NURS 821 Alterations in the Musculoskeletal System

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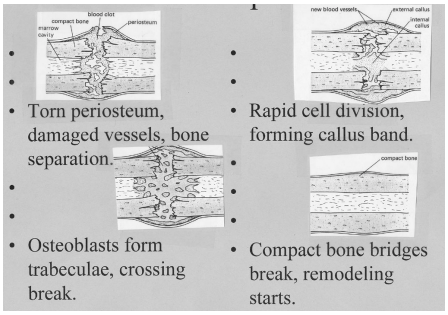
Lecture 12

Part 3 Bone Disorders

Bone Repair



- Torn periosteum, damaged vessels, bone separation.
- Osteoblasts form trabeculae, crossing break.
- Rapid cell division, forming callus band.
- Compact bone bridges break, remodeling starts.

Osteomyelitis

- **Definition**: Infection of bone caused by bacteria*, fungi, parasites, or viruses
- Categorized by organism mode of entry:
- Exogenous
  - Enters from outside body
    - Open fractures, penetrating wounds, surgical procedures, bites
- Endogenous
  - Hematogenous-frequently found in infants, children, elderly
  - Usually due to sinus, ear, dental, cutaneous, GI, URI, total joint replacements
  - Adults-more common in spine, pelvis, small bones

Osteomyelitis

- **Etiology**: usually Staph aureus
- Pathophysiology:
  - Inflammatory response
  - Exudate seals bone canaliculi
  - Exudate extends into metaphysis and marrow cavity into cortex
  - Children-cortical exudate causes abscesses that lift periosteum off underlying bone causing disruption of blood flow
  - Causes necrosis and death-sequestrum-devitalized bone
  - Intense osteoblastic response lays new bone (involucrum) with openings for exudate to escape

Osteomyelitis Complications

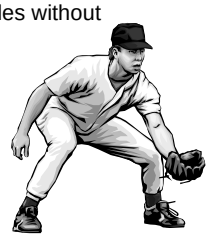
- Pain at inflamed area
- Constitutional signs of infection
- Soft tissue infection manifestations

Osteoporosis

- **Definition**: Most common metabolic bone disease resulting in decreased density of bone mass; remaining bone is histologically and biochemically normal but insufficient for skeletal integrity and support
- Types:
  - Generalized: involves most of axial skeleton
  - Regional: involves a segment of skeleton

Osteoporosis

- Population at risk:
  - Post-menopausal females without supplemental estrogen
  - Elderly
  - Malabsorption or decreased Ca uptake
  - Hormonal imbalances
  - Iatrogenic



Osteoporosis

- **Pathophysiology**: Disrupted prolonged remodeling cycle may take two years (normal – four months).
- Normally bone replacement constantly follows resorption. Equilibrium may be disrupted by:
  - Increasing number of activated multi-cellular units, increasing frequency of units activated, increasing resorption rate, delayed bone formation, cell deficiency in multi-cellular unit

Osteoporosis Manifestations

- Pain
- Deformity: Kyphosis
- Pathological fractures
  - Fatal complications

Osteomalacia

- **Definition**: Rare metabolic disease characterized by inadequate and delayed mineralization of osteoid in mature compact spongy bone. Called rickets in children.
- **At risk population**: Elderly, low birth weight premature babies, persons on rigid macrobiotic diet.

Osteomalacia

- **Etiology**: inadequate vitamin D
- **Pathology**: Normal remodeling cycle through osteoid formation but mineral calcification and deposition doesn't occur due to inadequate vitamin D
- **Unchanged bone volume, but replaced with soft osteoid (not rigid) bone**; trabeculae in spongy bone becomes thin; compact bone develops long channels and irregularities.

Manifestations of Osteomalacia

- Gross deformities of long bones, pelvis, and skull due to osteoid deposition beneath periostium
- Pain and tenderness in skeleton, especially in hips
- Bone fractures and vertebral collapse
- Muscle weakness

Differences Between Osteoporosis and Osteomalacia

- Osteoporosis
  - Decreased bone mass
  - Due to Ca lack
  - X-rays show fractures, loss of density
  - Ca level normal
  - Phosphate normal
- Osteomalacia
  - Demineralized bone
  - Due to Vitamin D lack
  - X-rays show fractures and pseudo-fractures
  - Ca low or normal
  - Phosphate high or normal

Legg-Calve'-Perthes Disease

- **Definition**: A self-limiting common osteochondroses occurring in childhood
- **Incidence**: age 3-10 (peak 6, increased in males 4:1)
- **Etiology**: Unknown, may be viral, infections, trauma, genetic link

Legg-Calve'-Perthes Disease

- Pathophysiology
  - **1st stage**: Over a few weeks, hip soft tissue becomes swollen, hyperemic, widening joint space, capsule bulges
  - **2nd stage**: Over months – years, active avascular necrotic stage - anterior half of epiphysis of femoral head, metaphysical bone at femoral neck, capital epiphysial plate are softened due to hyperemia and decalcification; dead bone invaded by procallus and blood vessels.

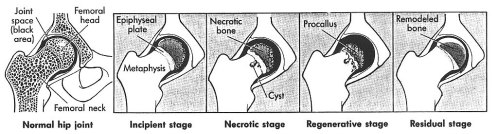
L-C-P Disease

- **3rd stage**: Lasts 2-4 years; dead femoral head replaced by procallus and new bone which collapses and flattens femoral head and shortens femoral neck
- **4th stage**: Residual remodeling stage. New bone is organized into live, spongy bone. May completely restore femoral head in younger children.

Manifestations of L-C-P

- Insidious onset
- Limping
- Pain:
  - Referred to knee, inner thigh, groin
  - Aggravated by activity;
  - May be minimal or absent

Stages of Legg-Calve'-Perthes Disease



Avascular Necrosis

- **Definition**-Bone necrosis due to interrupted blood supply
- **Incidence**-all ages, peak 30-40; M=F
- **Etiology**-
  - Injury-20% of hip dislocations
  - Non-traumatic avascular necrosis
    - Steroids-more severe and bilateral-35%-may interfere w/ fatty acid breakdown blocking blood supply to bone
    - alcohol –etiology same as steroids

(NIAMS, 2000)

Avascular Necrosis Manifestations

- Early: none
- Joint pain with weight bearing
- Pain at rest
- Pain: mild – severe
- With progression and collapse of bone and joint surfaces – pain progresses or dramatically increases
- Progression varies – months to years

(NIAMS 2000)

Scoliosis

- Types:
  - Postural scoliosis
  - Structural scoliosis

Musculo-skeletal Deformities