Degenerative joint disease (DJD)

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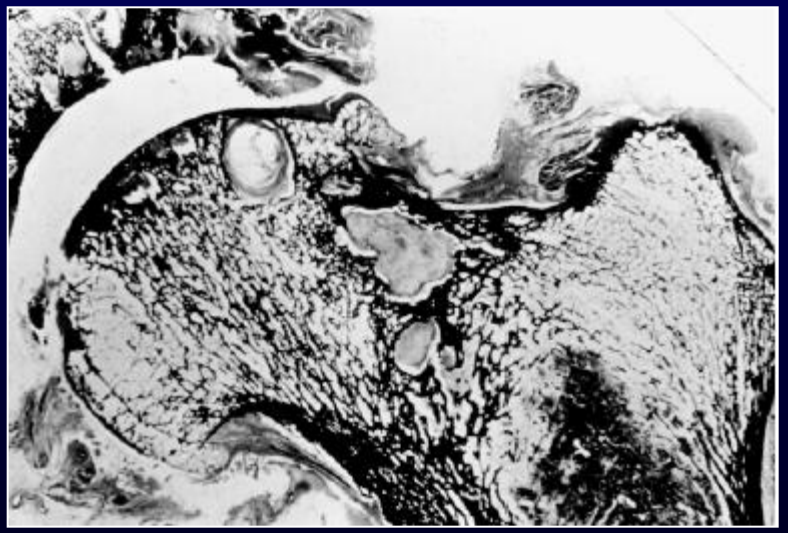
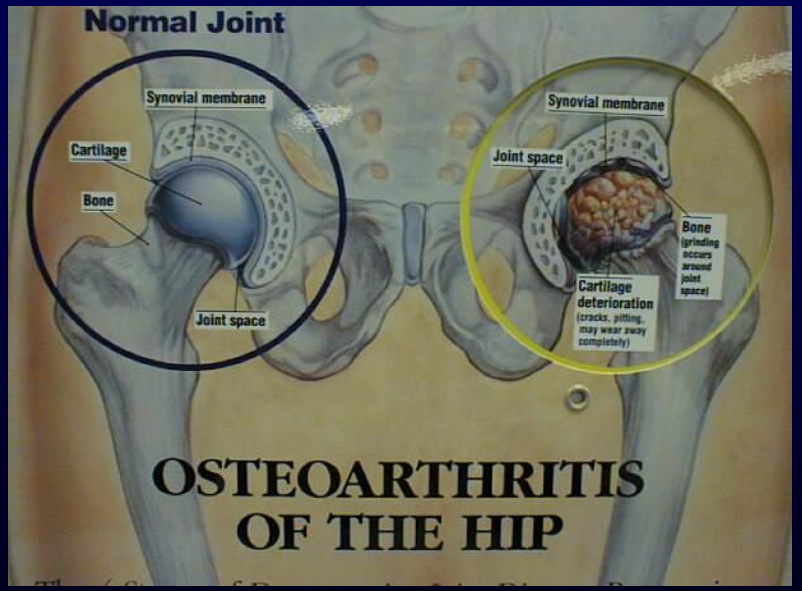


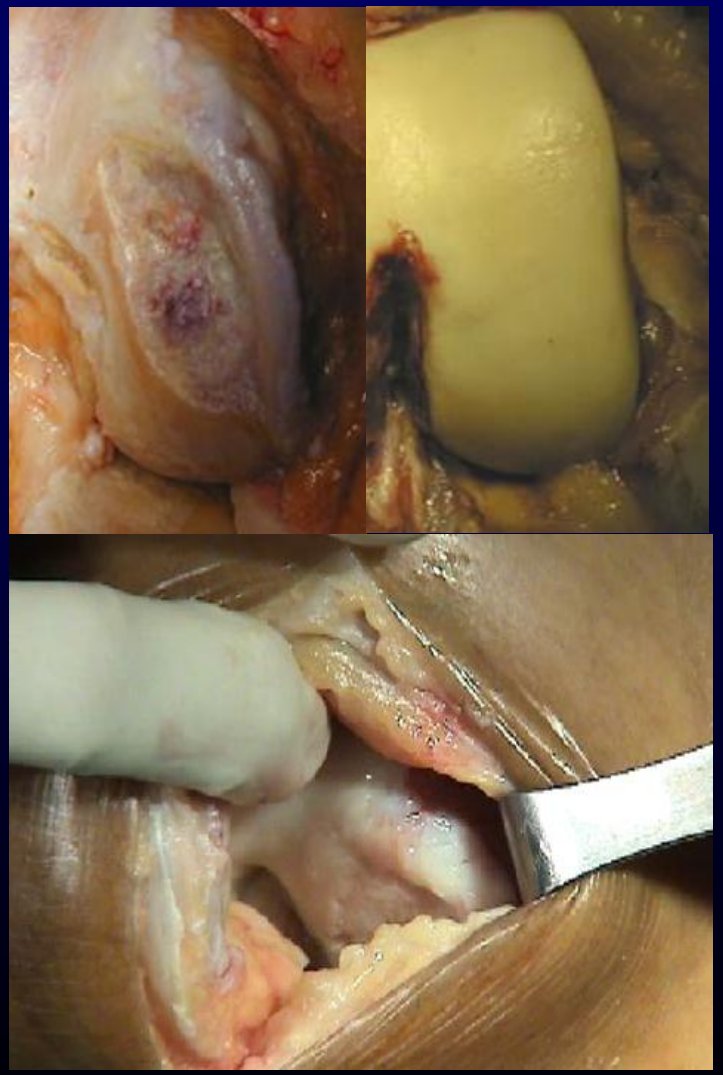
What is DJD?

- Commonly known as “osteoarthritis”
- Affects 80% of people > 60 yrs
- Disease character:
  - Different etiologies
  - Similar
    - Biologic
    - Morphologic
    - Clinical outcomes



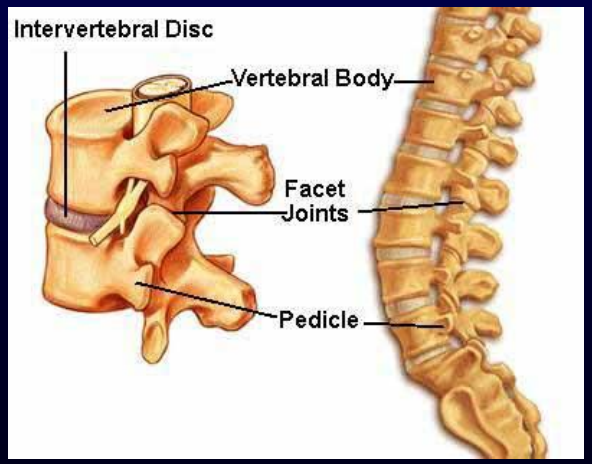
What is DJD?

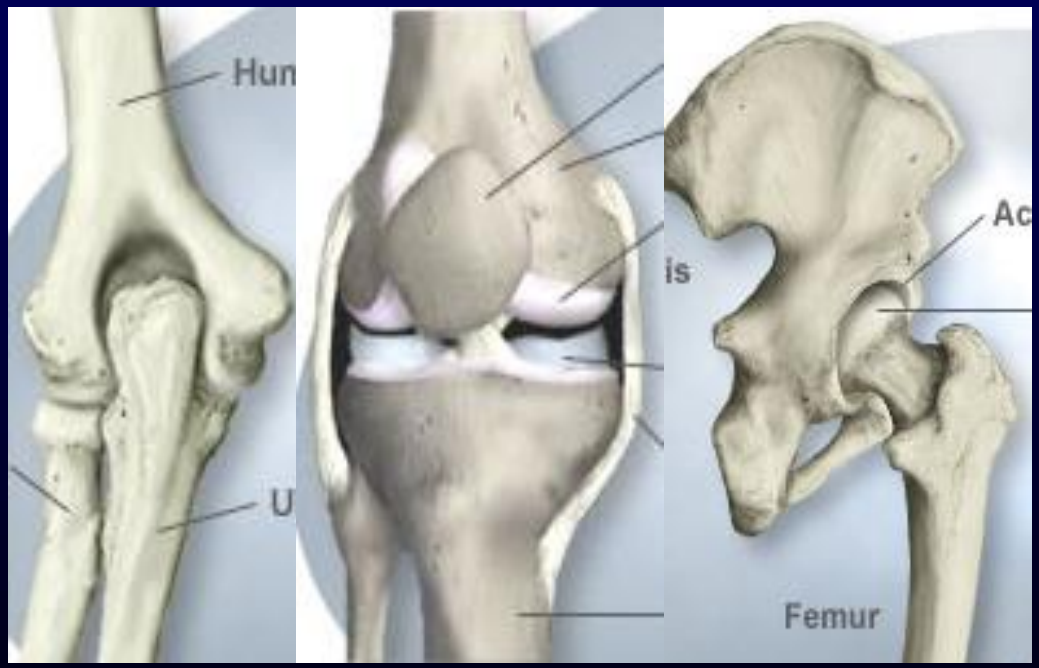
- Articular cartilage
  - Progressive loss
- Subchondral bone
  - Remodeling, sclerosis
  - Bone cysts, osteophytes
- Ligament
  - Attenuation
- Synovium and capsule
  - Thickening



Normal joint

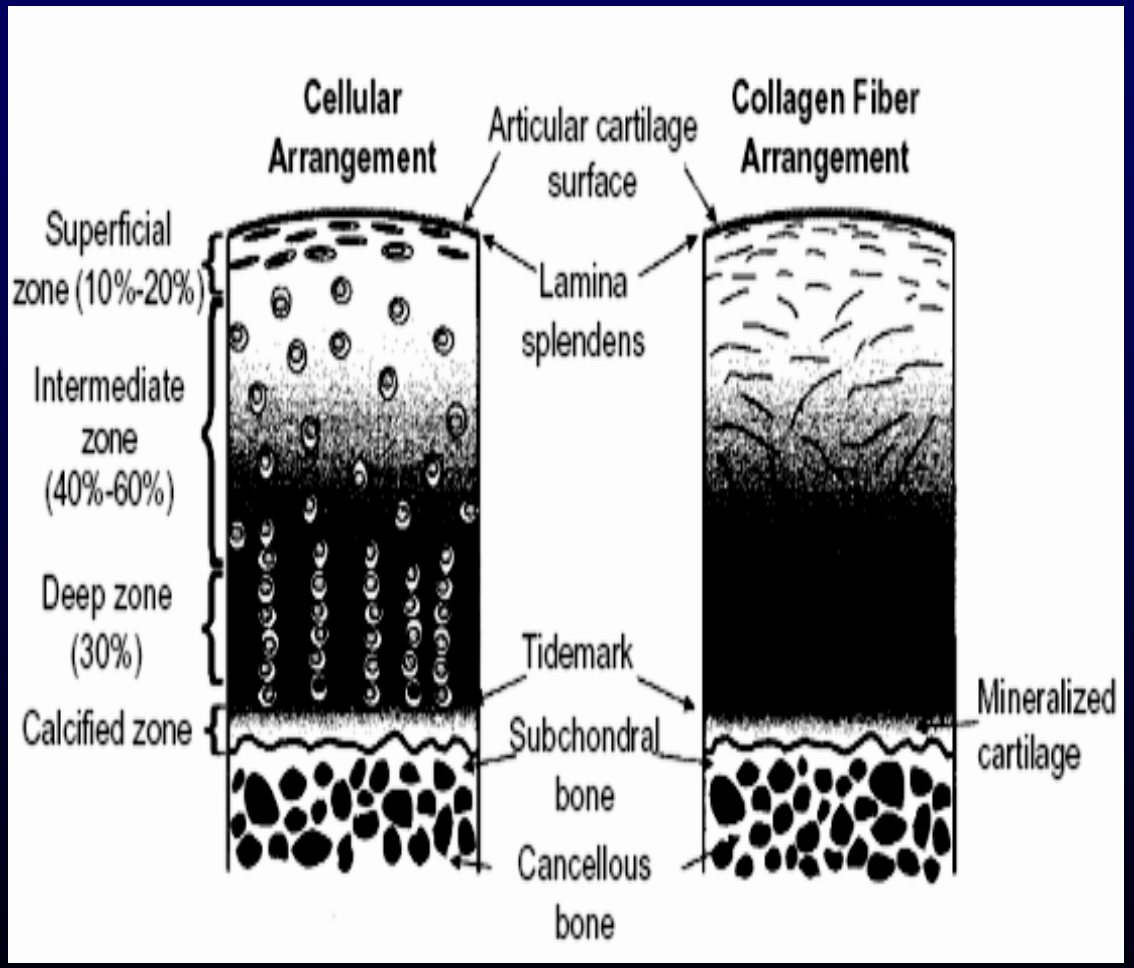
- Painless
- Mobile
- Stable





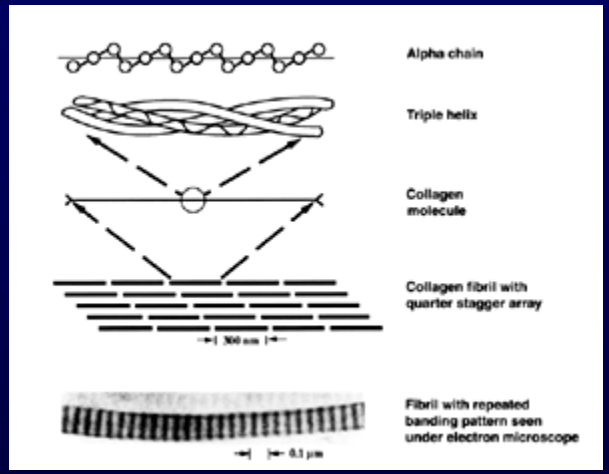
Mature articular cartilage

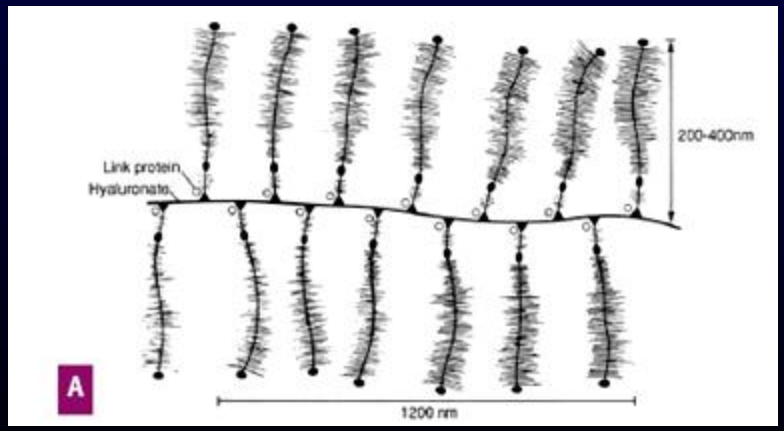
- 5 layers
  - Avascular
  - Aneural
  - Alymphatic
- Matrix
- Chondrocyte
- Nutrients
  - Diffusion
  - Synovial fluid

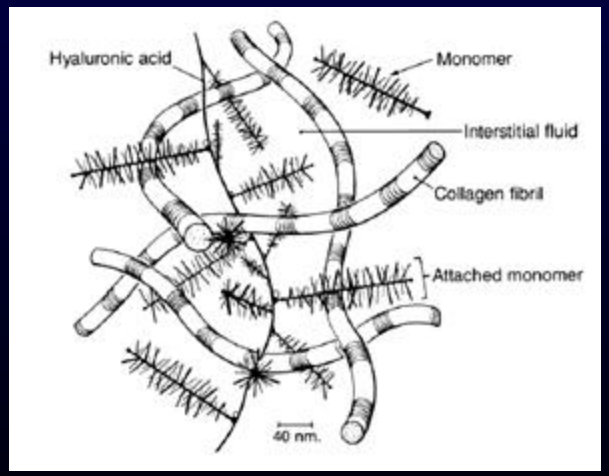


Articular cartilage structure

- Complex extracellular matrix 95%
  - Water: 65-80%
  - Collagen 10-20%
    - Type II 90-95%
    - Type V, VI, IX, X and XI
  - Proteoglycans 10-15%
- Cellular component 5%

