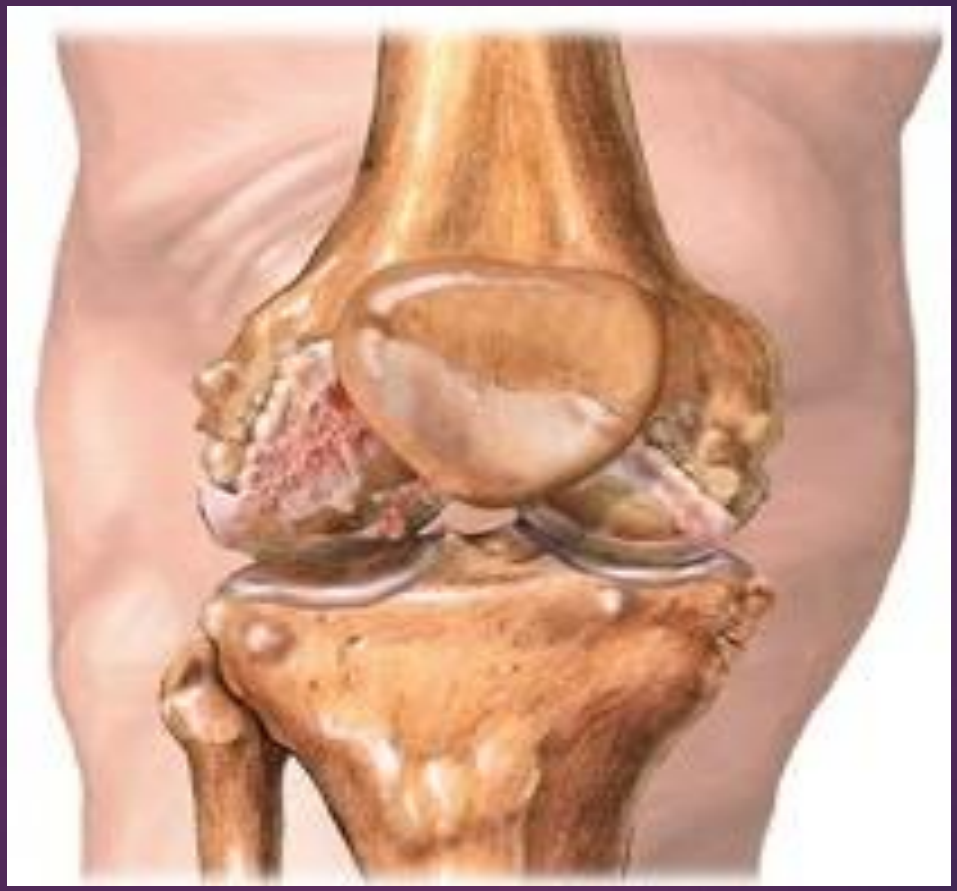
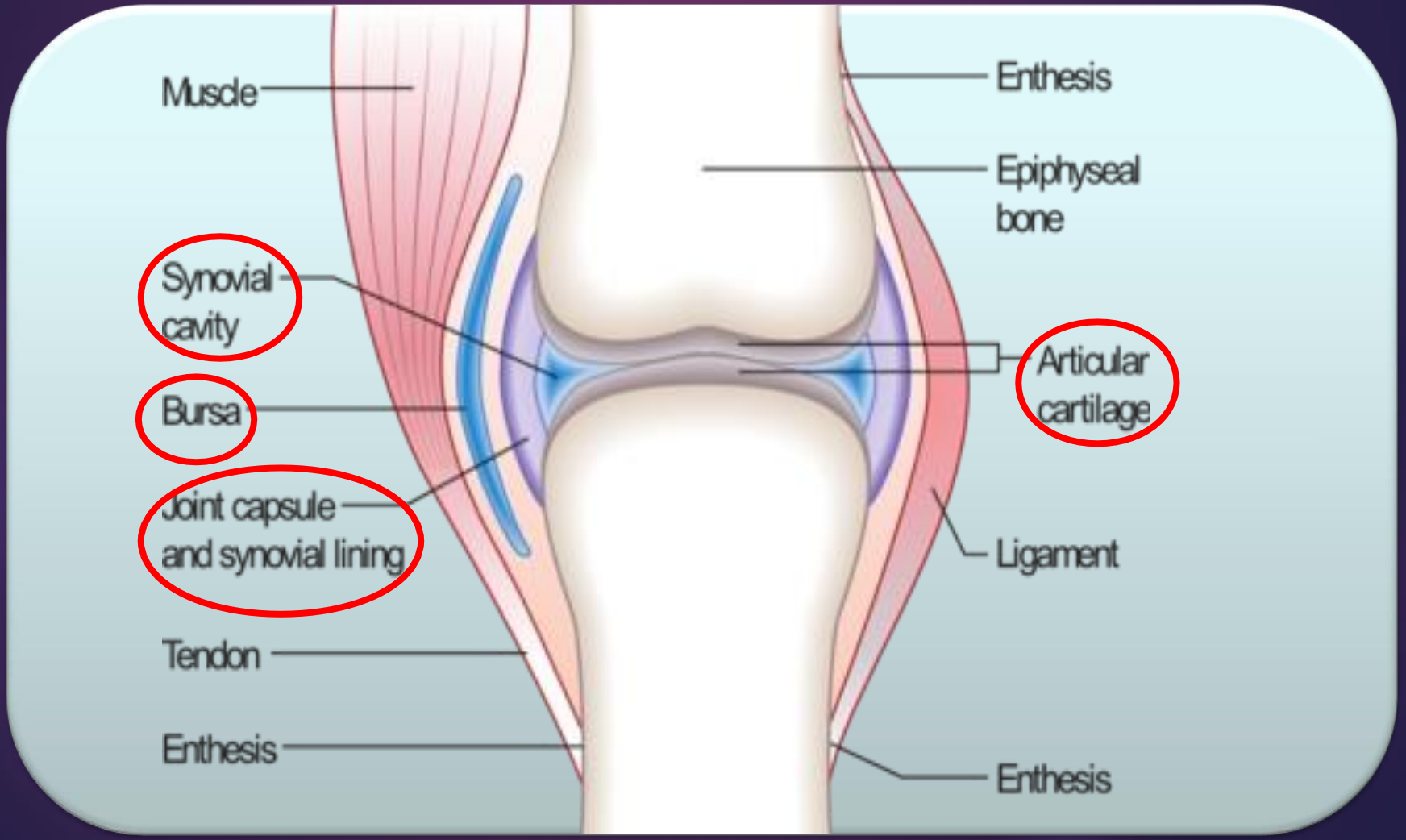


