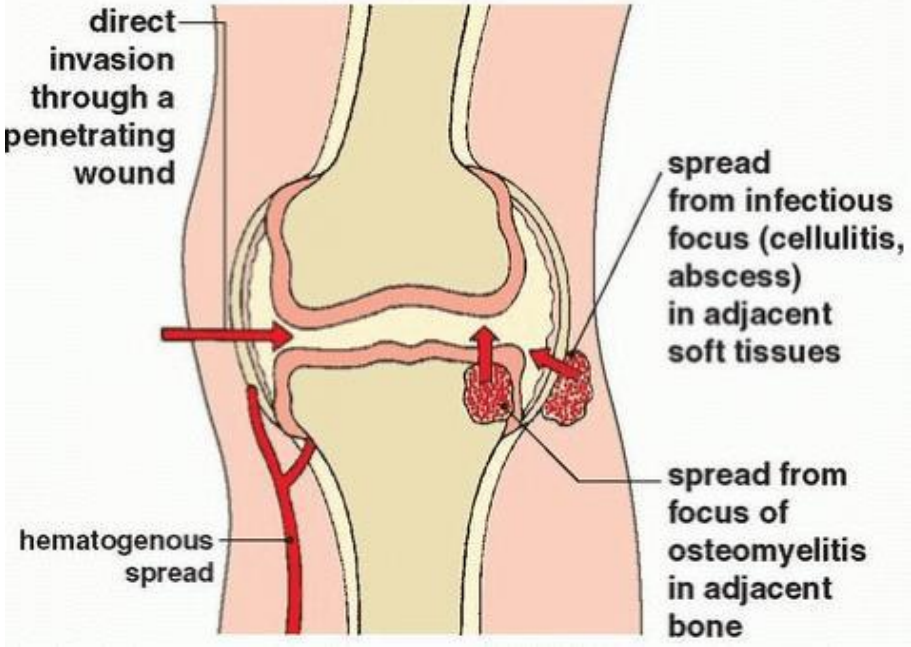
A. Septic Arthritis

- S. aureas: Commonest
- GAS
- GBS (Esp neonates)
- Pneumococcus
- Gonococcus (Esp. Adolescents)
- *H. influenzae* (Esp. Children)
- *Others: P. aeruginosa, E. coli, M. TB*

A. Septic Arthritis

Route of Infection:

1. Haematogenous: Distant infection through blood t the bone
2. Direct inoculation/contamination; May result from trauma
3. Contiguous spread; extension from adjacent bone infection e.g. osteomyelitis



A. Septic Arthritis

Risk Factors:

1. Prosthetic implant
2. Interventions (e.g., intra-articular injections)
3. Underlying joint disease, especially rheumatoid arthritis
4. Immunosuppressed state;
5. Diabetes mellitus
6. Age > 80 years
7. Chronic skin infections
8. IV drug use

A. Septic Arthritis

Pathogenesis:

1. Organism reaches the joint by one of the above routes, then begins an inflammatory response in the synovium (Synovitis) resulting in the exudation of fluid within the joint.
2. Joint cartilage is destroyed by inflammatory granulation tissue and lysosomal enzymes in the joint exudate.
3. Release of proteolytic enzymes (matrix metalloproteinases) from inflammatory and synovial cells,
4. cartilage, & bacteria may cause articular surface damage within 8hrs
5. Increased joint pressure may cause femoral head osteonecrosis if not relieved promptly

A. Septic Arthritis

Pathogenesis:

6. Outcome varies from complete healing to total destruction of joint.
7. Latter may result in a complete loss of joint movement (ankylosis).
8. The infective process can be summed up as;

    Serous or acute synovitis, Serofibrinous, Suppurative (purulent) arthritis

A. Septic Arthritis

Clinical features:

a. **General manifestations**: constitutional symptoms and signs of acute infection

b. **Local manifestation:**

Swelling, hotness and redness

Deformity with muscle spasm

Restriction of all movements of the joint

The joint is fixed in the position of ease

A. Septic Arthritis

Investigations:

Blood- FBC and diff count- shows neutrophilic leucocytosis. ESR is markedly elevated.

2. Blood culture may grow the causative organism.

3. Plain X-ray- AP and lateral view; findings normal, especially in early stages

Later often may reveal widening of the joint space, subluxation, or dislocation

Soft tissue shadow corresponding to distended capsule due to swelling of joint.

5. Ultrasound: may be helpful to identify effusion and to guide aspiration

A. Septic Arthritis

Investigations:

6. Aspiration:

- cell count with differential
- Gram stain, culture, and sensitivity
- Glucose and protein levels

 A septic joint aspirate will show

- High WBC count (> 50,000/mm3 with >75% PMNs)
- Glucose 50 mg/dl less than serum levels
- High lactic acid level with infections due to gram positive cocci or gram-negative rods

NB: If WBC >50,000 with >90% PMNLs suspect septic arthritis even if culture is negative.