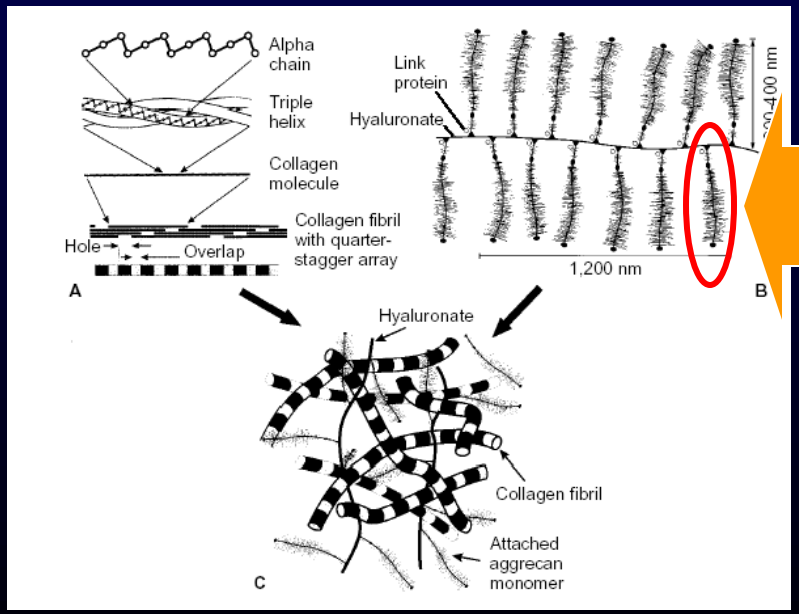


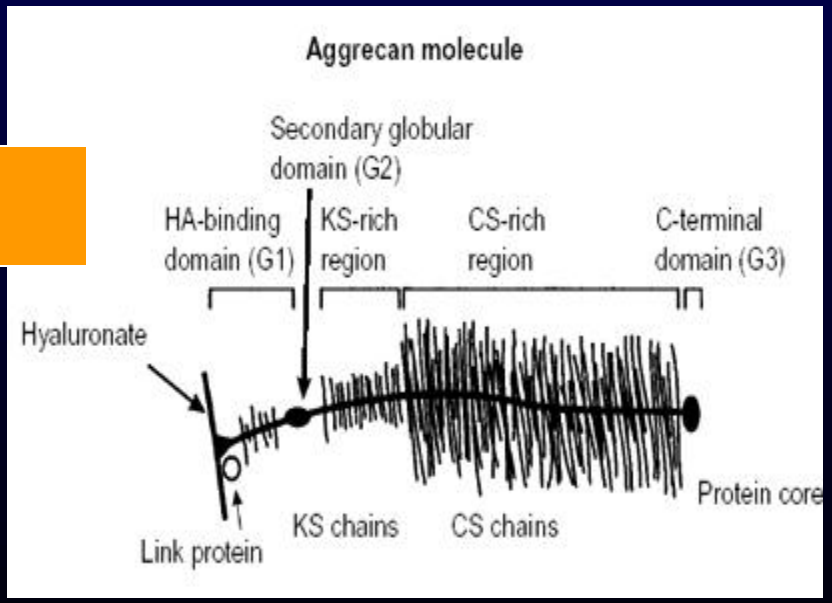




Proteoglycans

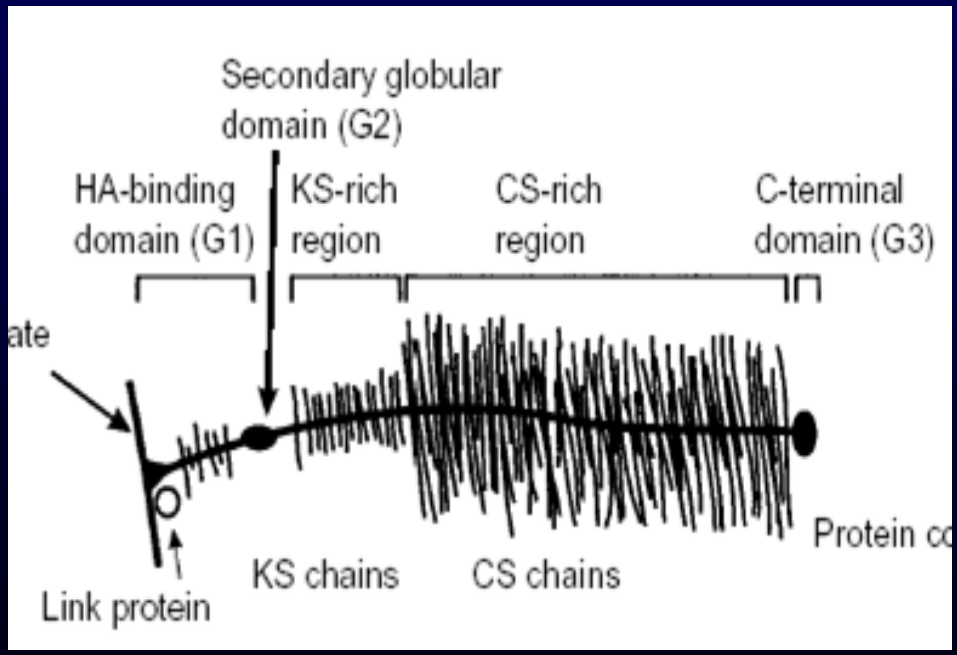
- Large macromolecules
- Produced by chondrocyte
- Chains of glycosaminoglycans (GAGs) attached to a linear core protein





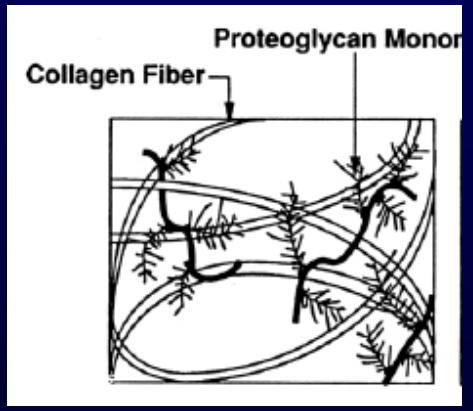
Glycosaminoglycans (GAGs)

- Disaccharide polymer
  - Chondroitin sulfate
  - Keratan sulfate
  - Dermatan sulfate
  - Heparan sulfate
  - Hyaluronic acid



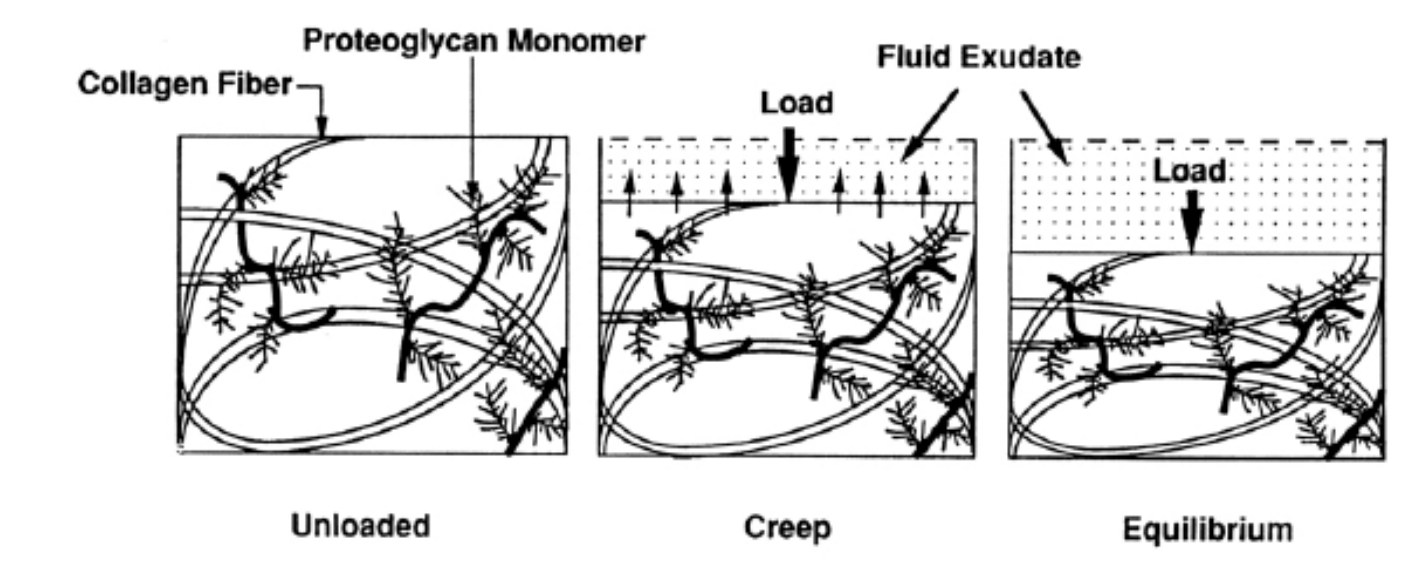
Articular cartilage function

- Water
  - Deformation of cartilage surface due to stress
  - Nutrition
- Collagen
  - Framework strength
  - Tensile strength
- Proteoglycans or aggrecan molecules
  - Negatively charged hydrophilic
  - Compressive strength
- Chondrocytes
  - Matrix and collagen



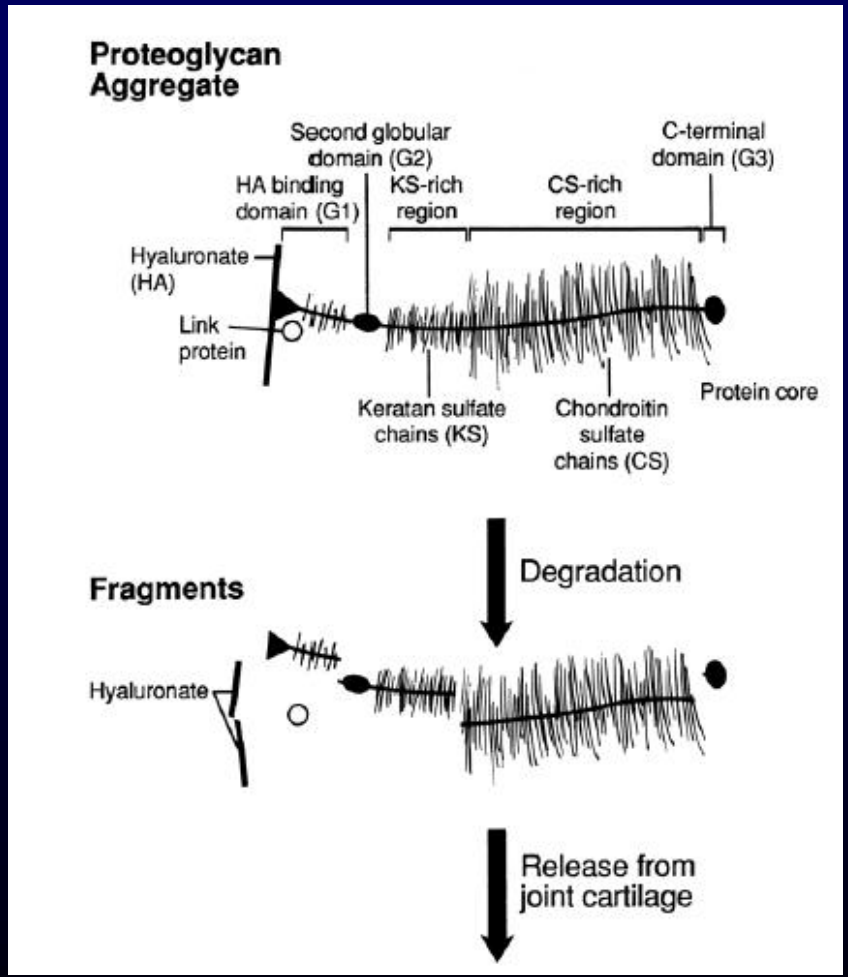
Normal articular cartilage

- Load distribution
- Decrease friction
- Resistance to compressive, tensile, and shear forces
- Minimizing loads on subchondral bone

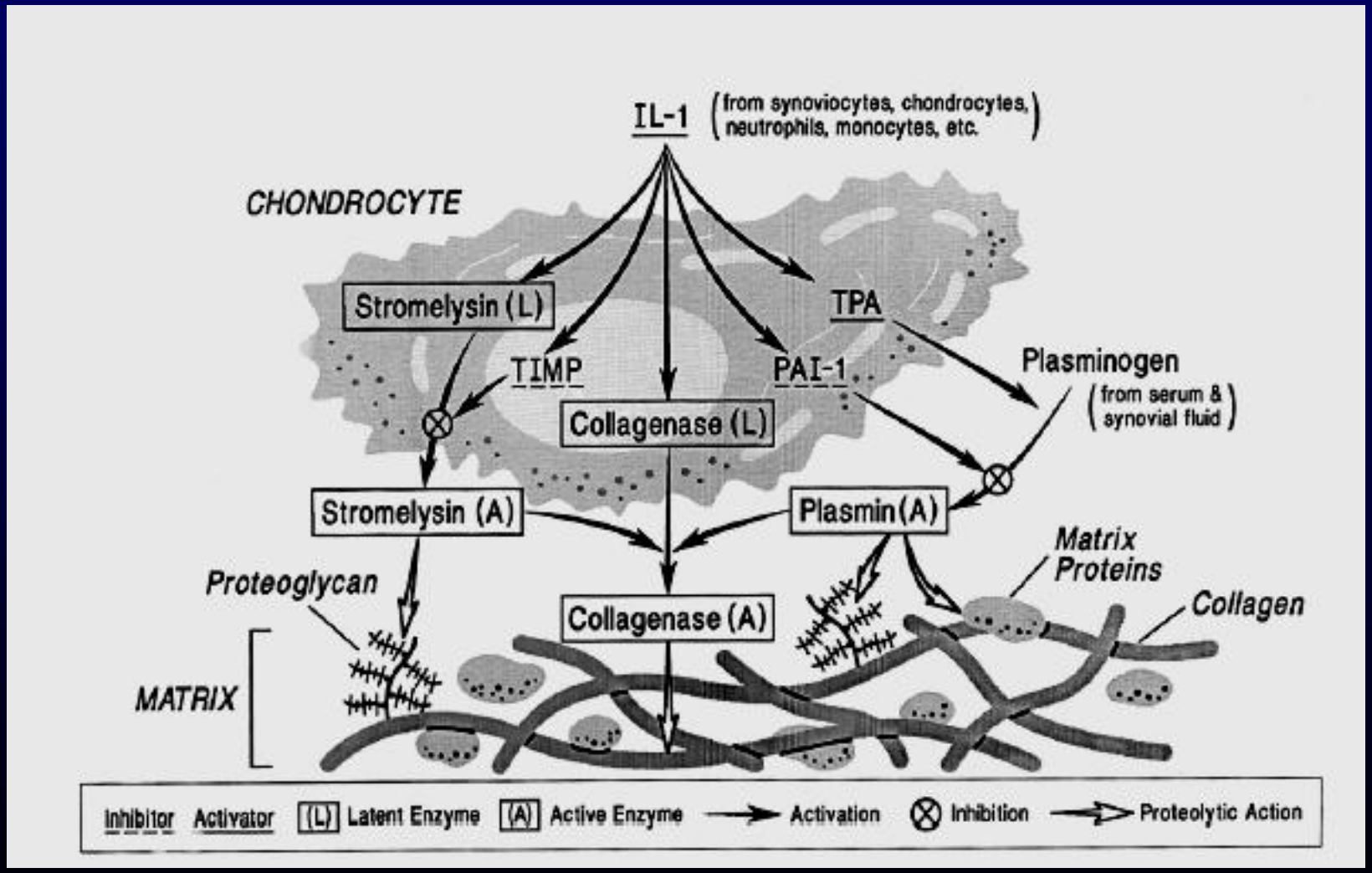


What causes DJD?

- Unknown cause
- Proteoglycans
  - Increase >> decrease
  - Shorter chain
- Water
  - Increase
- Biomechanical properties
  - Decrease

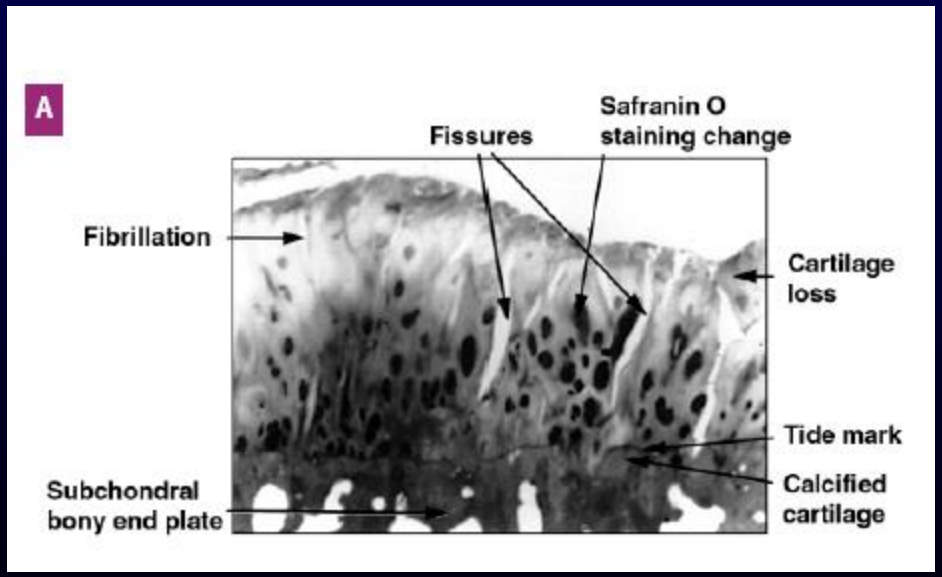


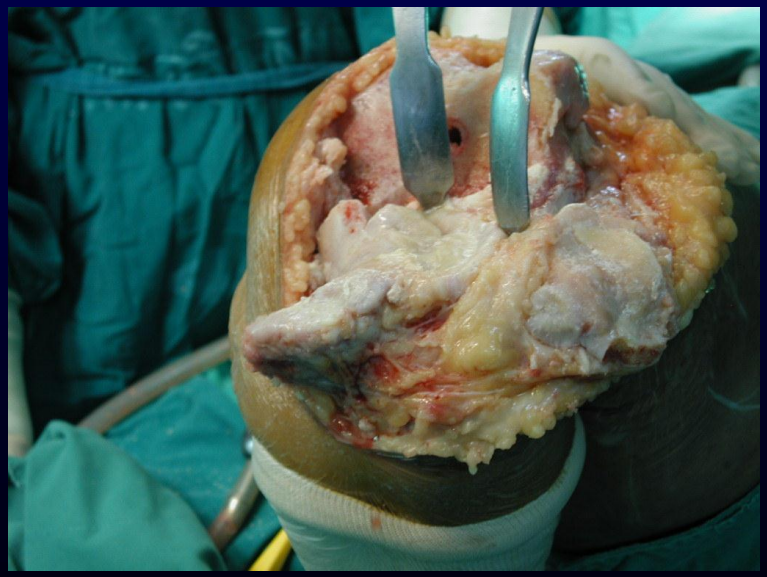
Pathology of DJD



Pathology of DJD

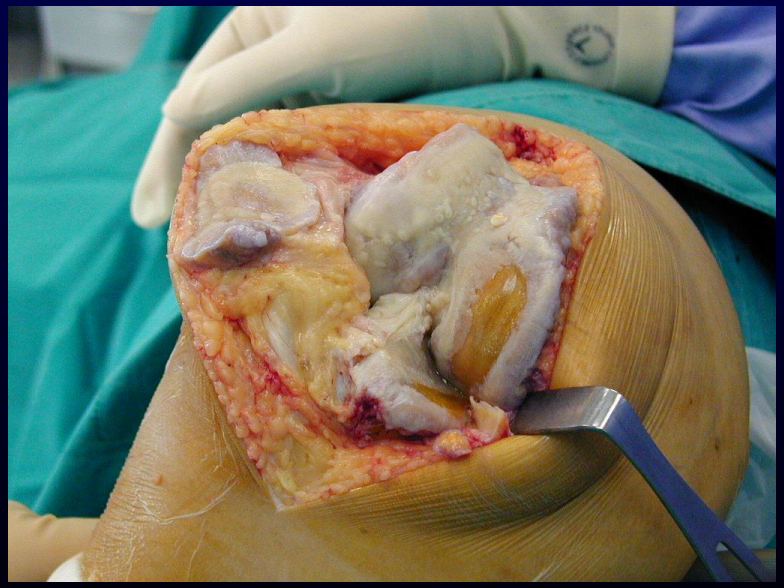
- Fissuring and focal erosive cartilage lesions
- Cartilage loss and destruction
- Subchondral bone sclerosis and cyst
- Large osteophyte formation at the margins of the joint





What contributes poor articular cartilage repair?