OSTEOARTHRITIS

PROTECTIVE MECHANISM OF SYNOVIAL JOINTS

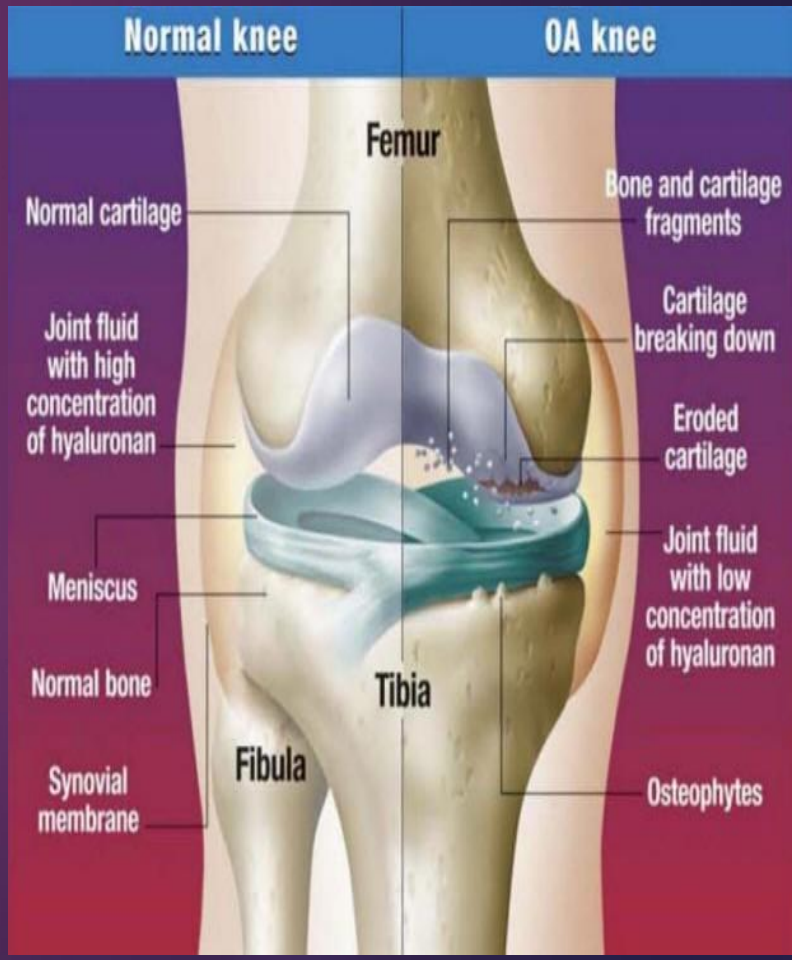


LEARNING OBJECTIVES

- Describe etiology and pathogenesis of osteoarthritis
- Discuss clinical and morphological features of osteoarthritis
- Enumerate complications of osteoarthritis

DEFINITION

Osteoarthritis is a degenerative disease of synovial joints characterized by focal loss of articular hyaline cartilage with proliferation of new bone & remodeling of joint contour.