A. Septic Arthritis

Complications:

1. Destruction of femoral head
2. Deformity and stiffness
3. Joint contracture
4. Hip dislocation
5. Gait deformity
6. Growth disturbance
7. Osteonecrosis
8. Pathological dislocation

A. Septic Arthritis

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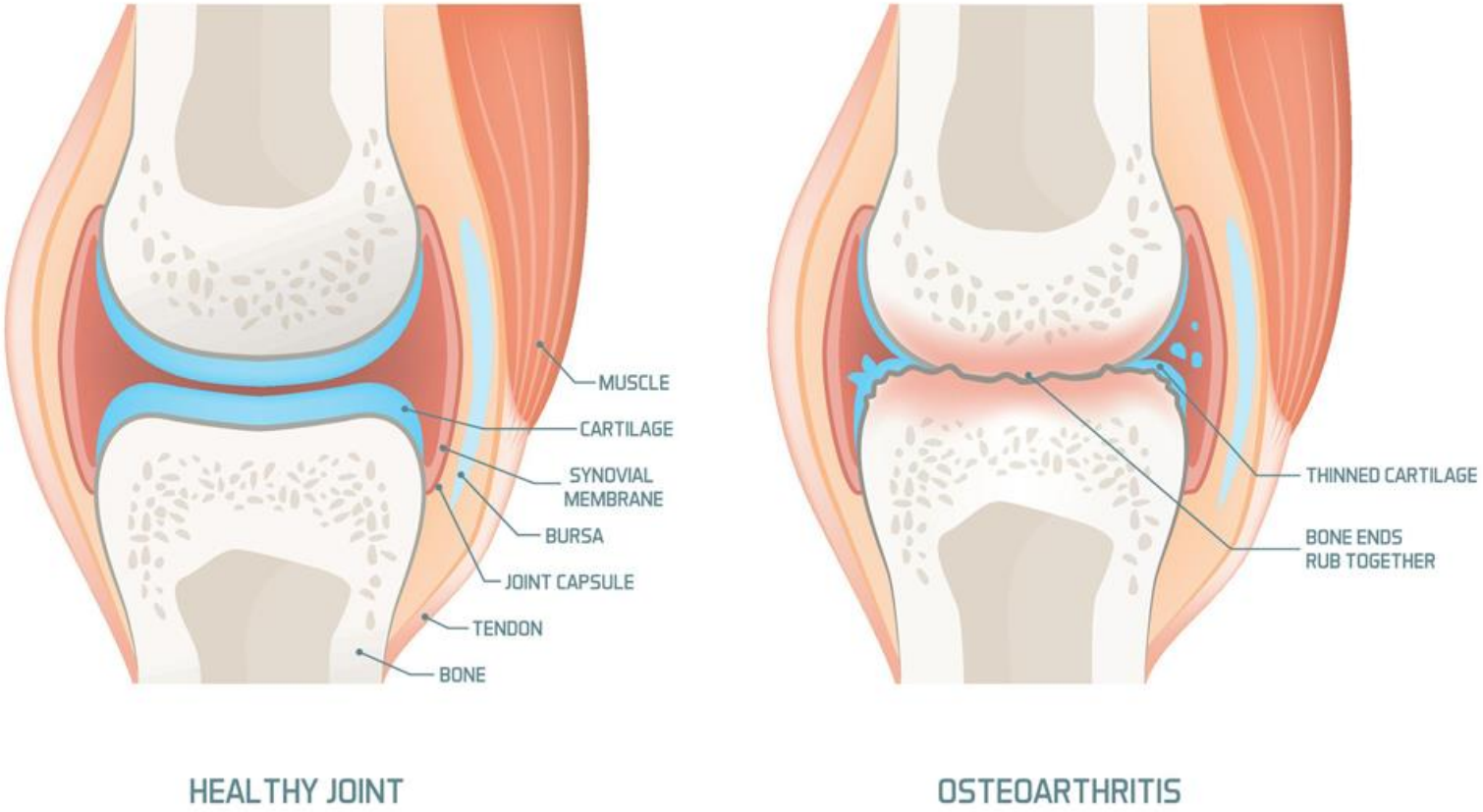


B. Osteomyelitis

Definition:

- Infection of the bone, with infection or inflammation of surrounding soft tissue
    - Acute osteomyelitis:

Infection of short-duration

Characterized by suppuration (ie. abscess), but not Biofilm

Often hematogenous osteomyelitis

No osteonecrosis yet

Systemic symptoms common

B. Osteomyelitis

- Chronic Ostemyelitis
  - Long-lasting infection- Months to years
  - Characterized by necrotic bone and bacterial colonies in protein/polysaccharide matrix (biofilm)
  - Often no systemic symptoms