- High ratio of matrix to cellular components
  - Poor ability of cartilage to repair
- Low mitotic activity of chondrocytes
  - Negligible healing of cartilage



What are risk factors for OA

- Older age
- Female sex
- Repetitive stress and joint overload
- Genetic factors
- Major trauma
- Obesity
- Prior inflammatory joint disease
- Metabolic disorder
- Proprioceptive defects

What are the symptoms of DJD?

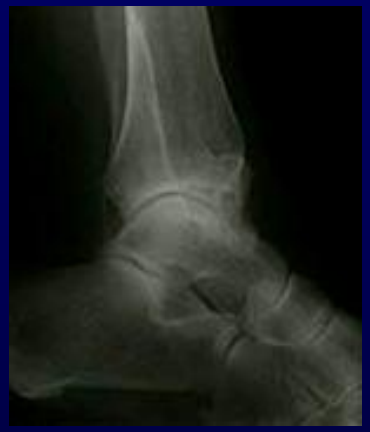
- With or without symptoms for a long time
- Symptoms
  - Subtle development of morning stiffness
  - Pain with movement and activity
  - Improve with rest
  - Decreased range of motion
  - Abnormal sound
  - Unstable joints

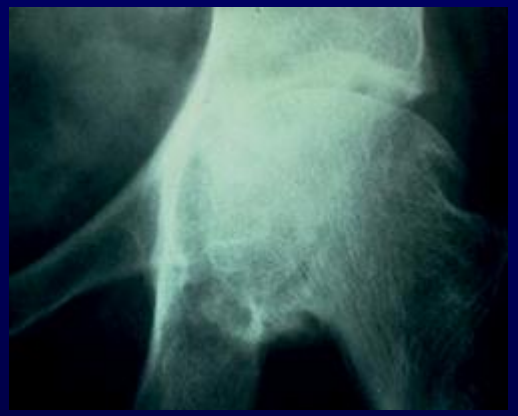
How is DJD diagnosed?

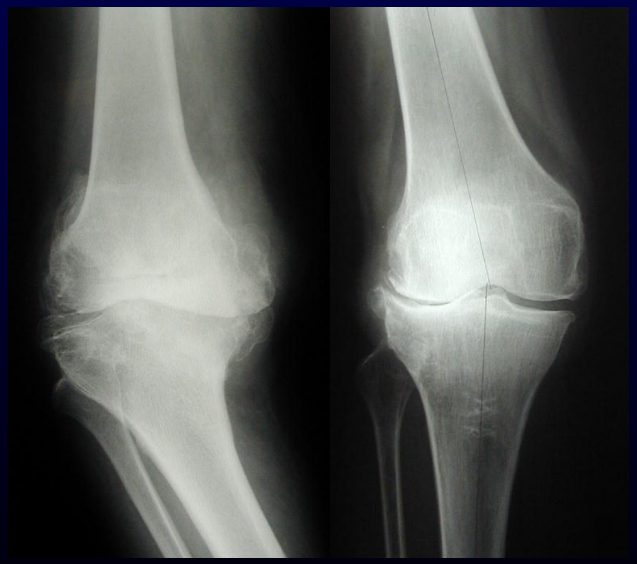
- Symptoms and signs
  - Joint pain
  - Restriction of motion
  - Crepitus with motion
  - Joint effusions
  - Deformity
  - Instability
- Radiographic findings

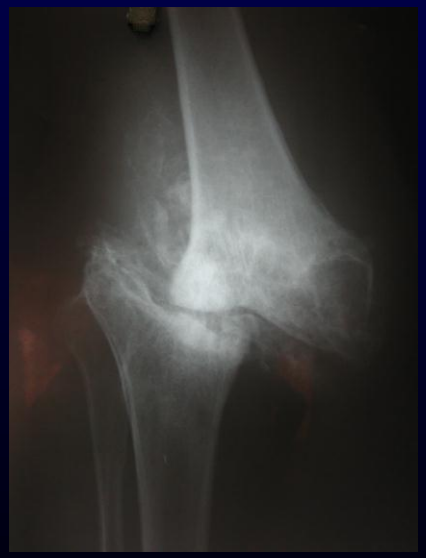
Radiographic findings

- Subchondral bone changes
  - Osteophyte
  - Cyst formation
  - Sclerosis
- Narrowed joint spaces
- Deformity



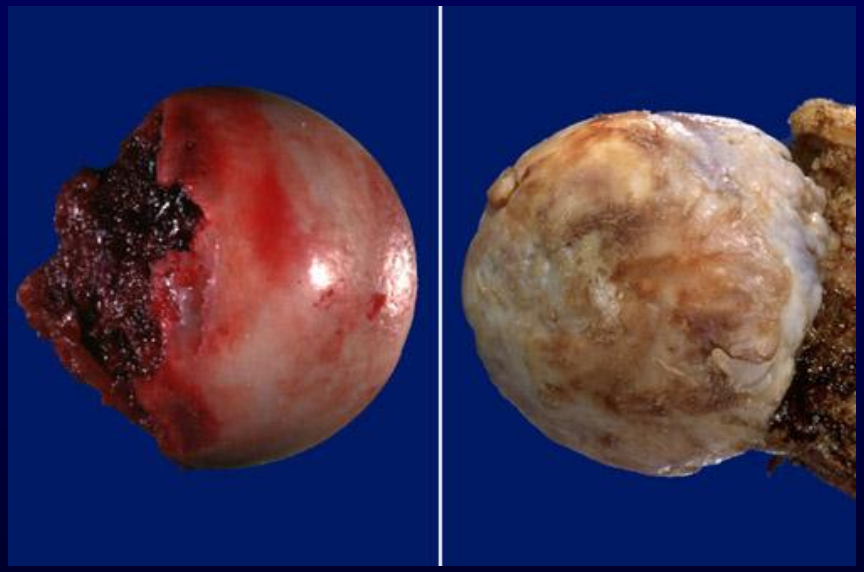






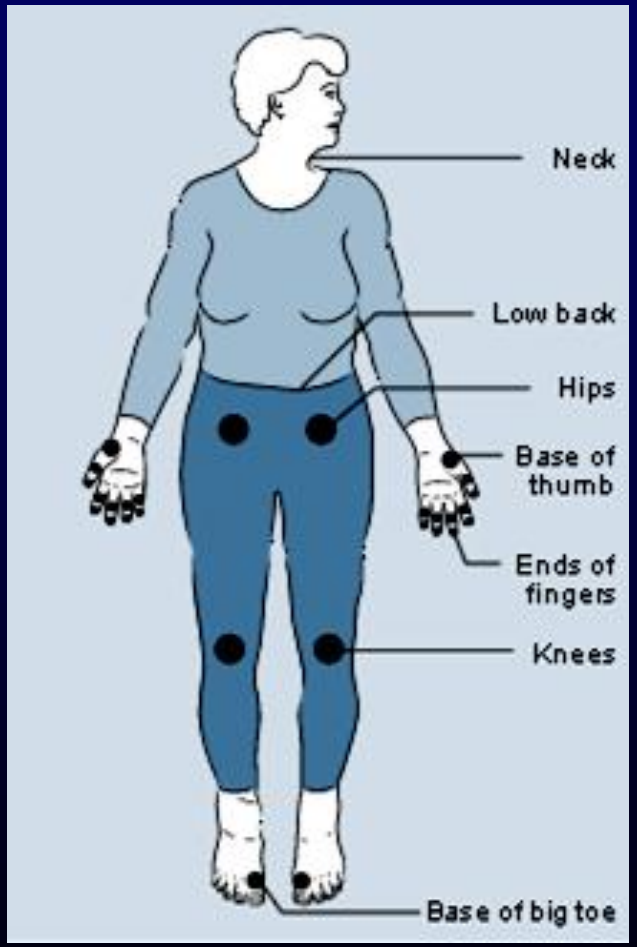
Primary OA

- Idiopathic
- Strong family history
- Alterations in structural macromolecules
- Mutations in the type II collagen gene
- Progressive wear and tear degenerative condition



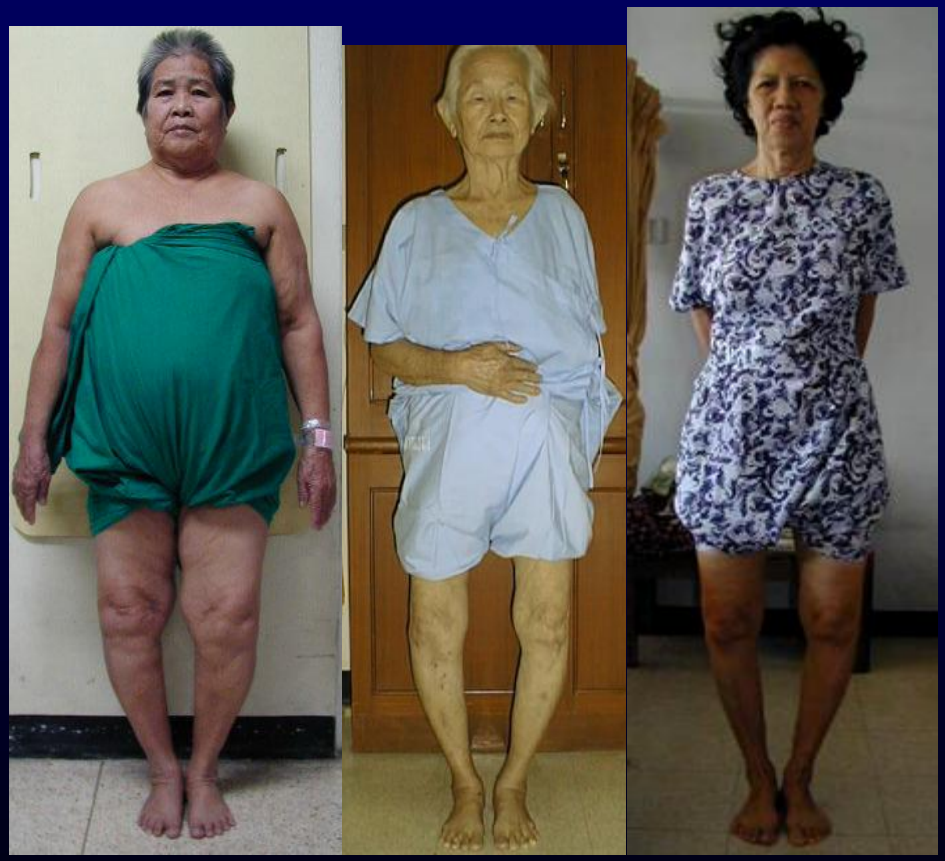
Primary OA

- Common
  - Knee
  - Spine
  - Hip
  - IPJ of hand
  - IPJ of foot



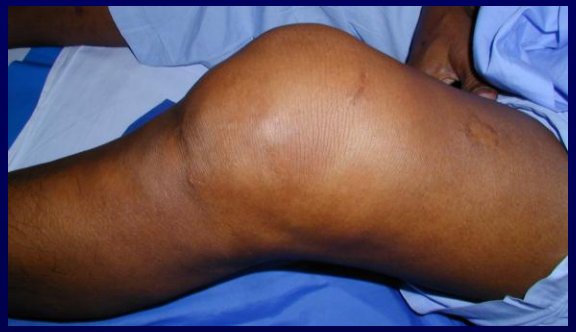
Primary OA: knee

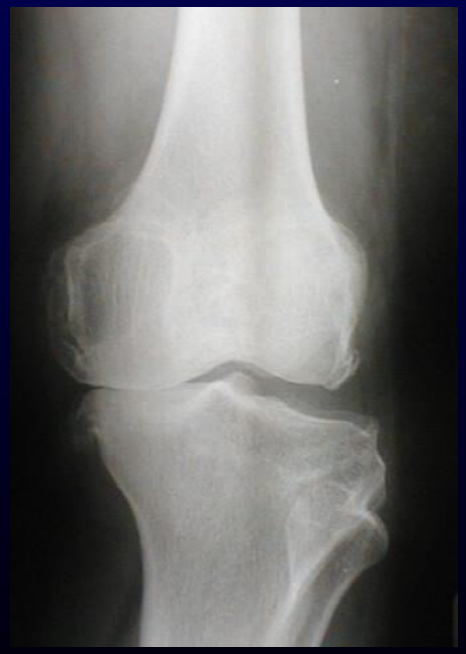
- No relationship between premature arthrosis of the knee and activity levels
- Radiographically osteoarthritis
  - 45 to 64 yrs
    - 20% to 30%
  - > 65 yrs
    - 85%



Primary OA: knee

- Pain
- Stiffness
- Swelling
- Limited ROM
- Limping
- Crepitation
- Deformity





Primary OA: knee

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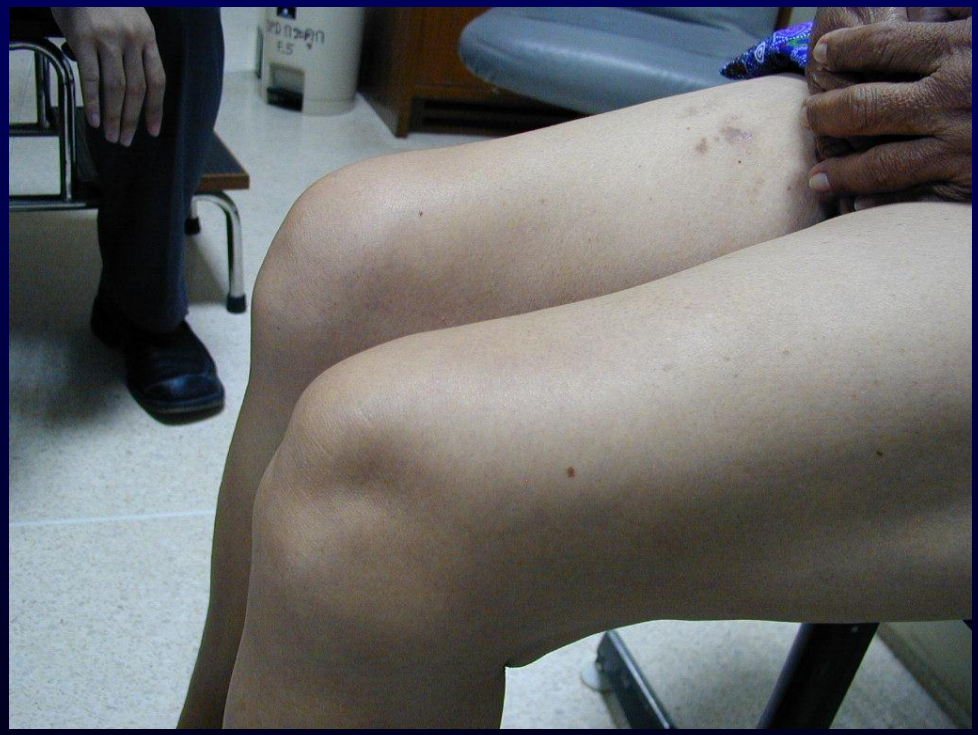
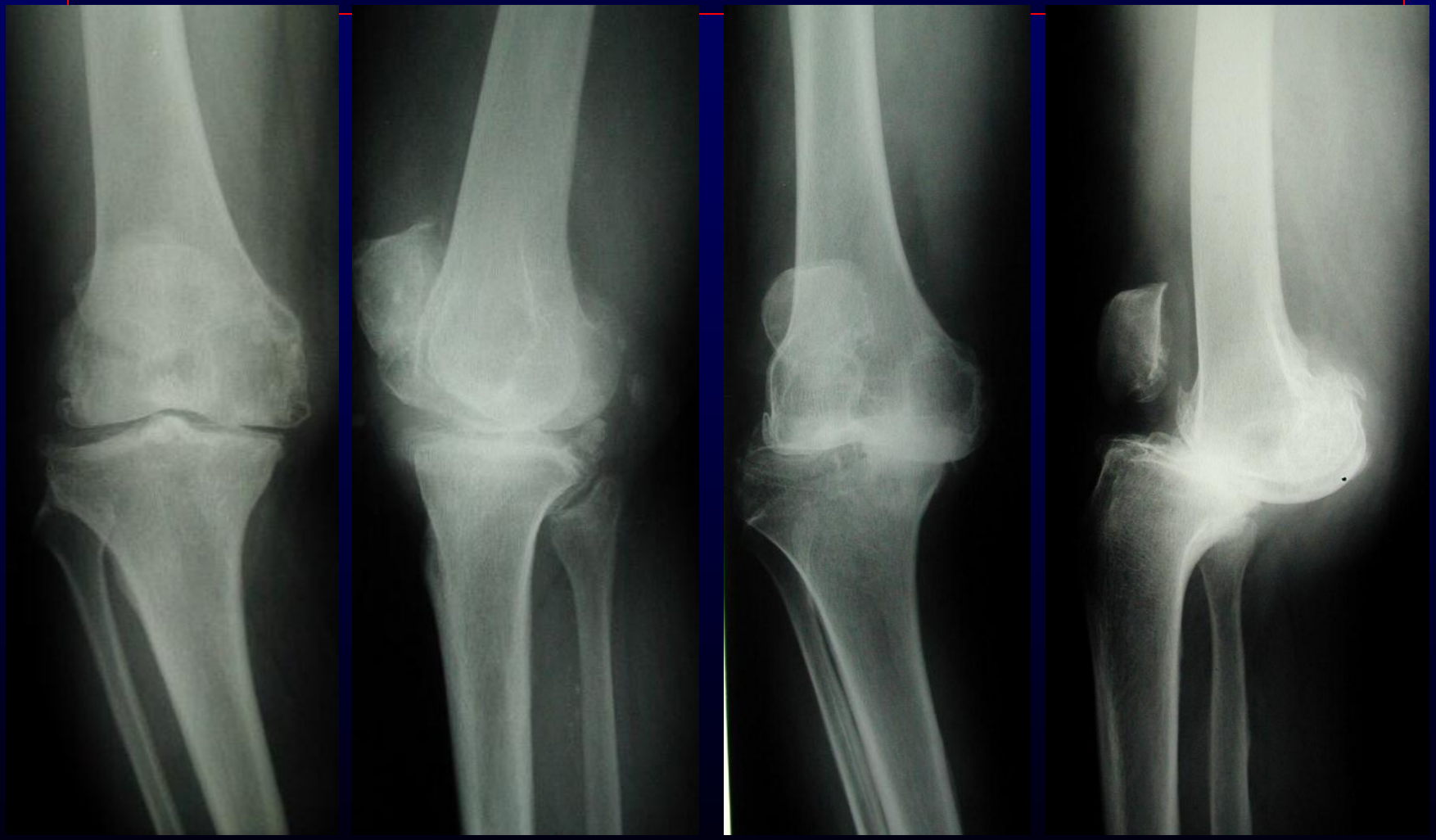