EPIDEMIOLOGY

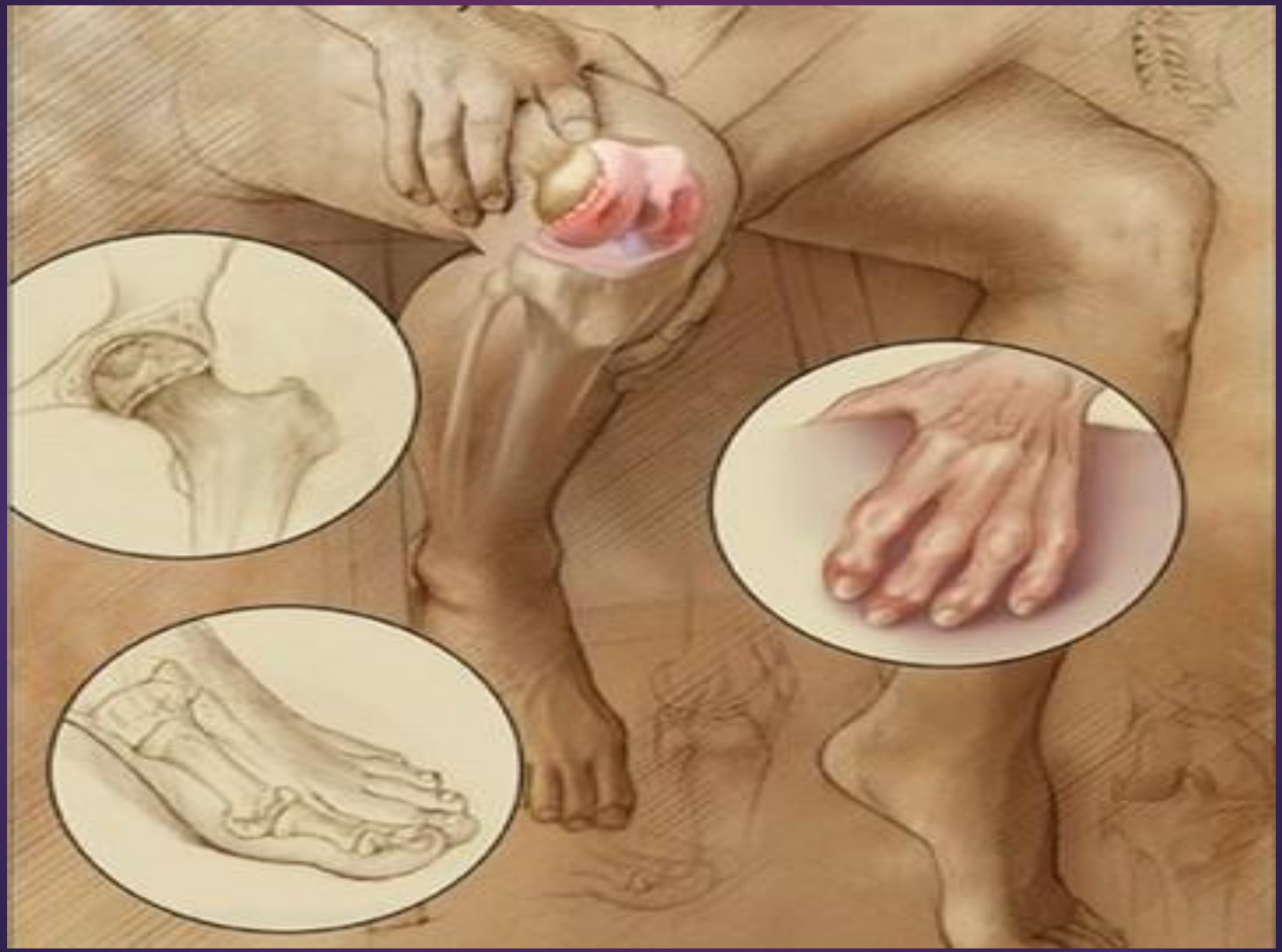
- Weight bearing joints e.g. knee & hip joints.
- Arround3.3% population in the world.

- Age > 65 years.
  - 80% have radiographic features.
  - 25-30% have symptoms.

- More common in women.

- Familial tendency.

DISTRIBUTION OF DISEASE



ETIOLOGY

- **PRIMARY / IDIOPATHIC:**

  When there is no obvious predisposing factor. Common form of OA.

