MUSCULOSKELETAL PATHOLOGY-3

OSTEONECROSIS, OSTEOMYELITIS, FRACTURE

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OSTEONECROSIS

- Also known as: avascular necrosis
- Ischemic infarction of bone and bone marrow
- Most patients are middle-age adults

Caused by:

- Vascular injury (surgery, trauma, vasculitis)
- Drugs (corticosteroids)
- Radiation
- Thrombosis (sickle cell disease)

TYPES: MEDULLARY AND SUBCHONDRAL

- Medullary: infarction of trabecular bone and bone marrow, the cortical bone is spared due to presence of collateral circulation there
- Subchondral: appears triangular (wedge)- shape, results in collapse of bone, fracture and sloughing of articular cartilage
- The overlying articular cartilage is intact due to nutrients in synovial fluid
- Microscopically: no visible osteocytes (empty lacunae)
- Osteoclasts from adjacent viable area start resorption of dead bone
- Repair of subchondral infarction is slow

SYMPTOMS

- Pain, begins in association with activity, then becomes constant
- If articular cartilage is sloughed, secondary osteoarthritis develop

OSTEOMYELITIS

- Inflammation of bone and bone marrow
- Almost always is infectious in origin
- Can be caused by bacteria, viruses and fungi

PYOGENIC OSTEOMYELITIS

- Caused by bacterial infection
- Organisms reach bone either by:
- A) Hematogenous route
- B) Extension from adjacent site (joint, soft tissue)
- C) Direct implantation (open fracture, surgery)
- In children: most commonly hematogenous and affect long bones
- In adults: most commonly secondary to fractures, surgery or diabetes (diabetic foot)

PYOGENIC OSTEOMYELITIS

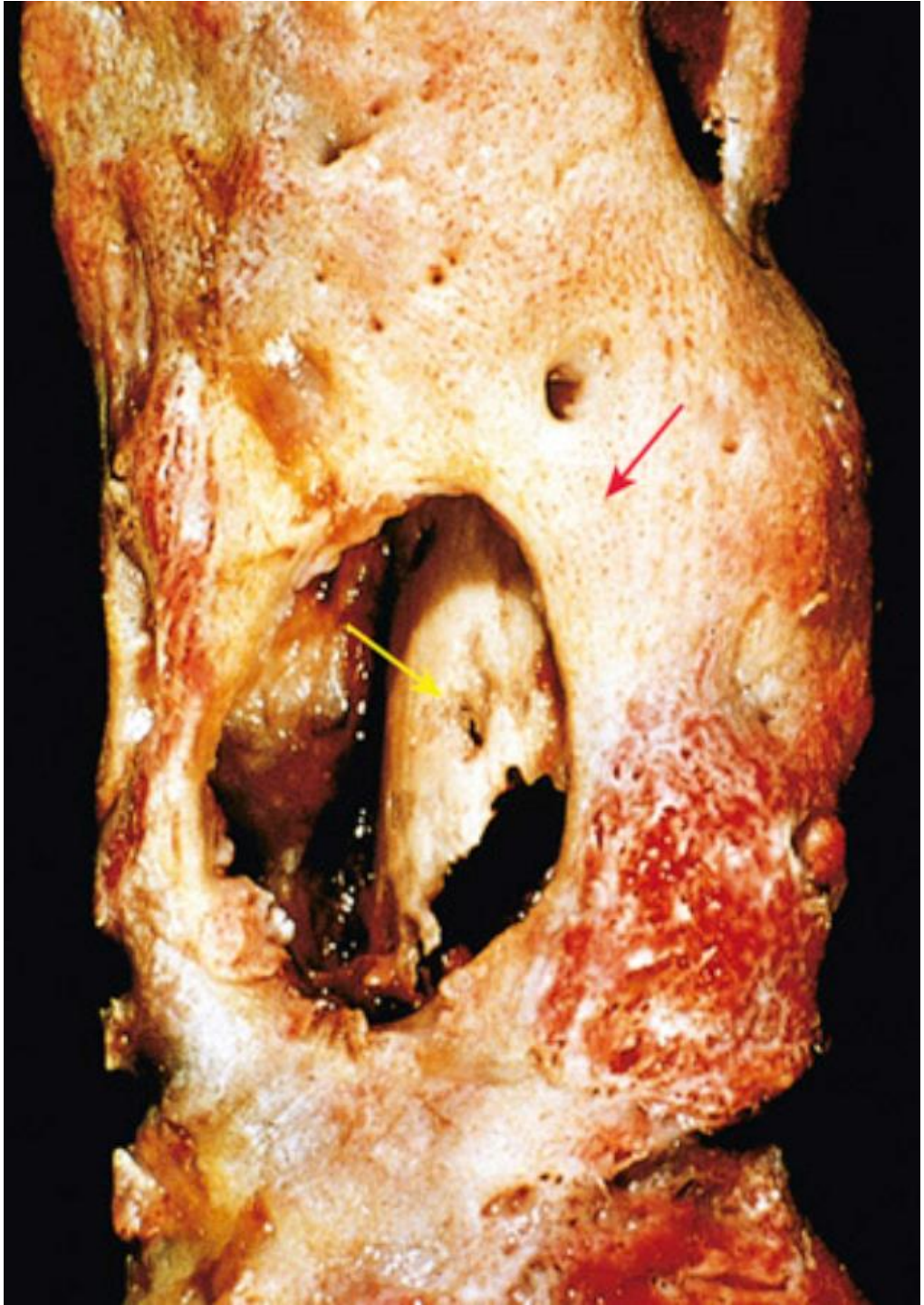
- Staph Aureus is overall the most common microorganism, it binds easily to collagen in osteoid matrix
- In neonates, group B-streptococci and E. Coli are the most common (maternal origin)
- Mixed bacteria is seen in open fracture and surgery
- Salmonella and gram-negative bacteria are the most common in patients with sickle-cell anemia