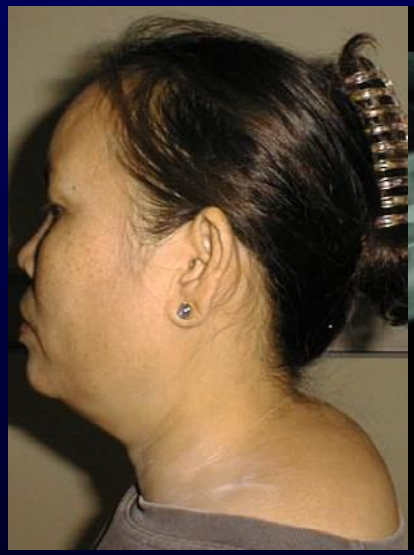
Primary OA: knee

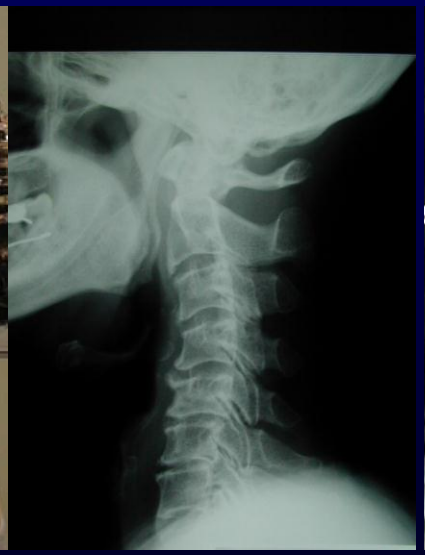
Primary OA: knee

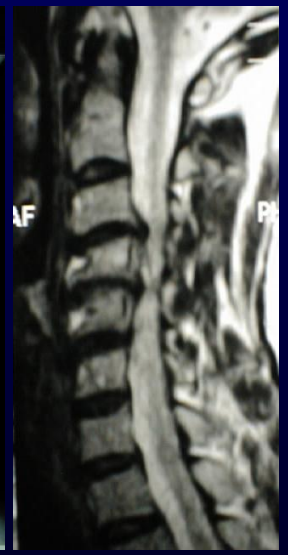


Spine

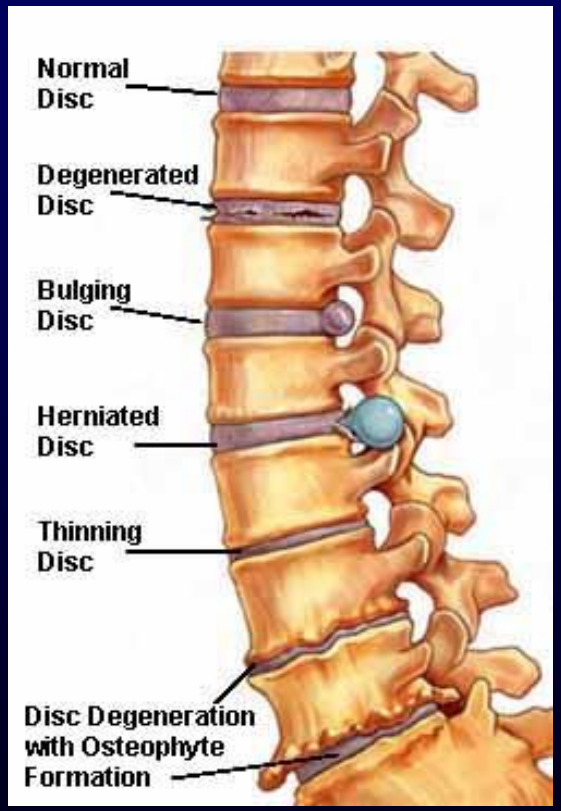
- Spondylosis
  - Pain
  - Stiffness
  - Limited ROM
  - Deformity
  - Radiculopathy
  - Myelopathy

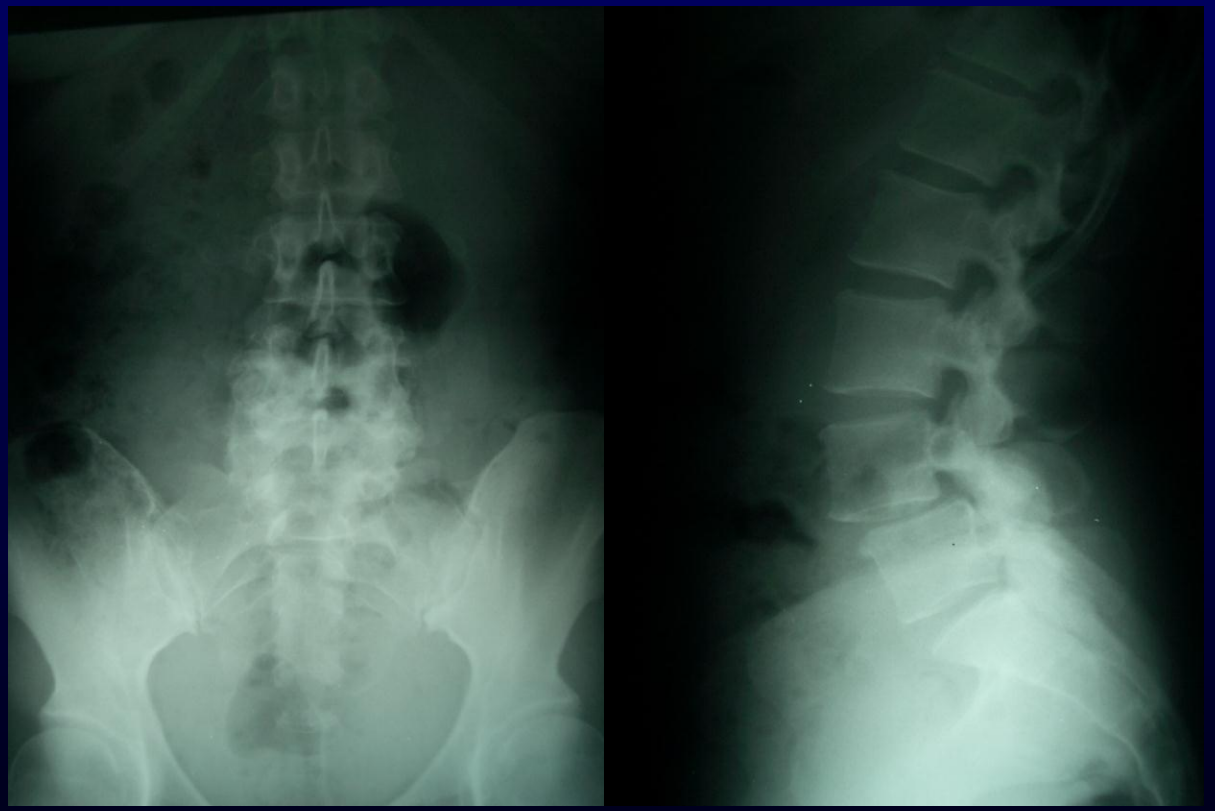






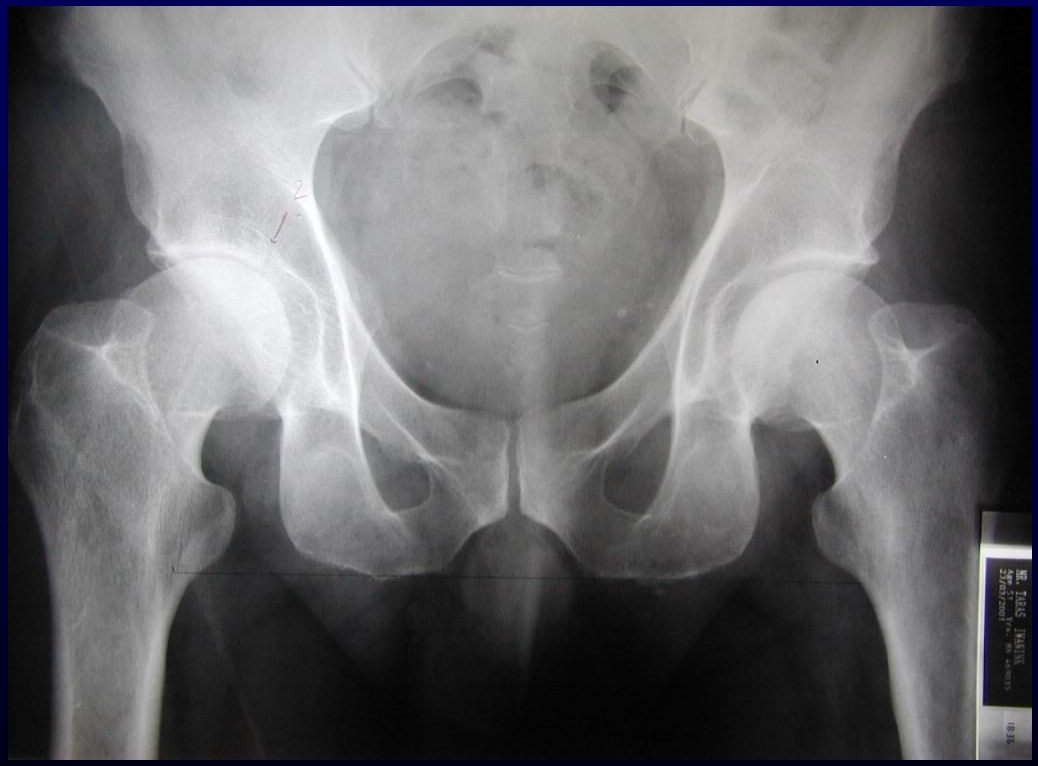
Spine


Normal Disc
Degenerated Disc
Bulging Disc
Herniated Disc
Thinning Disc
Disc Degeneration with Osteophyte Formation