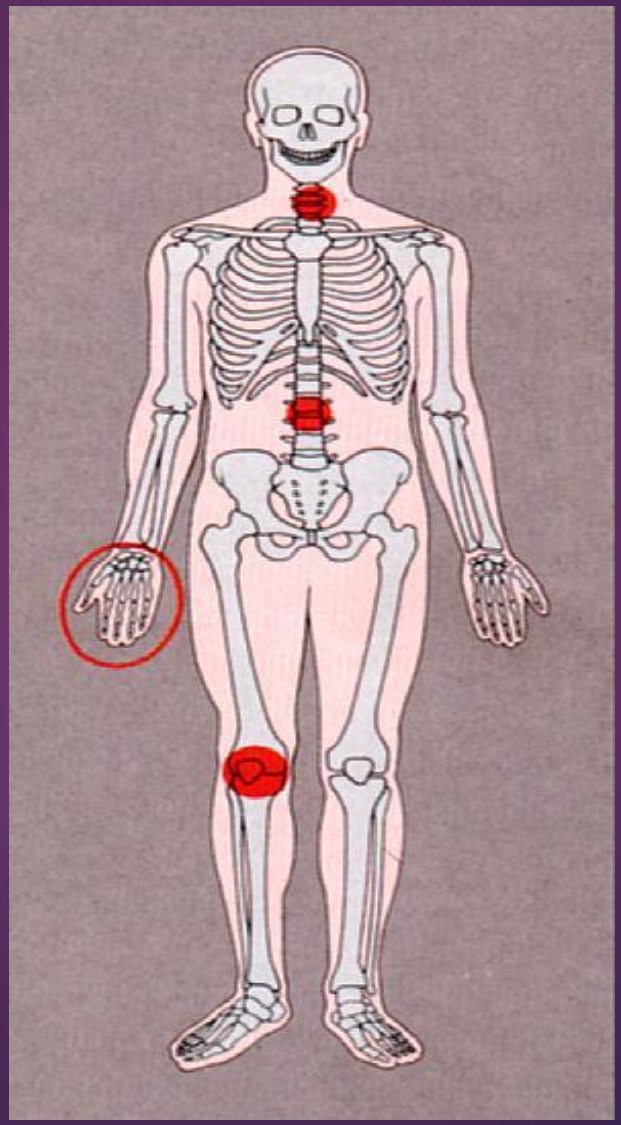
- **SECONDARY:**

  When degenerative joint changes occur in response to a recognizable local or systemic factor.

RISK FACTORS FOR PRIMARY OA

- Age
- Sex
- Obesity
- Genetics
- Trauma (daily)