MORPHOLOGY

- Acute phase: neutrophilic inflammation, liquefactive necrosis
- Infection then spread radially, reaching the periosteum
- In children, the periosteum is loosely attached to cortical bone, so it detaches, sub-periosteal abscess forms, dissecting it, compressing blood supply and causing necrosis of bone.
- Bone abscess may spread to adjacent soft tissue, ultimately reaching skin through a sinus tract
- Epiphyseal infection may spread to joint structures, causing septic arthritis and cartilage destruction (disability)

MORPHOLOGY

- After one week of bone infection, chronic inflammatory cells appear (lymphocytes and plasma cells)
- Secreted cytokines cause bone resorption, growth of fibrous tissue and new bone formation
- Dead bone is called sequestrum وشيظ
- Newly formed bone is called involucrum كساء عظمي, appears as a shell around dead tissue



- Resected femur in a patient with draining osteomyelitis. The drainage tract in the subperiosteal shell of viable new bone (involucrum, yellow arrow) shows the original cortex (sequestrum, red arrow), which is necrotic.

CLINICAL FEATURES

- Throbbing pain over affected area
- Fever, leukocytosis
- Unexplained fever in infants
- X-ray and MRI show bone changes
- 5-25% of acute osteomyelitis cases persist, transform to chronic osteomyelitis, manifest as recurrent flares and dormancy
- Complications of chronic osteomyelitis: pathologic fracture, amyloidosis, squamous cell carcinoma in sinus tract, sarcoma in bone (rare)

MYCOBACTERIAL OSTEOMYELITIS

- Affects 1-3% of patients with TB
- Hematogenous spread, or direct extension from lung (ribs, spine)
- Pott disease: vertebral infection, occurs in 40% of osseus TB, affects multiple vertebral bones, destruction to intervertebral discs causing deformity and spinal cord damage, may spread to soft tissue and psoas muscle