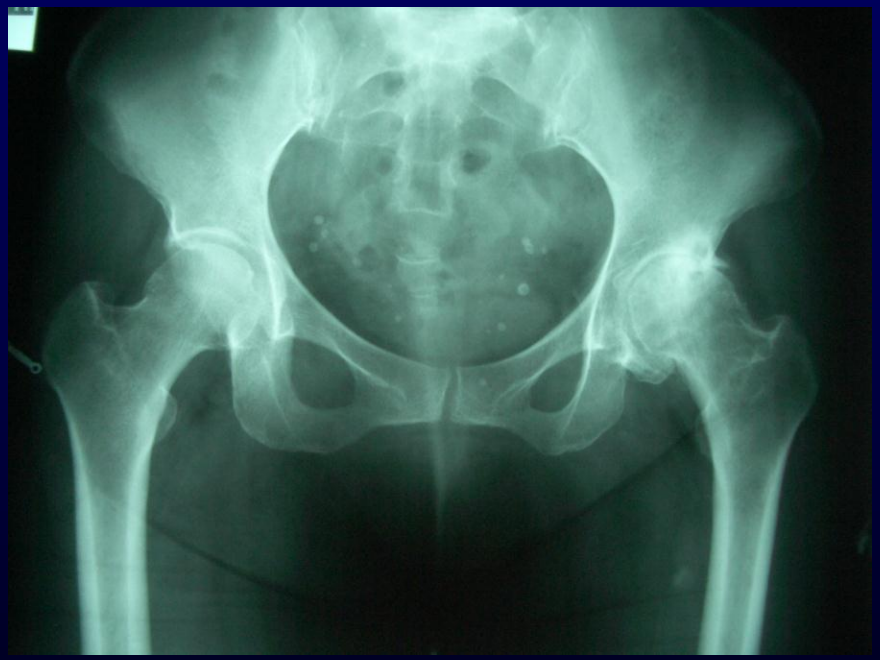


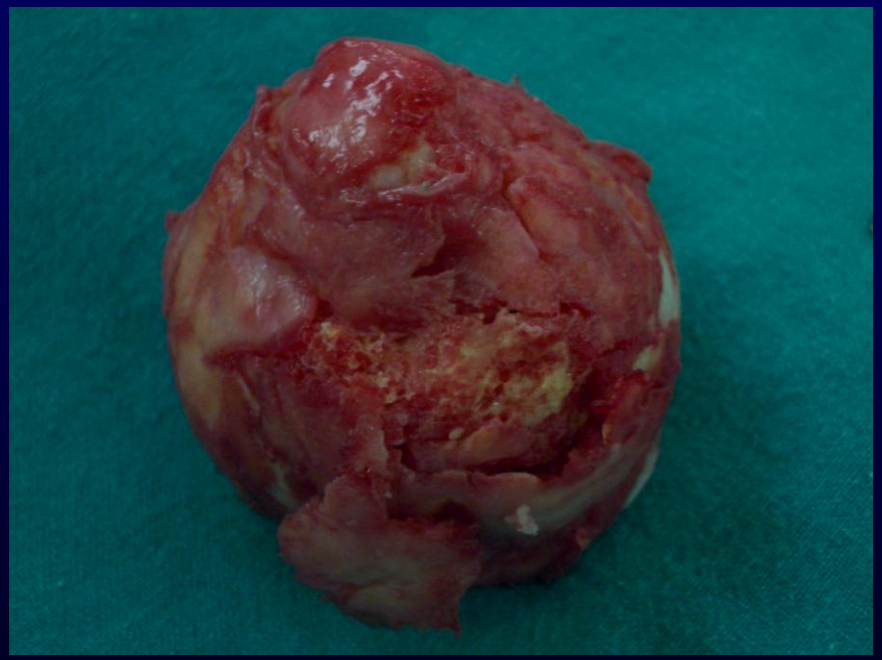
Primary OA: hip

- Pain
- Stiffness
- Limited ROM
- Limping



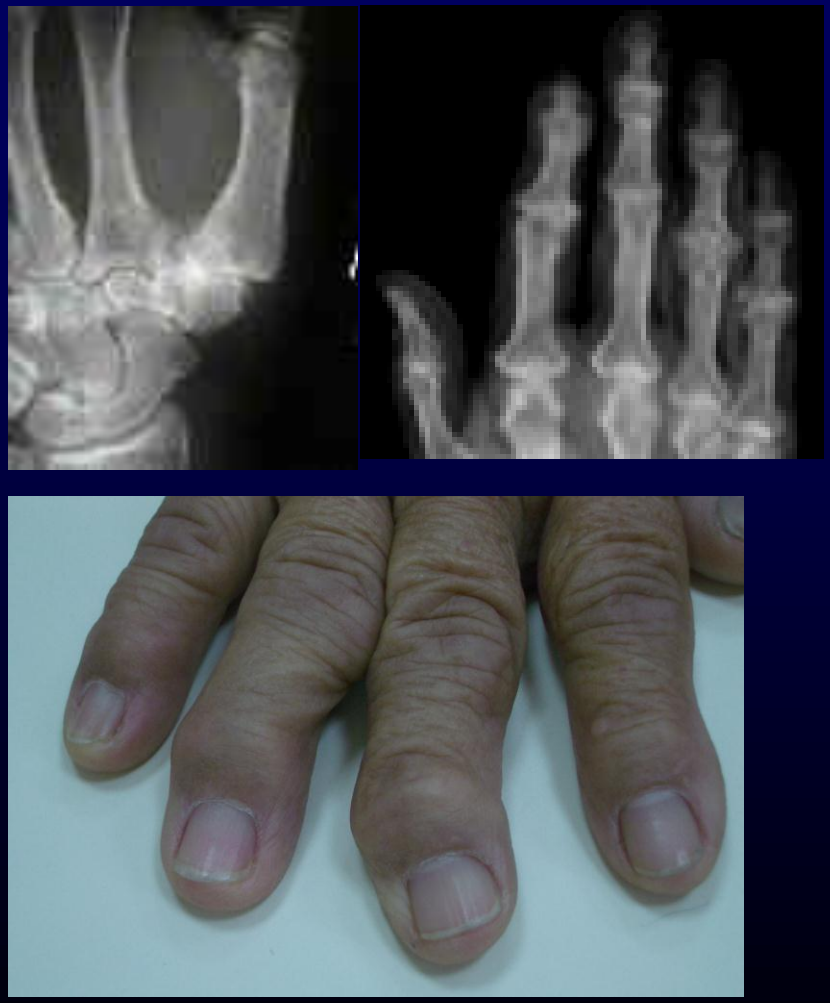
Primary OA: hip



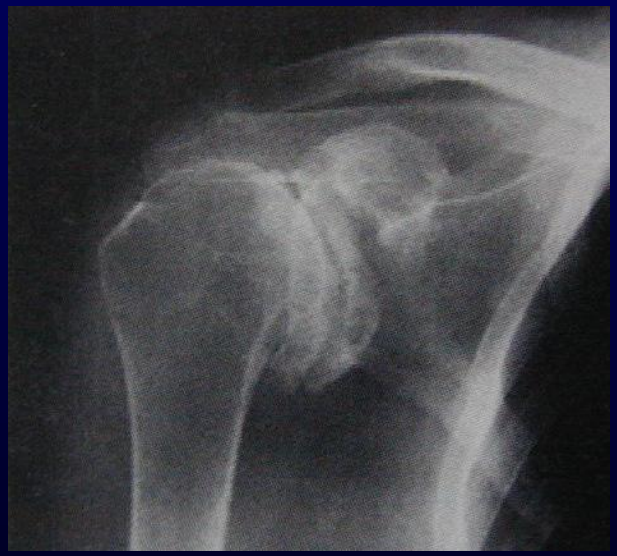


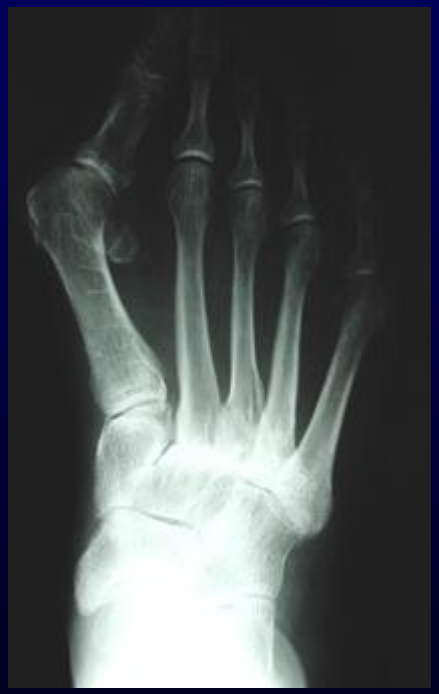
Primary OA: hand & finger

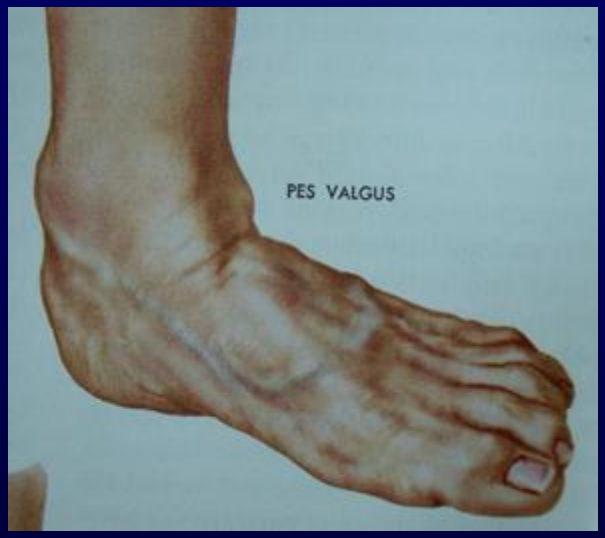
- Pain
- Swelling
- Limited ROM
- Heberden's nodes
- Bouchard's nodes

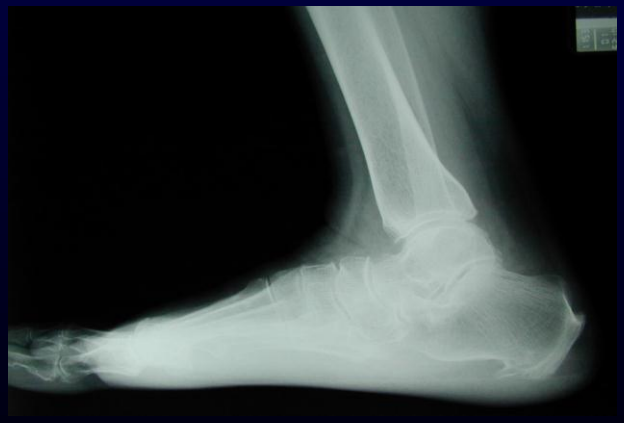


Primary OA: other joints









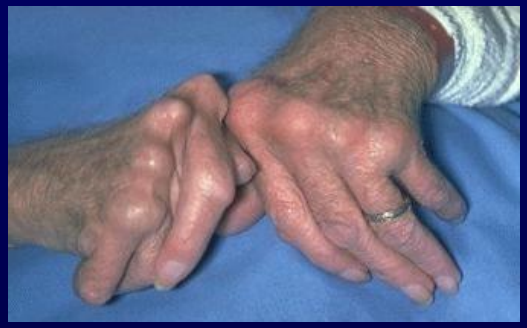
Secondary OA

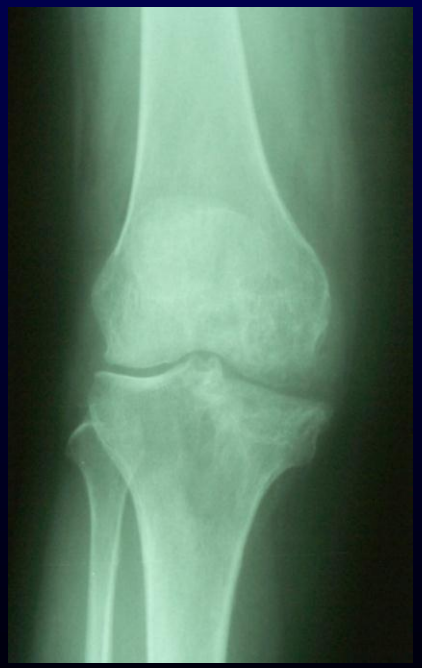
- Direct response to abnormal mechanical loading with articular cartilage
- Various causes

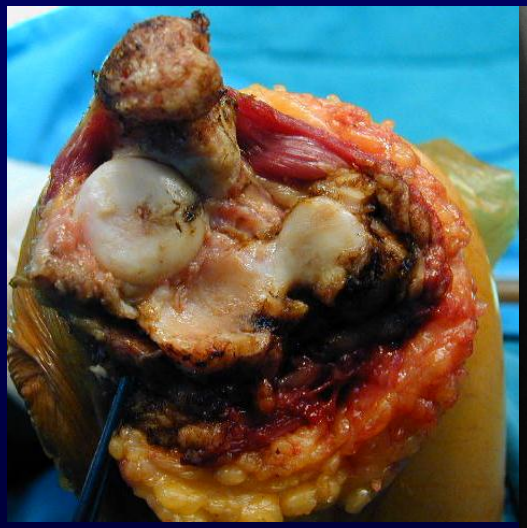
Table 1
Causes of Joint Degeneration (Secondary Osteoarthritis)

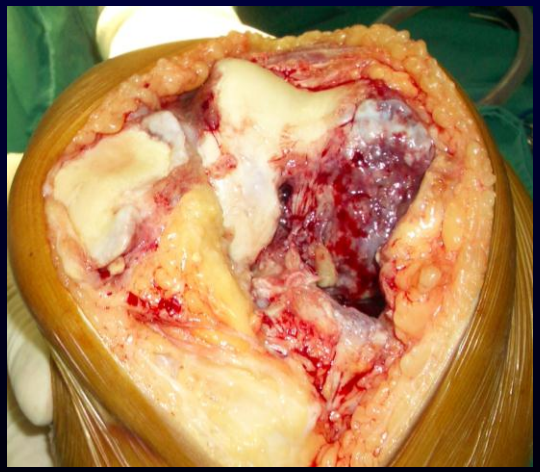
Cause	Presumed Mechanism
Intra-articular fractures	Damage to articular cartilage and/or joint incongruity
High-intensity impact joint loading	Damage to articular cartilage and/or subchondral bone
Ligament injuries	Joint instability
Joint dysplasias (developmental and hereditary joint and cartilage dysplasias)	Abnormal joint shape and/or abnormal articular cartilage
Aseptic necrosis	Bone necrosis leads to collapse of the articular surface and joint incongruity
Joint infection (inflammation)	Destruction of articular cartilage
Hemophilia	Multiple joint hemorrhages
Calcium pyrophosphate deposition disease	Accumulation of calcium pyrophosphate crystals in articular cartilage
Neuropathic arthropathy (Charcot joints, syphilis, diabetes mellitus, syringomyelia, meningomyelocele, leprosy, congenital insensitivity to pain, amyloidosis)	Loss of proprioception and joint sensation results in increased impact loading and torsion, joint instability and intra-articular fractures

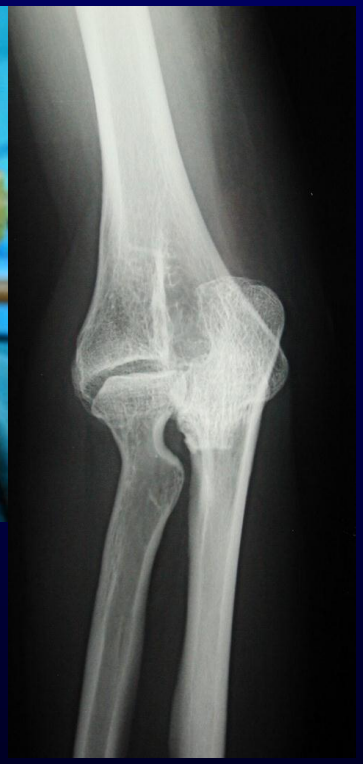
Secondary OA



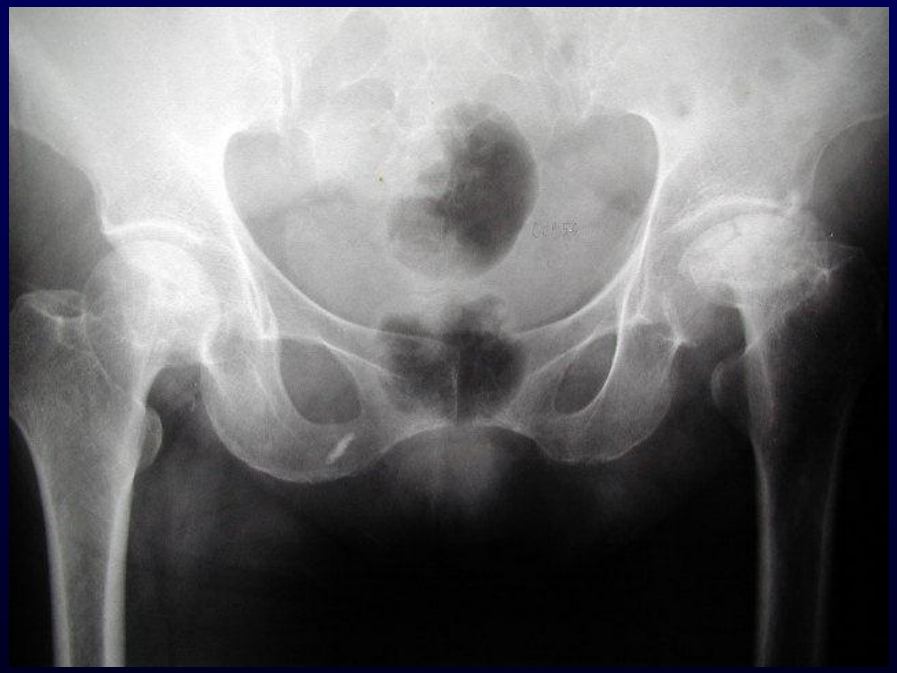




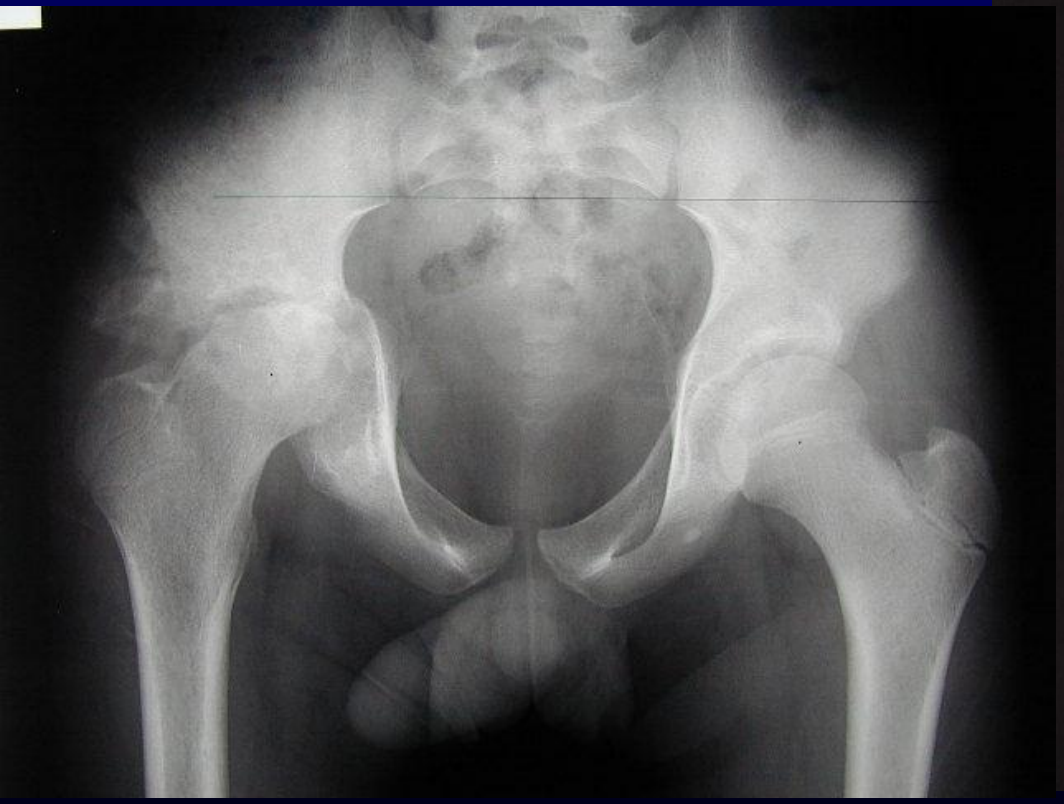


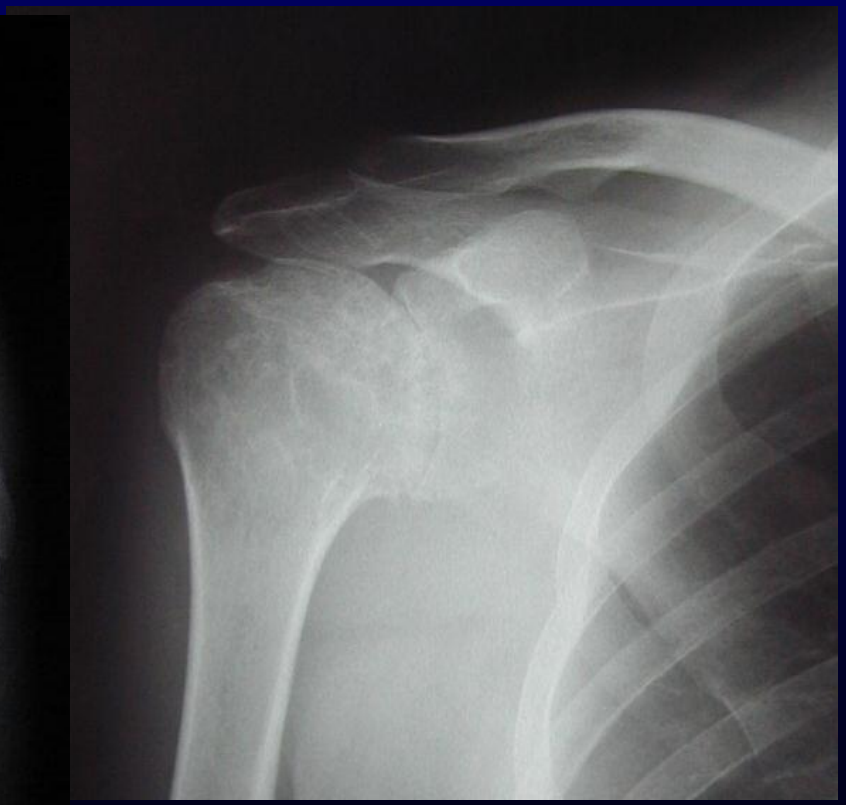


Secondary OA



Secondary OA





How is DJD treated?