MORPHOLOGY OF PRİMARY OA



CAUSES OF SECONDARY OSTEOARTHRITIS

DEVELOPMENTAL

**HIP DYSPLASIA*

ENDOCRINE

**ACROMEGALY*

TRAUMATIC

**INTRA-ARTICULAR FRACTURE*

**OCCUPATIONAL*

**MENISECTOMY*

INFLAMMATION

**RA*

**GOUT*

**SEPTIC ARTHRITIS*

METABOLIC

**ALKAPTONURIA*

**WILSON'S DISEASE*

ASEPTIC NECROSIS

**CORTICOSTERIOD USE*

**SLE*

SICKLE CELL DISEASE

NEUROPATHIES

**SYRINGOMYELIA*

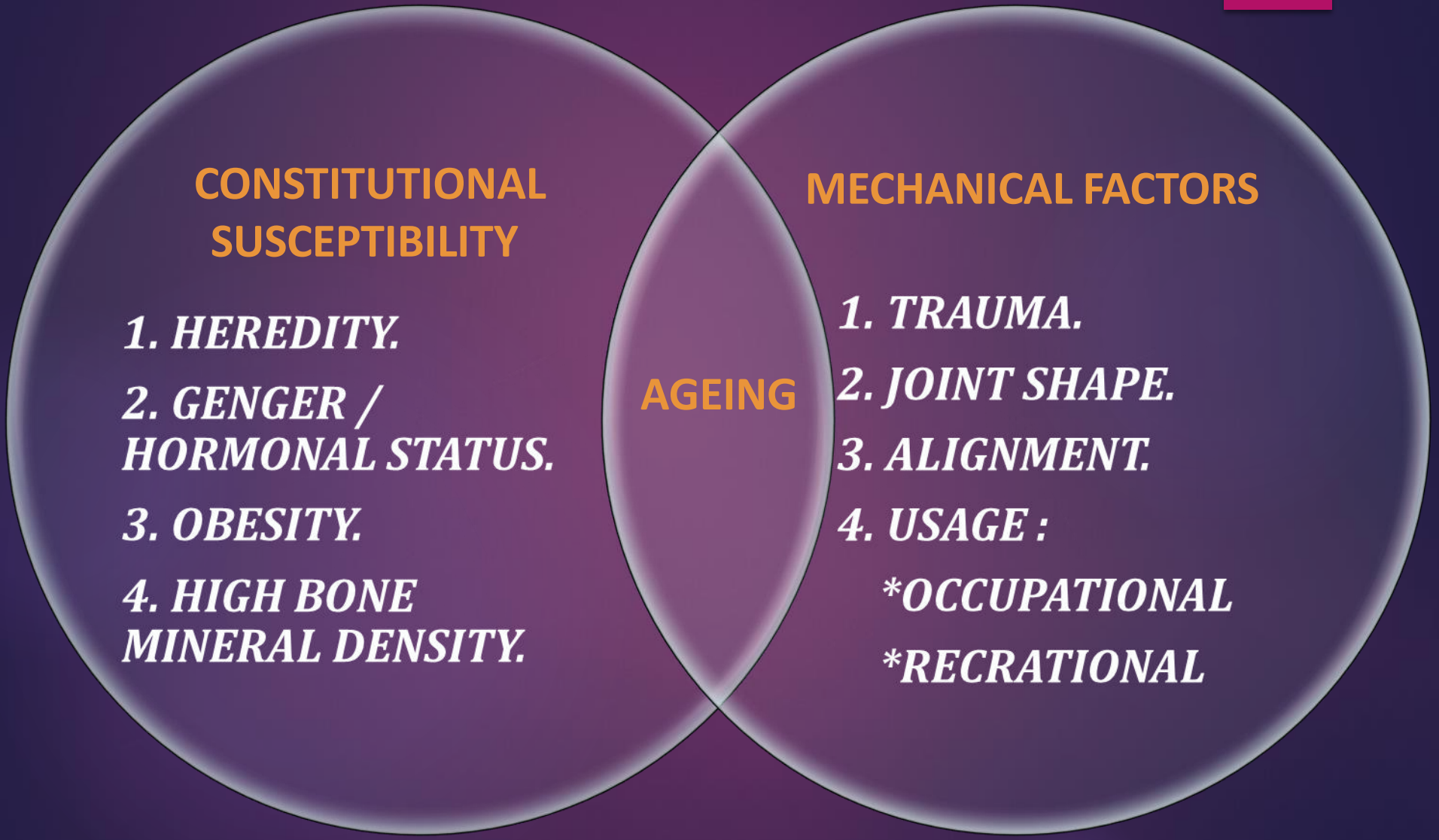
**DIABETES MELLITUS*

MISCELLANOUS

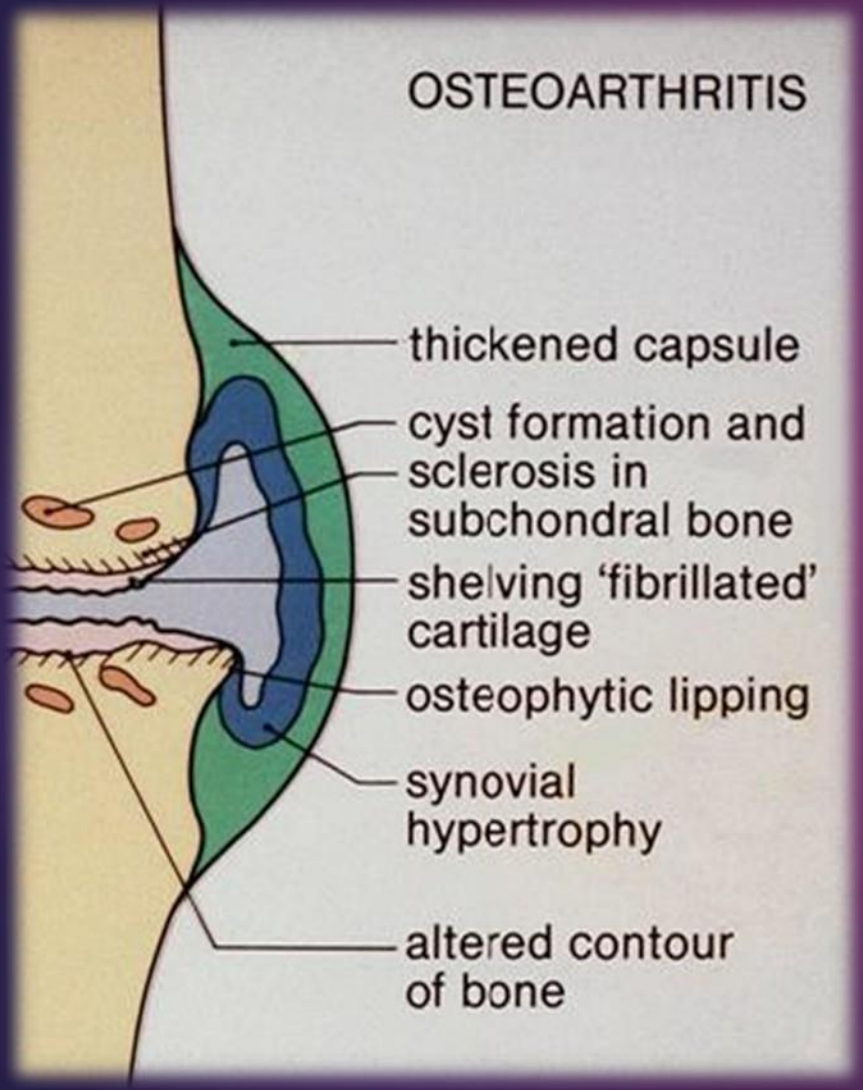
**PAGET'S DISEASE*

**GAUCHER'S DISEASE*

RISK FACTORS



PATHOGENESIS



- Progressive destruction & loss of articular cartilage with an accompanying peri-articular bone response leads to exposure of sub-chondral bone which becomes sclerotic, with increased blood vascularity & cyst formation.

Contd

- The lesions of osteoarthritis (OA) stem from degeneration of the articular cartilage and its disordered repair.
- The changes regarding chondrocytes can be divided into three phases:
- (1) chondrocyte injury, related to genetic and biochemical factors.
- (2) early OA, in which chondrocytes proliferate and secrete inflammatory mediators, collagens, proteoglycans, and proteases, which act together to remodel the cartilaginous matrix and initiate secondaryinflammatory changes in the synovium and subchondral bone.
- (3) late OA, in which repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive subchondral bone changes.

- Cytokines IL-1, IL-6, TNF-α
- ↓
- Cell destruction
- ↓
- Membrane phospholipids
- ↓
- Arachidonic acid
- ↓
- Cox-1, Cox-2