B. Osteomyelitis

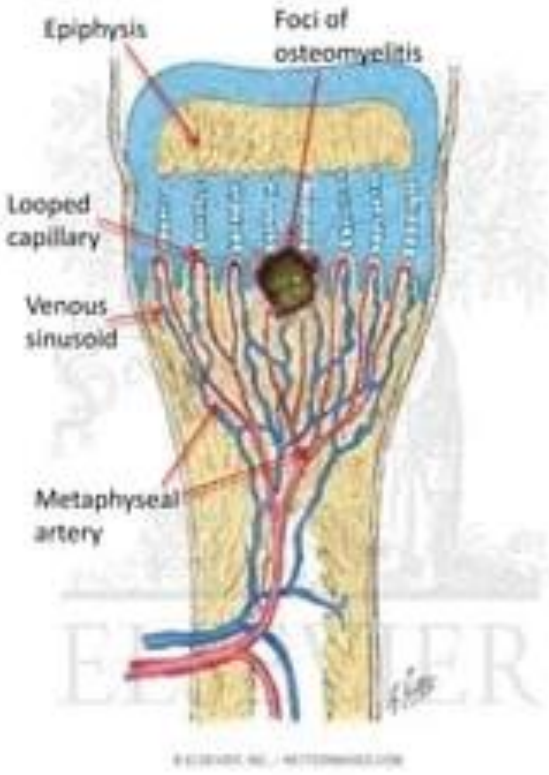
Aetology

- Hematogenous seeding from remote source
  - Most common form in young children
  - Sluggish metaphyseal capillaries
- Contiguous spread from soft-tissue or joint infection
  - Common in older adults near arthroplasty
  - Lower extremity infections related to diabetes or vascular disease
- Direct inoculation from penetrating trauma or surgery
  - Common in young adults

B. Osteomyelitis

Pathophysiology

- Bacteremia seeds long bones commonly around epiphyseal endplates
  - Slow capillary metaphyseal blood flow deposit bacteria
- Local inflammatory response increases intramedullary pressure
  - Occludes normal blood flow causing necrosis
  - Forces infection to break through cortex and form subperiosteal abscess
- Erosion through periosteum compromises bone blood supply further, worsening necrosis



- Terminal branches of metaphyseal artery form loops at growth plate and enter irregular afferent venous sinusoid. Blood flow slowed and turbulent, predispose to bacterial seeding plus, lining cell have little/no phagocytic action made it favourable for bacteria.

B. Osteomyelitis

Pathophysiology

- SAC form in center of abscess surrounded by fibrin deposits
- S. aureus releases coagulase and von Willebrand factor-binding protein
  - Activates prothrombin
  - Polymerizes fibrin network around SAC
  - Protects from immune clearance
  - Immune cells unable to penetrate network
- Bacteria binding to sequestrum release extracellular polymeric substance matrix
  - Forms biofilm glycocalyx
  - Reduced O2 tension and metabolic activity of bacteria
  - Barrier prevents immune clearance and penetration of antibiotics

B. Osteomyelitis

Pathophysiology

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B. Osteomyelitis

Pathophysiology

- S. aureus and epidermidis can become intracellular inside osteoblasts
  - Further perpetuates chronic infection by limiting clearance by host immune system
- Staphylococcus surface-associated material (SAM) stimulates osteoclast activity
  - release of IL-1, IL-6, TNF-α.
- S. aureus protein A (SpA) binds to osteoblasts affecting metabolic activity and proliferation.
  - leading to osteolysis

B. Osteomyelitis

Pathophysiology

- Necrotic bone separates to form the Sequestrum
  - A mixture of bone and purulence
- Reactive bone may form around the sequestrum, termed Involucum
  - May not completely surround sequestrum
  - Drainage from sequestrum may still occur through incomplete involucrum