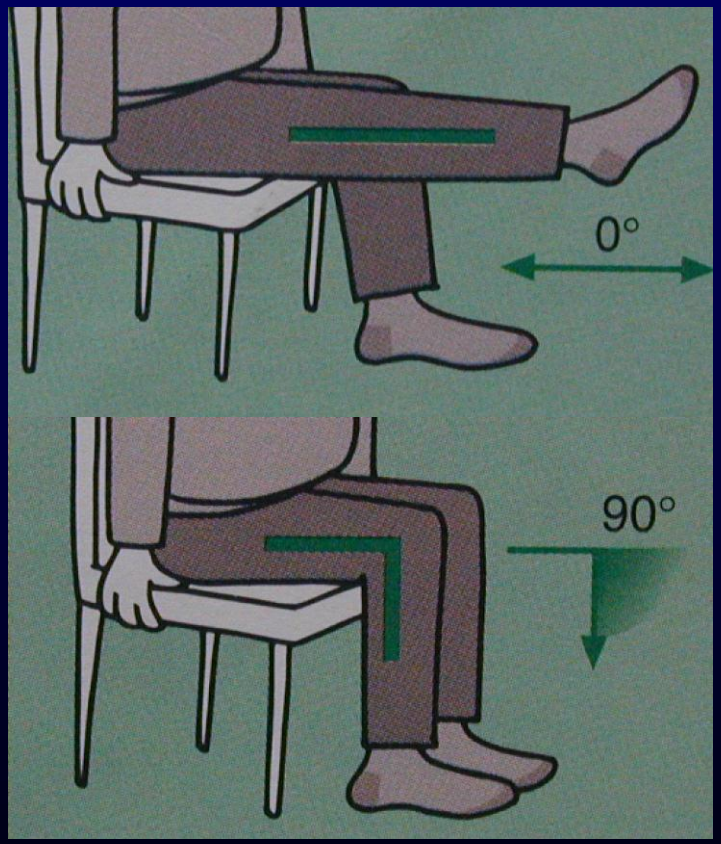
- Goal of treatment
  - Decrease pain
  - Muscle strengthening
  - Improve or maintain joint function
    - Activity of daily living
    - Sports activity

Conservative treatment of OA

- Activity modification

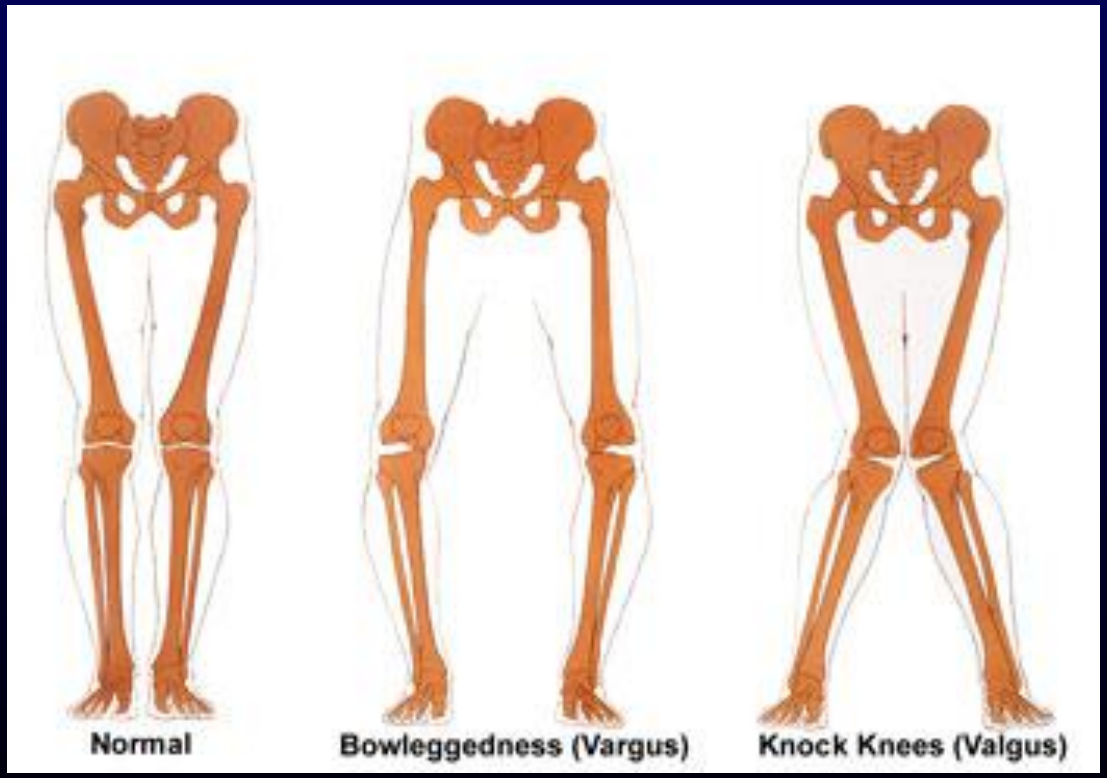
Conservative treatment of OA

- Activity modification
- Analgesics
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Appropriate physical therapy
- Weight loss
- Low impact exercise
- Intra-articular administration of hyaluronic acid



Surgical treatment of OA

- Realignment osteotomy
  - Normalizes the biomechanical loads on the involved joint



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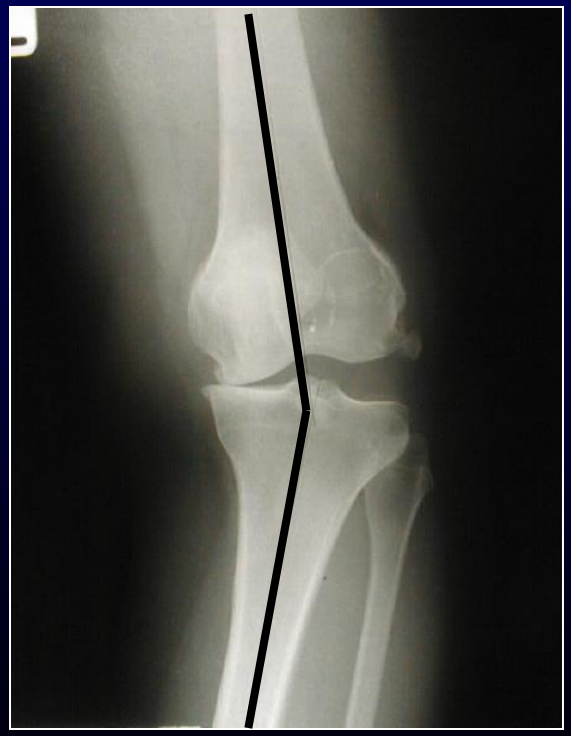
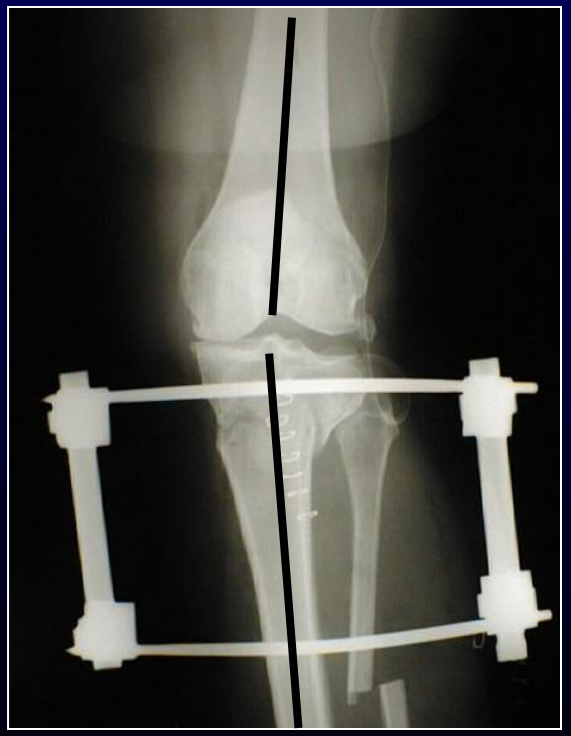
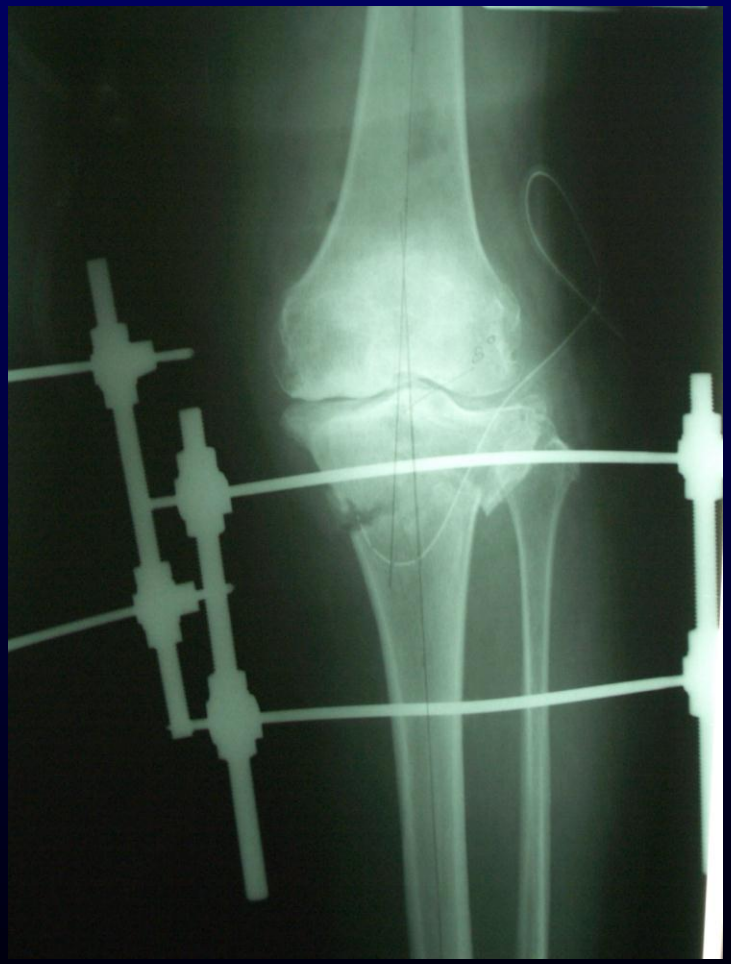




Surgical treatment of OA



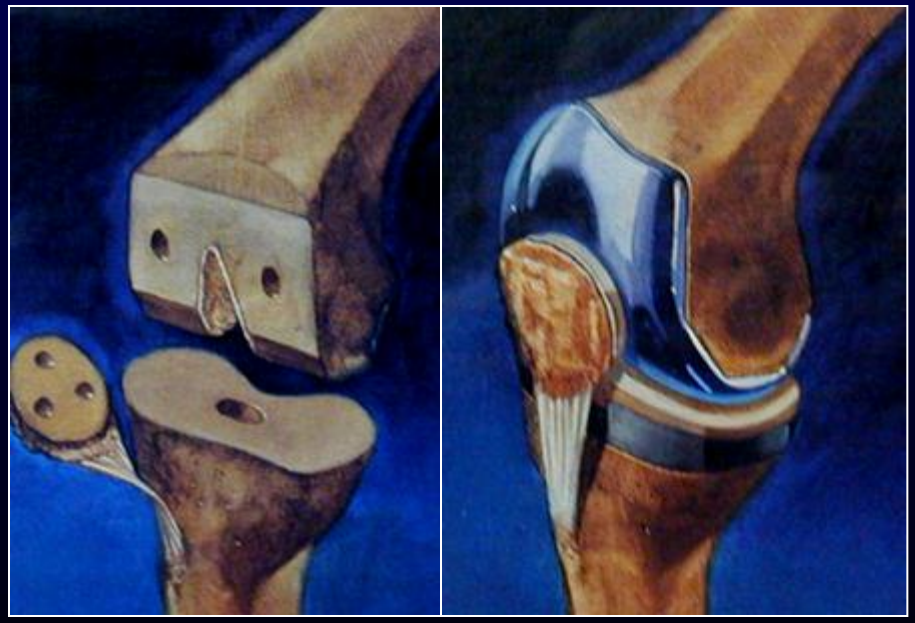
Surgical treatment of OA

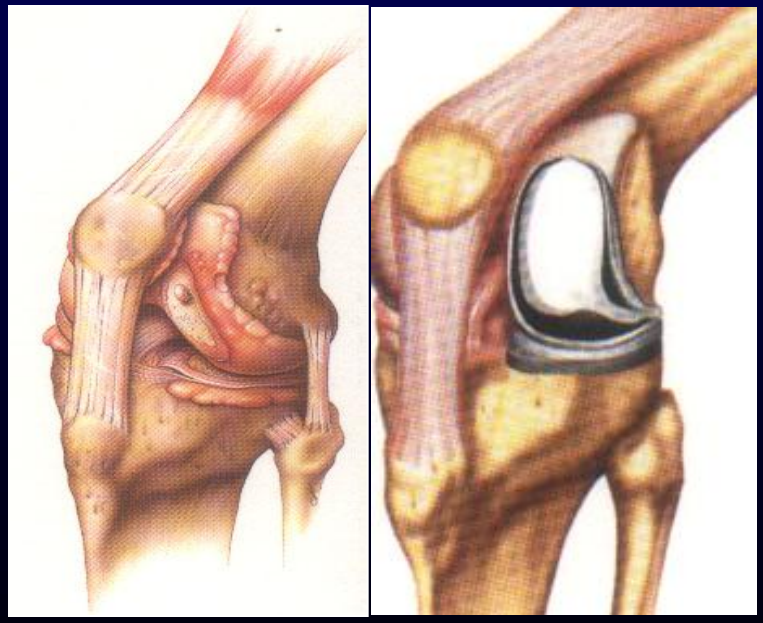
Surgical treatment of OA

- Autogenous chondrocyte transplantation
  - Repair isolated, limited size (2 to 7 cm2) chondral defects
- Osteochondral autografts "mosaicplasty”
  - Autografts of up to 10 mm is transferred into prepared defects

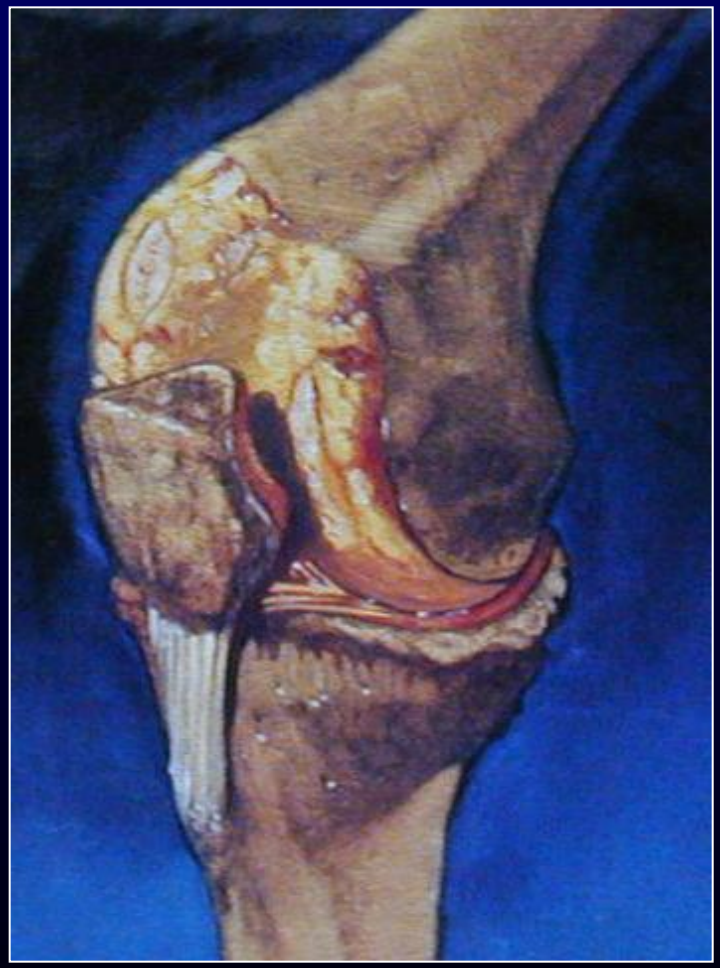
Surgical treatment of OA

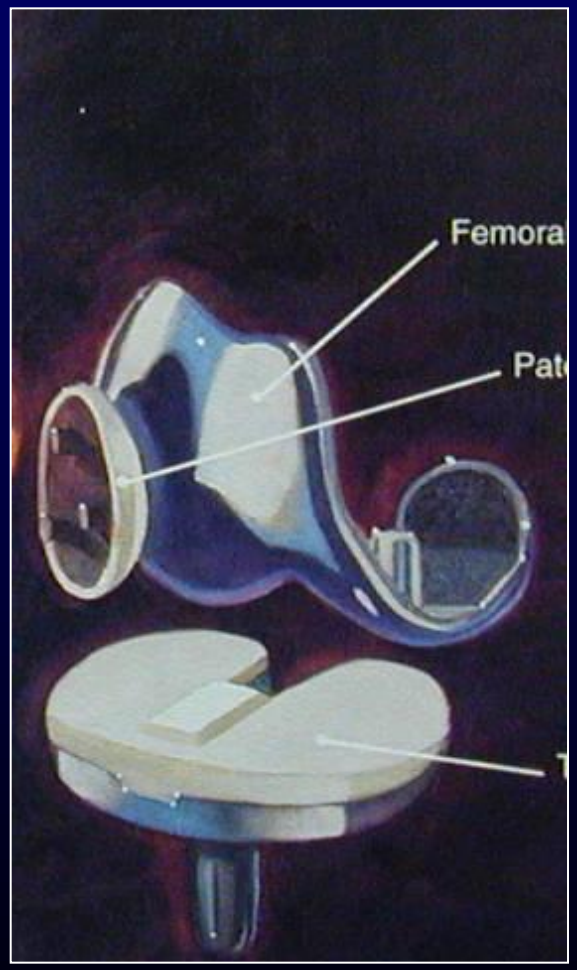
- Knee arthroplasty
  - Unicompartmental knee arthroplasty
  - Total knee arthroplasty