- IL-1 and metalloproteases have been found to play an important role in cartilage destruction.

- Local growth factors, especially transforming growth factor (TGF) are involved in the formation of osteophytes

MORPHOLOGY

- In the early stages of osteoarthritis, the chondrocytes proliferate,. Concurrently, the water content of the matrix increases and the concentration of proteoglycans decreases. The normally horizontally arranged collagen type II fibers in the superficial zone are cleaved, yielding fissures and clefts at the articular surface . This manifests as a granular soft articular surface. Eventually, chondrocytes die and full-thickness portions of the cartilage are sloughed. The dislodged pieces of cartilage and subchondral bone tumble into the joint, forming loose bodies called as joint mice.

Contd

- The exposed subchondrl bone plate becomes the new articula surface, and friction with the opposing surface smooth the exposed bone, giving it the appearance of polished ivory (bone eburnatio)
- Small fractures through the articulating bone are common, and the fracture gaps allow synovial fluid to be forced into the subchondral regions in a one-way, ball valve-like mechanism.
- Mushroom-shaped osteophytes (bony outgrowths) develop at the margins of the articular surface and are capped by fibrocartilage and hyaline cartilage that gradually ossify.

CLINICAL FEATURES

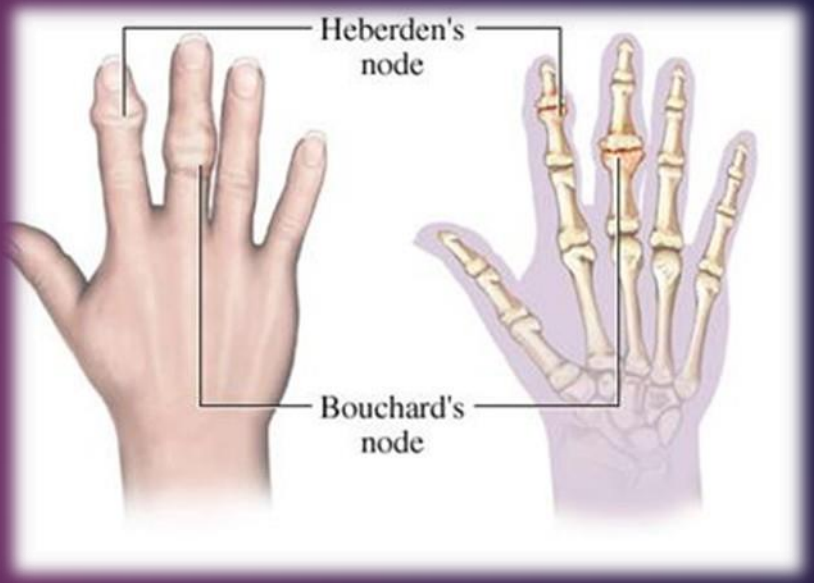
- **Pain:**
  - Activity & weight-bearing related, relieved by rest.
  - Variable over time.
  - Only one or few joints involved.