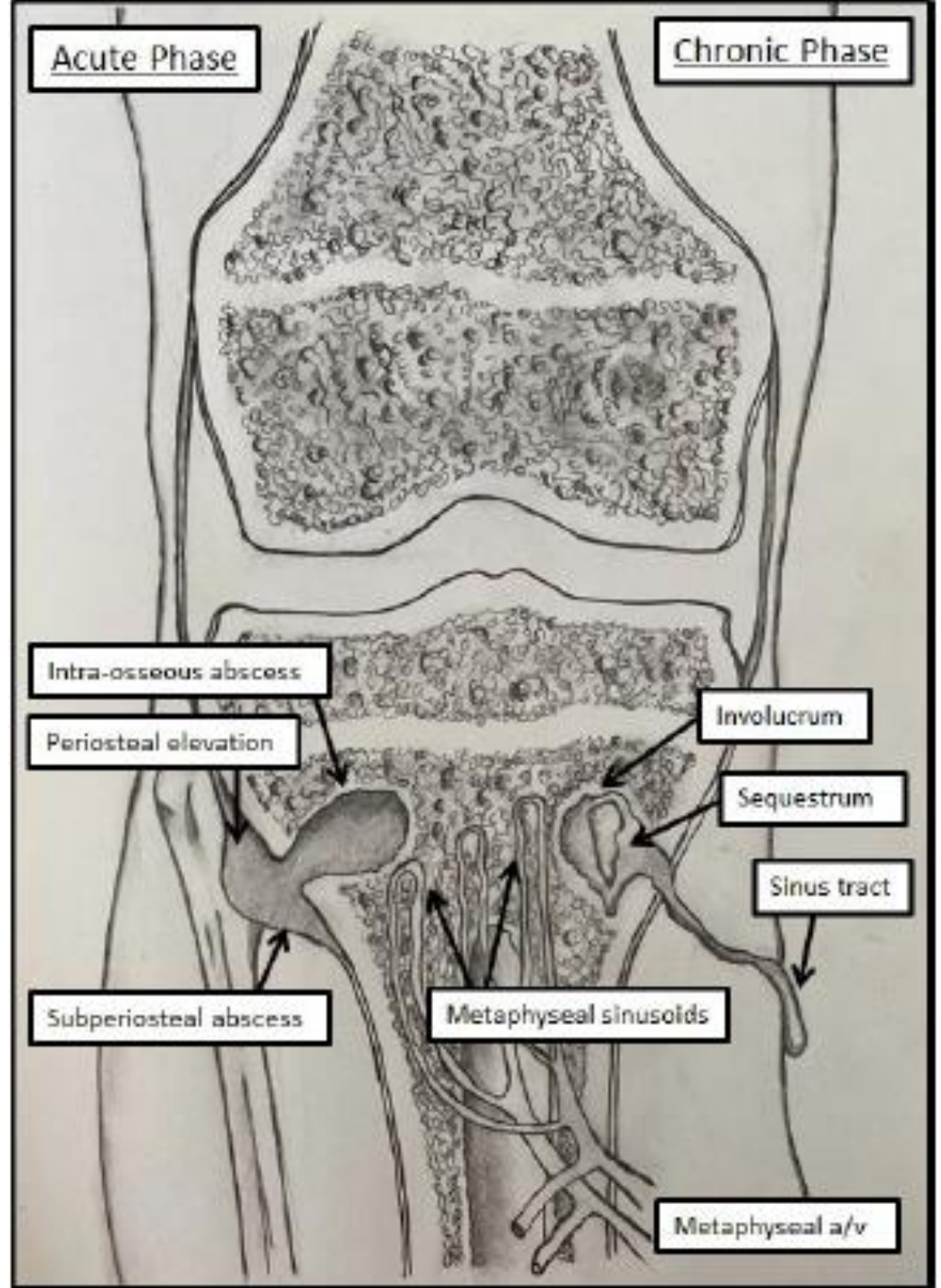
B. Osteomyelitis

Pathophysiology

Organisms vary based on age, type of osteomyelitis and location

- Hematogenous commonly monomicrobial
- Contiguous or direct inoculation can be monomicrobial or polymicrobial

- Children -S. aureus> S. pneumoniae
  - Sickle cell predisposes to Salmonella but *S. aureus* still more common
- Adults -S. aureus, coag-neg Staph
  - S. aureus overall most common
  - Incidence of osteo increased with incidence of diabetes in population


Acute Phase
Chronic Phase
Intra-osseous abscess
Periosteal elevation
Involucrum
Sequestrum
Sinus tract
Subperiosteal abscess
Metaphyseal sinusoids
Metaphyseal a/v

B. Osteomyelitis

Clinical Features

- Acute
  - Pain
  - Erythema
  - Edema
  - +/- Fever (more common in pediatric)
  - Associated wound or sinus tract if direct/contiguous source
- Chronic
  - Generalized/systemic signs less common
  - Localized pain and edema
  - Sinus tract, non-healing ulcerations in vasculopath
  - Vertebral may present with neurological symptoms

B. Osteomyelitis

Diagnosis

- Clinical Features
- Laboratory
  - FBC, ESR,CRP
  - Imaging: X-ray, etc
  - Culture + Sensitivity of bone biopsy (Gold std)

B. Osteomyelitis

Treatment

- Surgery (Debridement + sequestrectomy)
- Bone cement with vancomycin
- Oral Antibiotics
    - Cephalosporins
    - Fluoroquinolones
    - Combination therapy