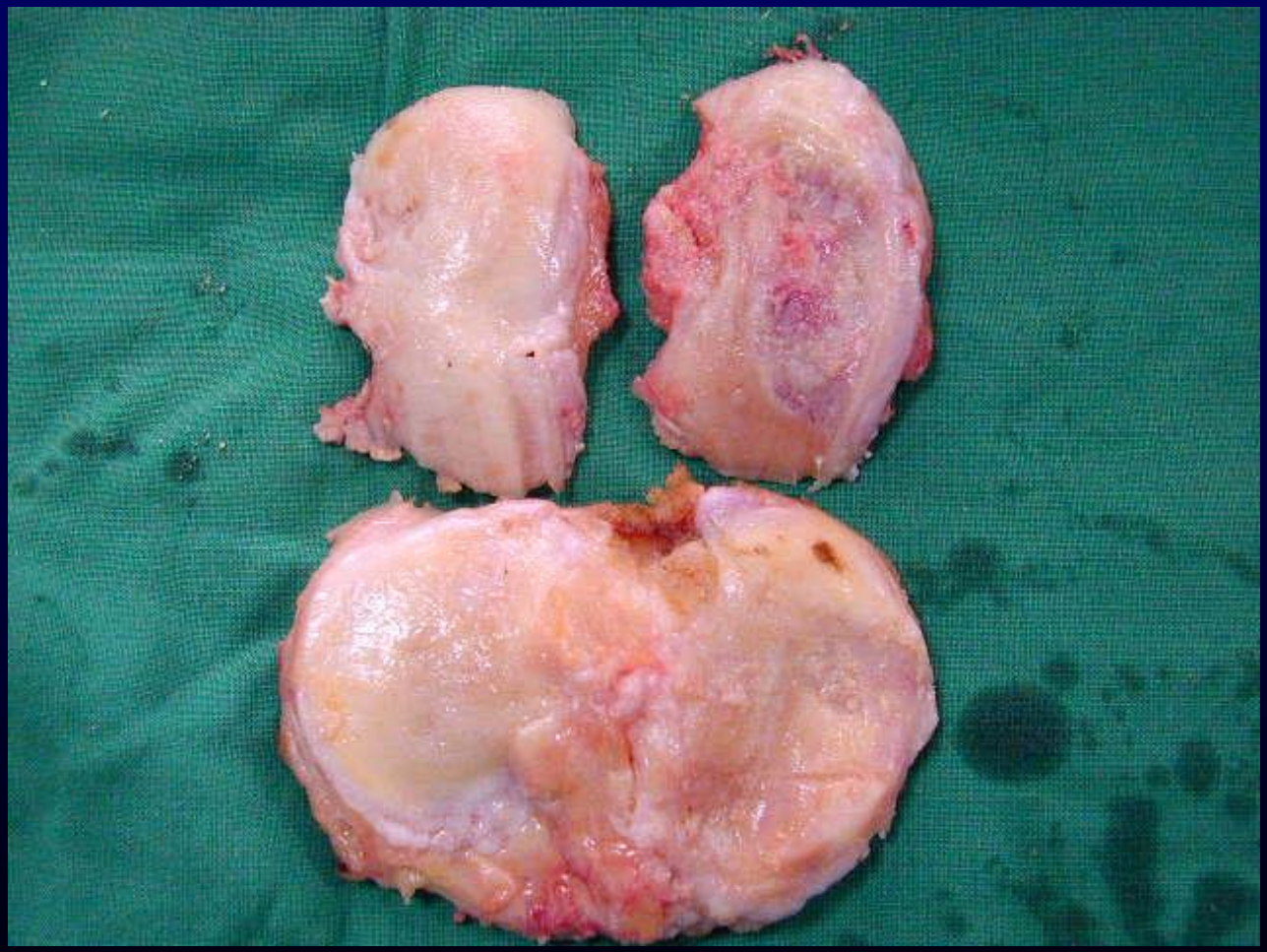
Surgical treatment of OA



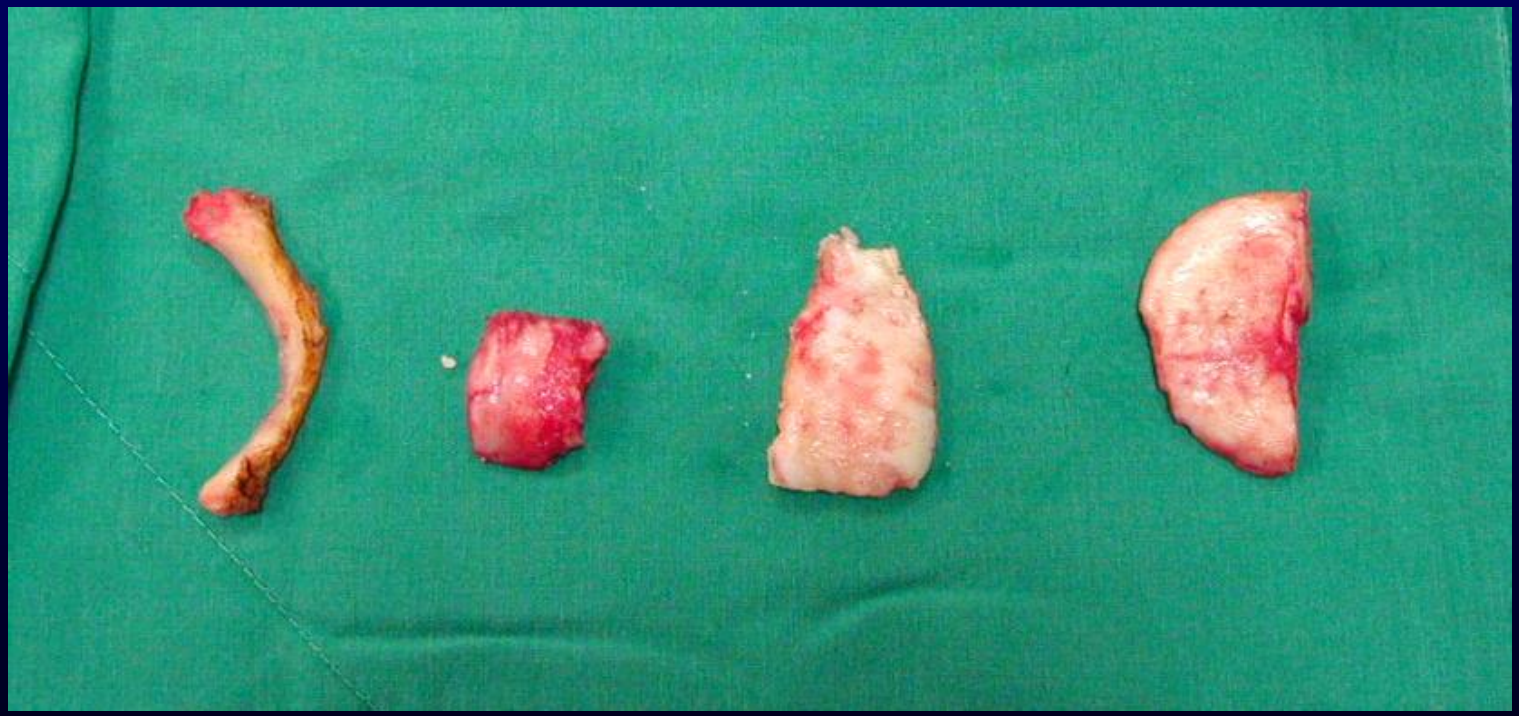


Surgical treatment of OA

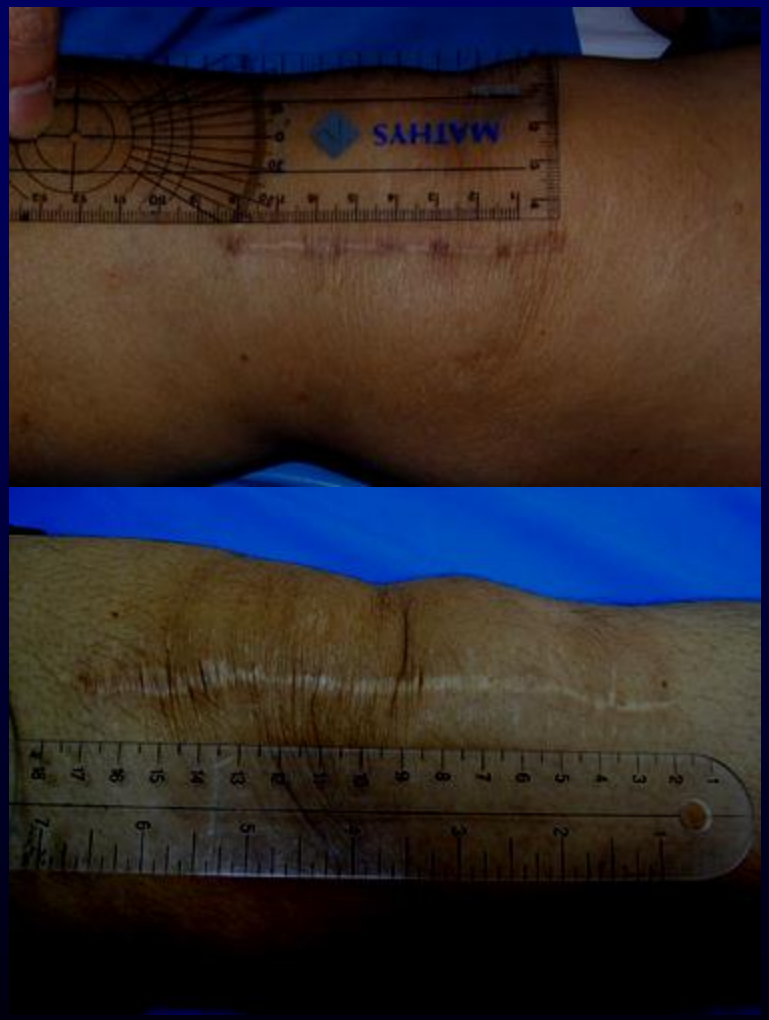
Surgical treatment of OA

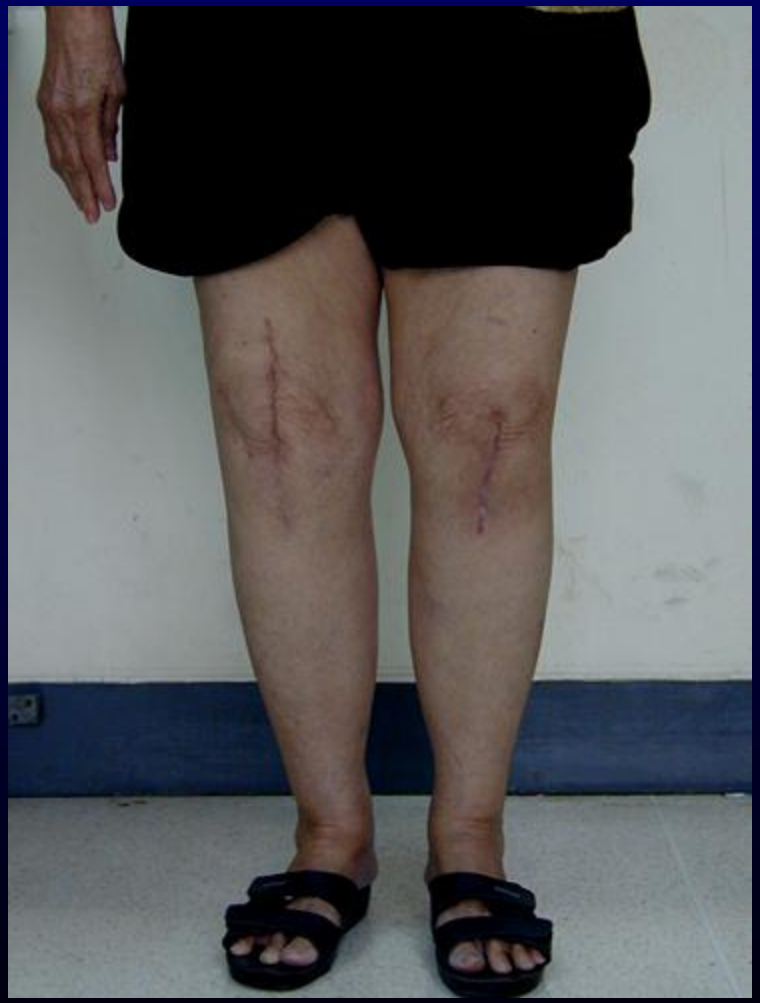


Surgical treatment of OA

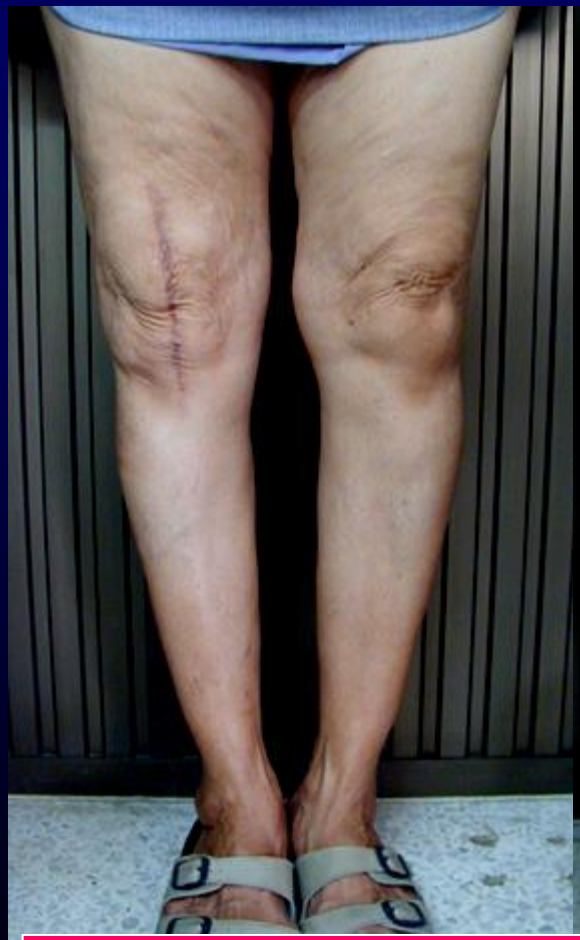


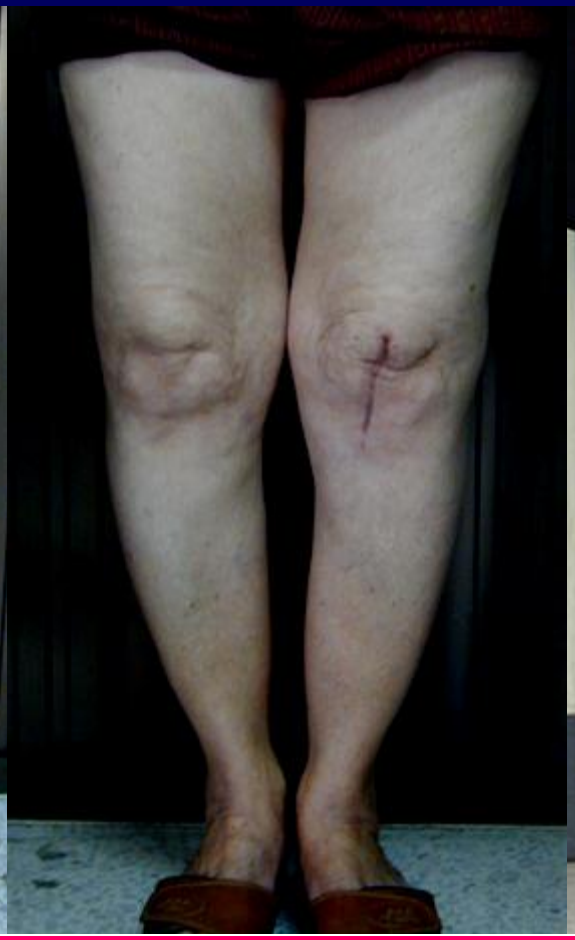
Surgical treatment of OA





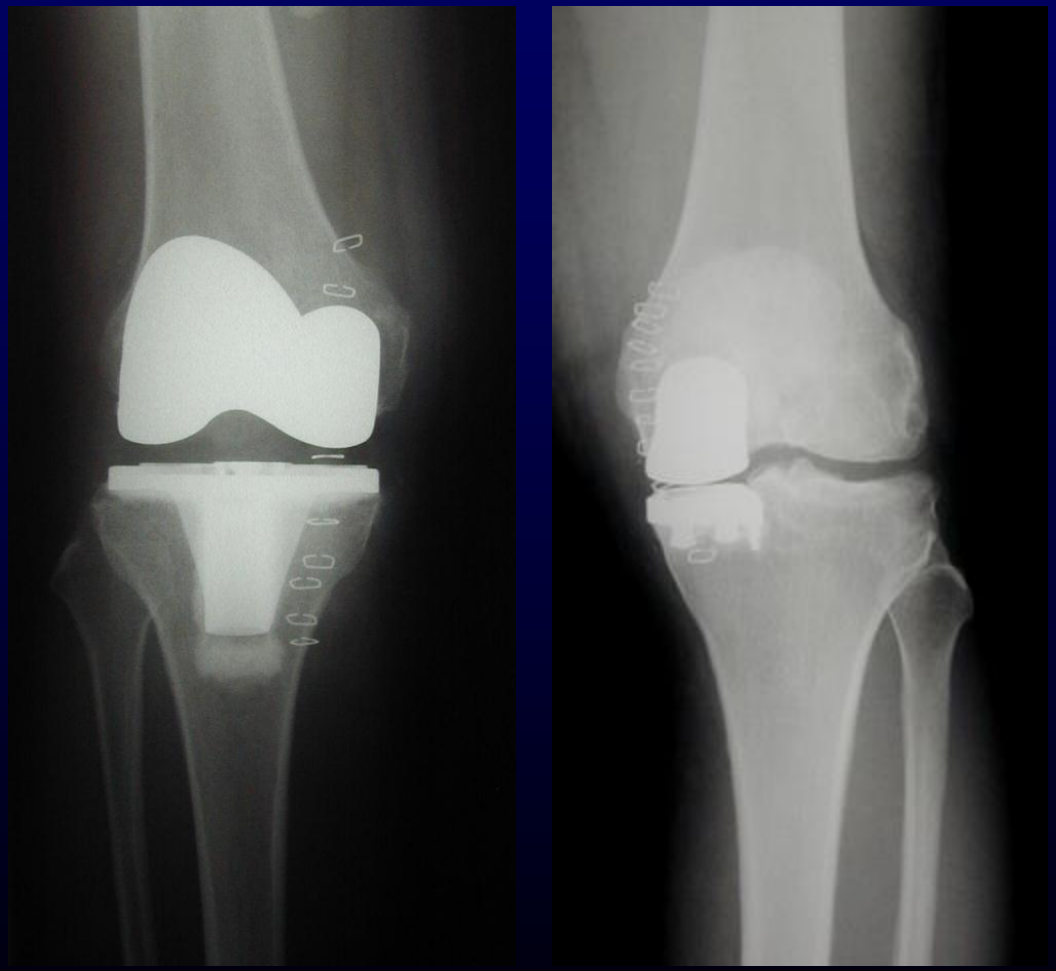
Surgical treatment of OA



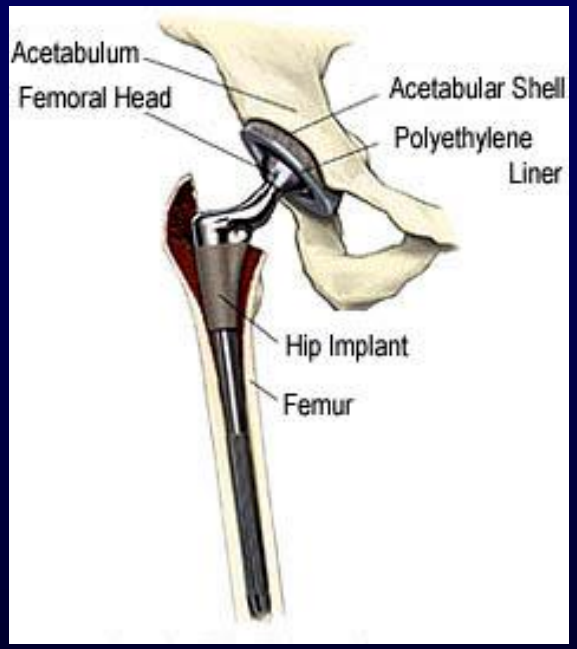