- **Morning stiffness only brief <30 minutes.**

- **Restricted functionality:**
  - Capsular thickening.
  - Blocking by osteophytes.

CLINICAL FINDINGS IN NODAL GENERALIZED OA

- Presentation typically in women. (40 & 50 years)
- Pain.
- Stiffness.
- Swelling of one or few finger interphalangeal joints (distal > proximal).
- Heberden’s nodes (+/- Bouchard’s nodes).
- Involvement of first carpometacarpal joint is common.
- Predisposition to OA at other joints specially knees.

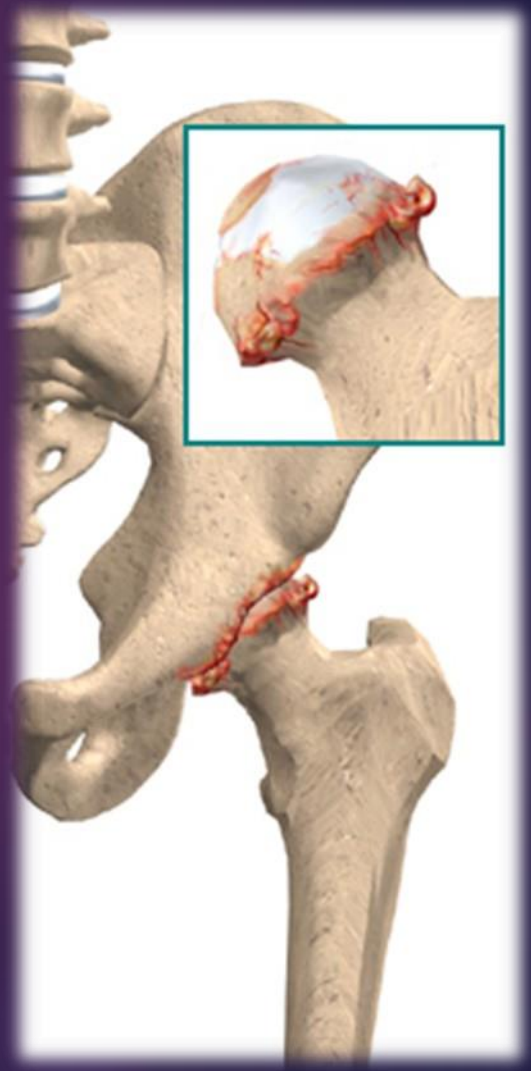


CLINICAL FINDINGS IN KNEE OA

- Targets patello-femoral & medial tibio-femoral compartments of knee.
- Pain is localized to anterior or medial aspect of knee & upper tibia.
- Jerky gait.
- Varus deformity
- Joint line &/or periarticular tenderness.
- Weakness & wasting of quadriceps muscle.
- Restricted extension & flexion.
- Bony swelling around joint.

CLINICAL FINDINGS IN HIP OA

- Targets mostly superior aspect & less commonly medial aspect of joint.
- Pain is maximally deep in groin area.
- Antalgic gait.
- Weakness & wasting of muscles (quadriceps & gluteal).
- Pain & restricted internal rotation with flexion.