FRACTURE

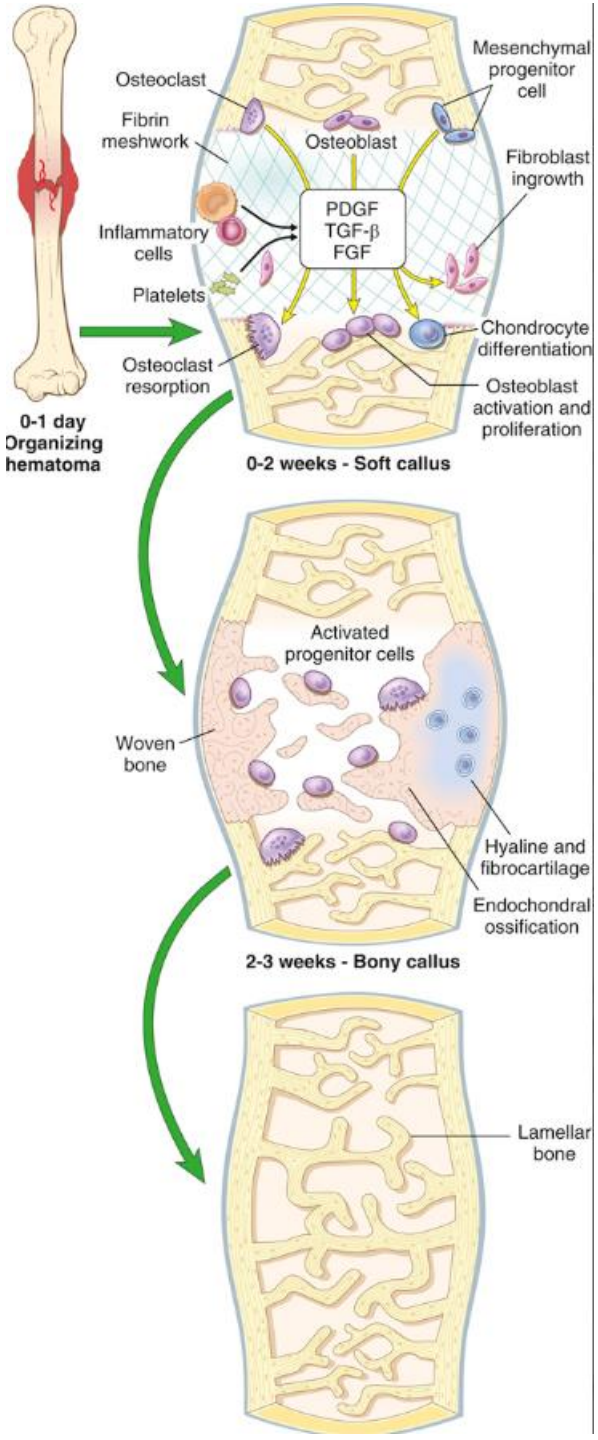
- Loss of bone integrity
- Results from physical force and/or decreased bone strength
- Pathologic fracture (#): called when the bone is weak secondary to disease
- Simple #: skin is intact (vs. compound #)
- Comminuted #: bone is fragmented
- Displaced #: distal ends of bones are mal-aligned
- Stress #: repetitive small forces cause #
- Greenstick #: part of the bone width is fractured, common in infants and children

HEALING PROCESS

- Fracture causes disruption of blood vessels, a hematoma forms
- Hematoma contains fibrin network, which guide inflammatory cells, fibroblasts and new capillaries
- Inflammatory cells and platelets secrete platelets-derived growth factor (PDGF), transforming growth factor-β (TGF-β) and fibroblast growth factor (FGF), activating osteoprogenitor cells and stimulate osteoblasts and osteoclasts.

CALLUS

- After one week, a soft callus دُشبذ (procallus) is formed, which is composed of uncalcified new tissue that fills the cavity
- After two weeks, soft callus is converted to bony callus by deposition of woven bone.
- In some cases, fracture site shows new cartilage formation, which undergoes endochondral ossification
- Callus undergoes remodeling according to weight-bearing forces, reduces in size and shape
- Contour of new bone is re-established and shows lamellar bone
- Formation of medullary cavity is the last step in bone healing



- The reaction to a fracture begins with an organizing hematoma. Within 2 weeks, the two ends of the bone are bridged by a fibrin meshwork in which osteoclasts, osteoblasts, and chondrocytes differentiate from precursors. These cells produce cartilage and bone matrix, which, with adequate immobilization, remodels into normal lamellar bone. FGF, Fibroblast growth factor; PDGF, platelet-derived growth factor; TGF-β, transforming growth factor-β.

SUBOPTIMAL HEALING OF BONE

- Comminuted # causes deformity
- Inadequate immobilization: movement of callus causes delayed union of bone or non-union. Non-union results in cystic degeneration and a false joint formation
- Infection of fracture site (compound open #)
- Malnutrition
- Diabetes
- Pathologic fracture (needs surgical immobilization)