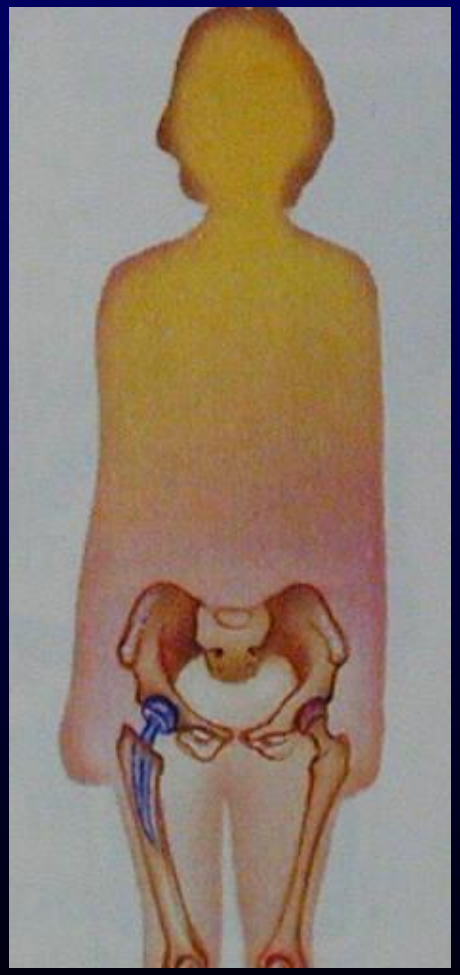
STD MIS: TKA MIS: UKA

Surgical treatment of OA

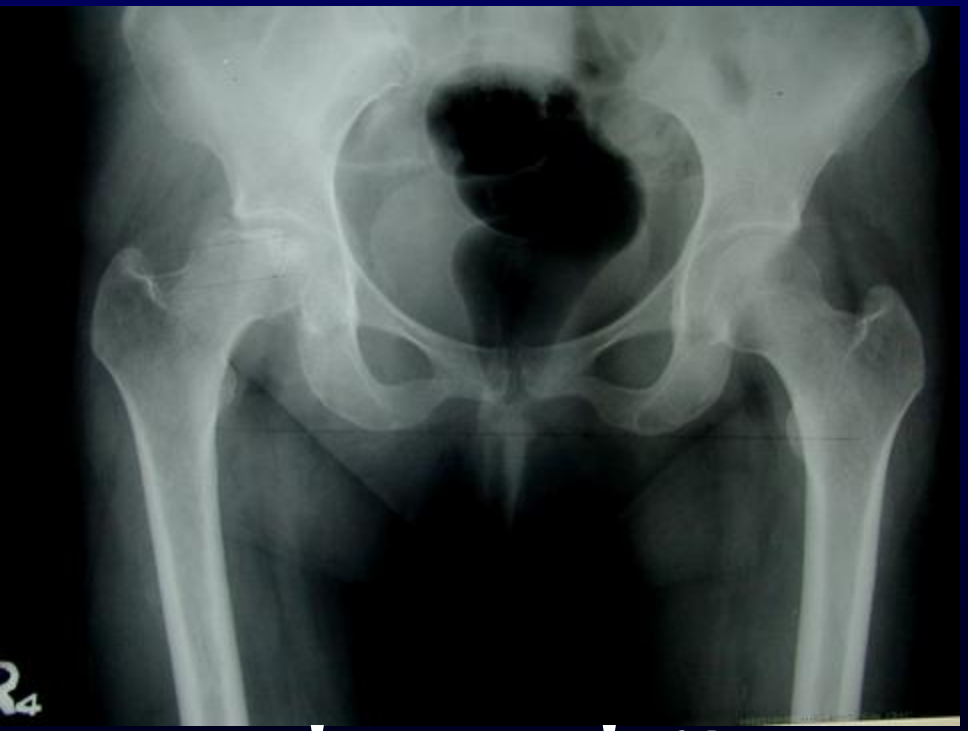


Surgical treatment of OA

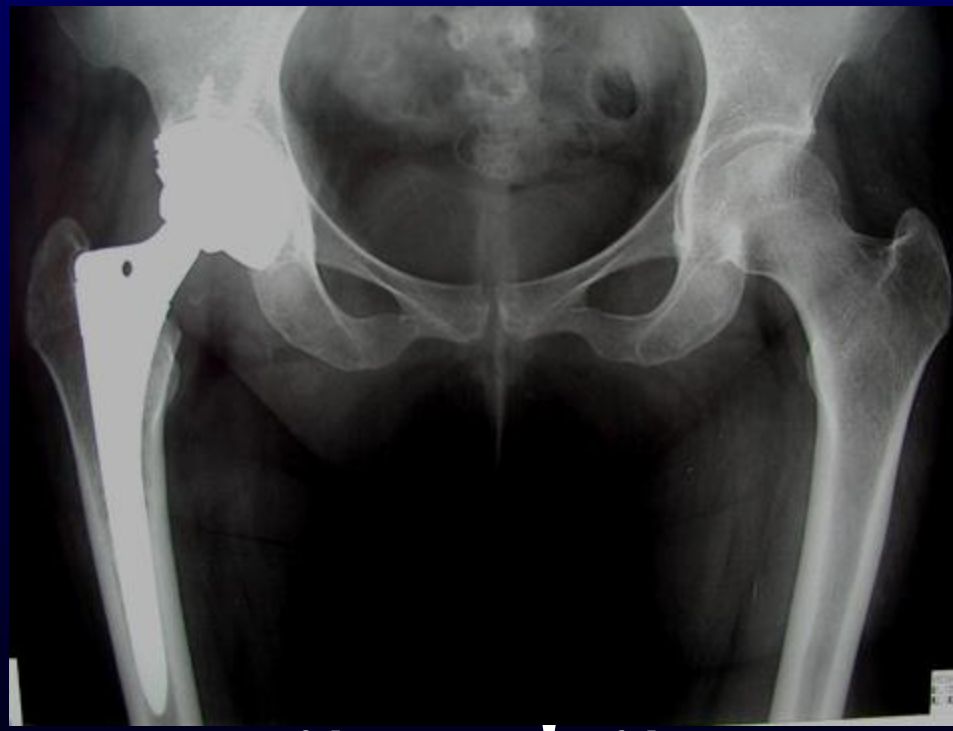




Surgical treatment of OA

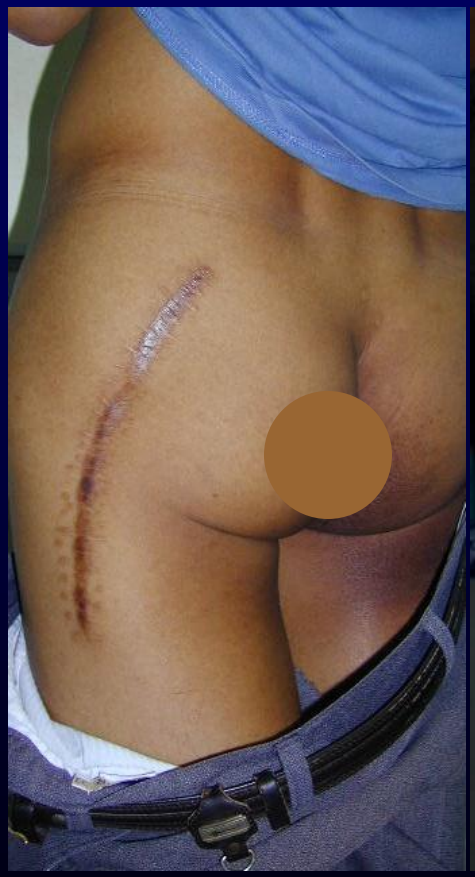


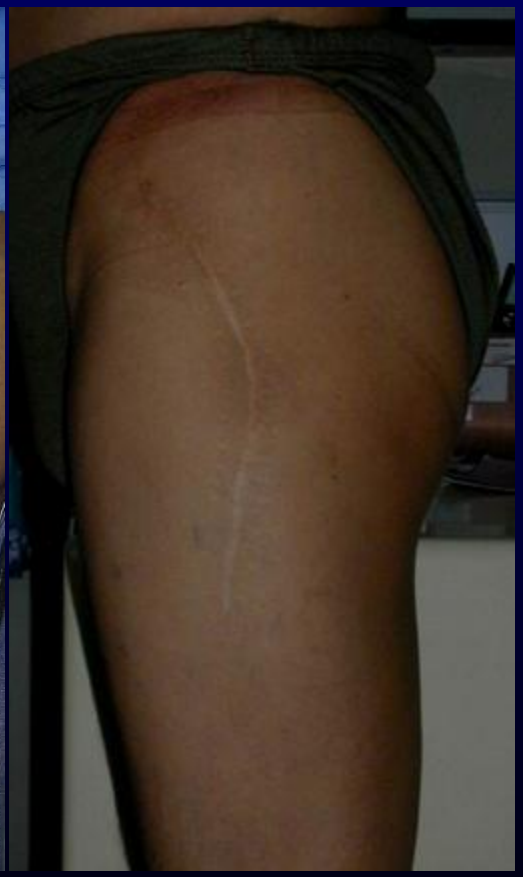
ก่อนการผ่าตัด

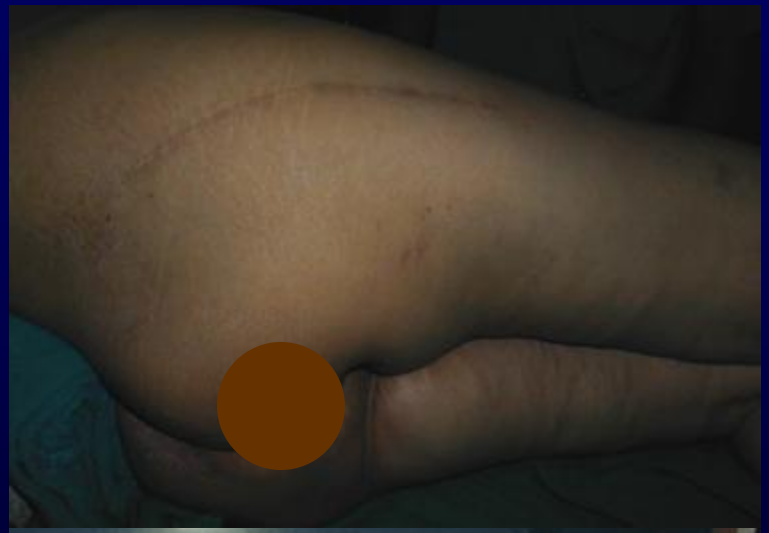


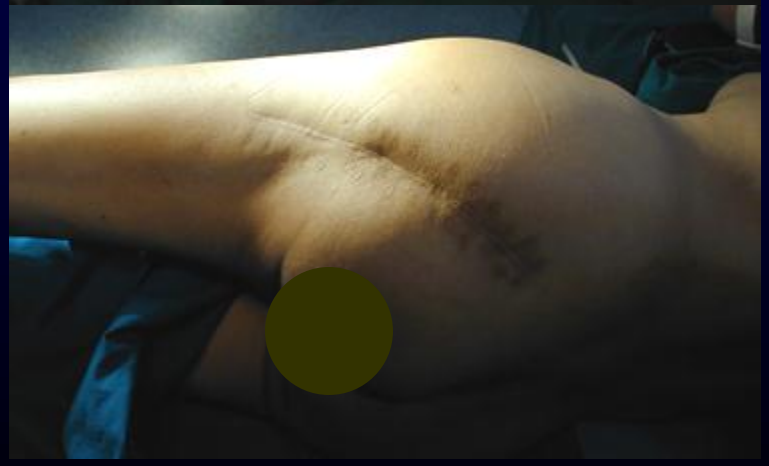
หลังการผ่าตัด

Surgical treatment of OA

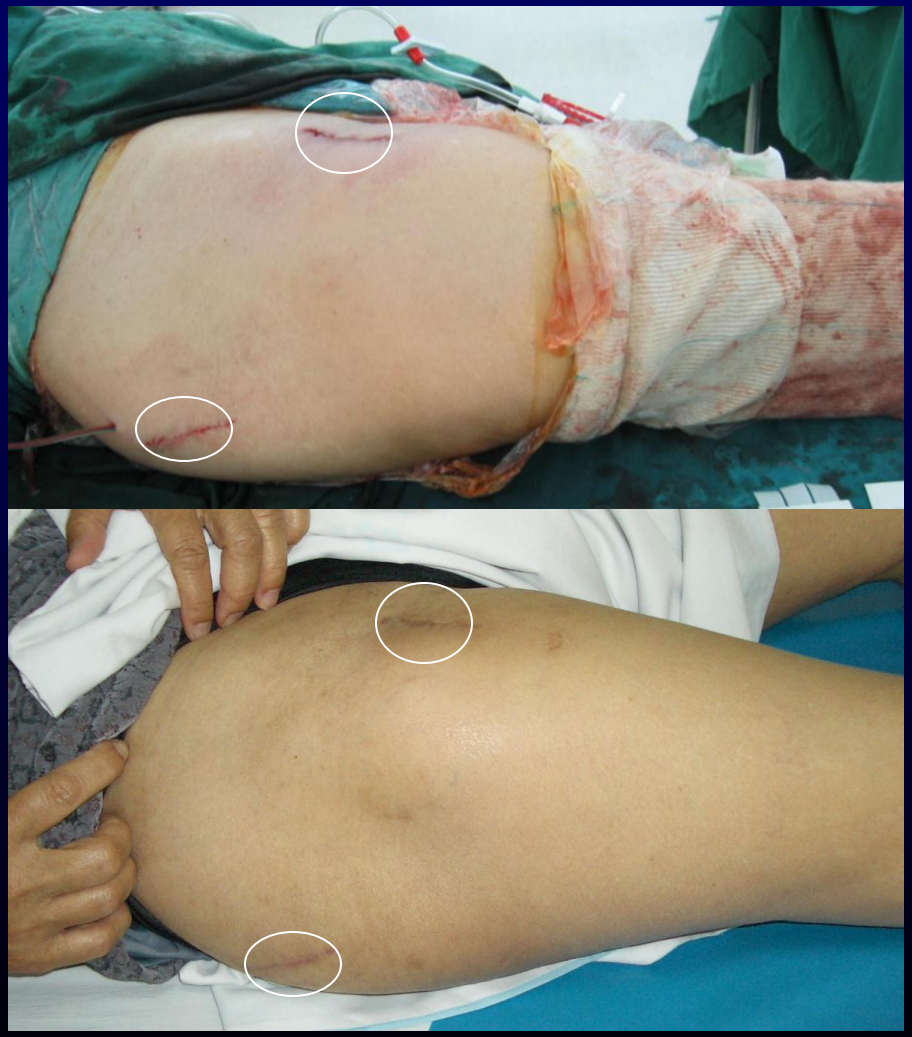








Surgical treatment of OA



Thank you