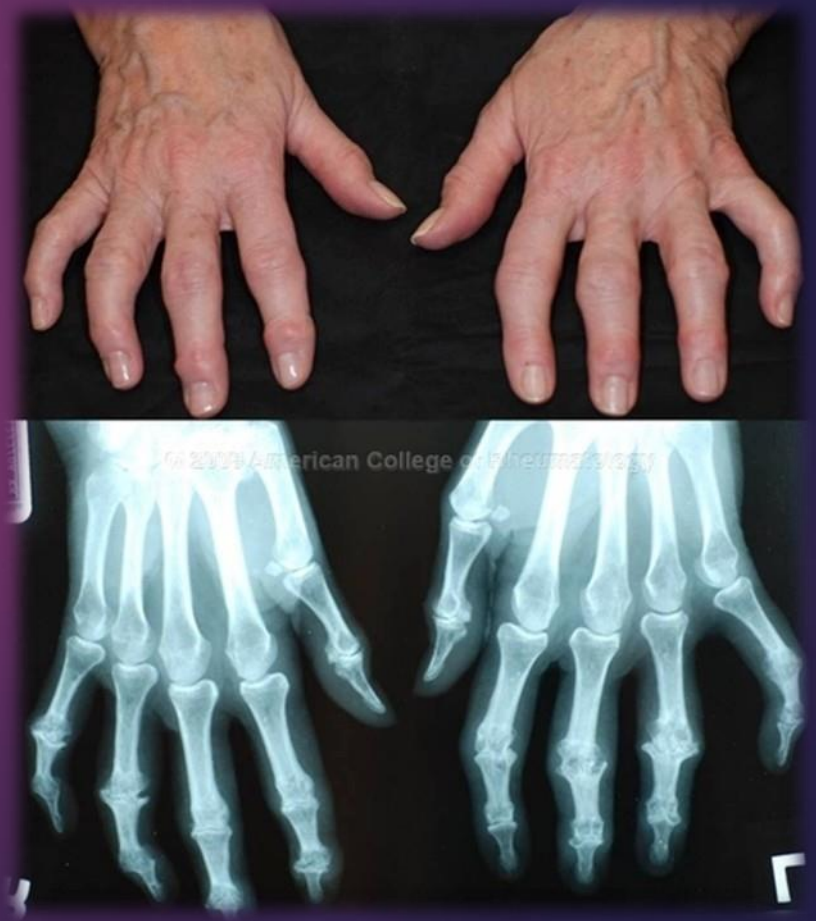


CLINICAL FINDINGS IN EARLY-ONSET OA

- Before the age of 45 years.
- Single or multiple joint involvement.
- Typical signs & symptoms of OA.

CLINICAL FEATURES IN EROSIVE OA

- Preferentially targeting proximal IPJs.
- Common development of IPJ lateral instability.
- Sub-chondral erosions on x-rays.
- Ankylosis of IPJs.

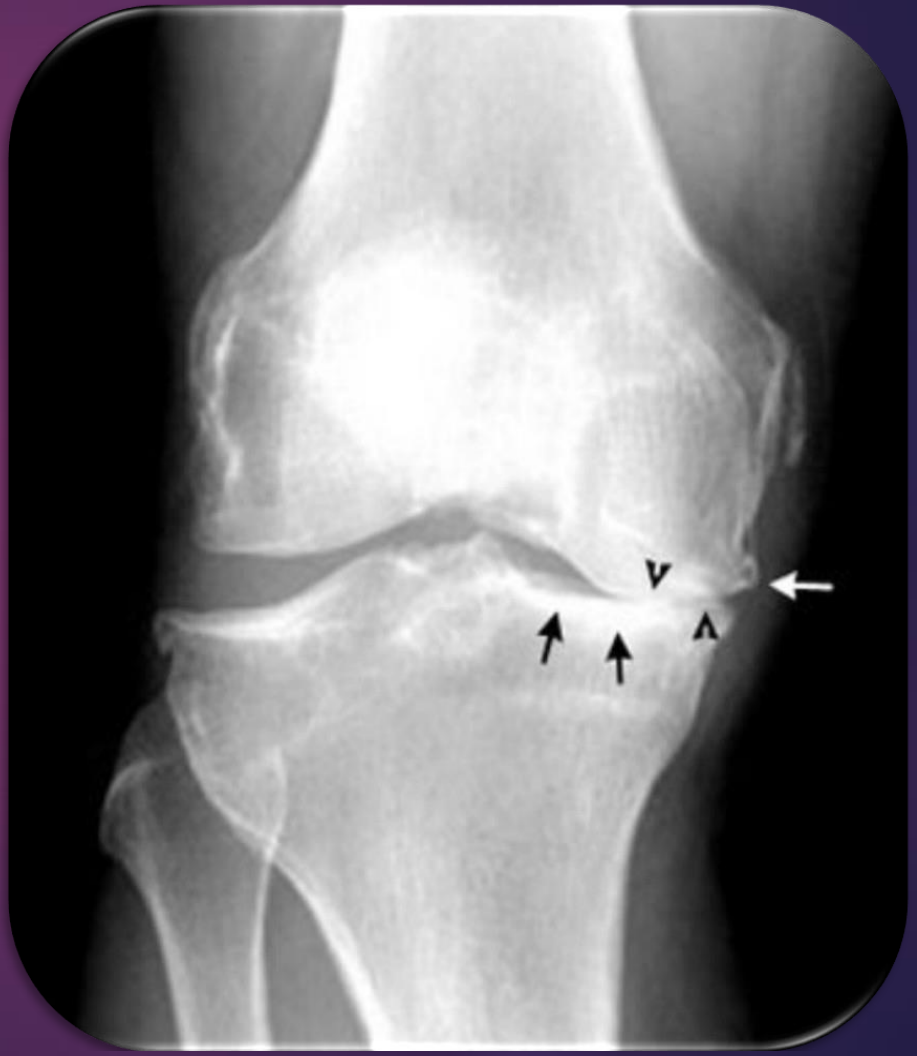


DIFFERENTIAL DIAGNOSIS

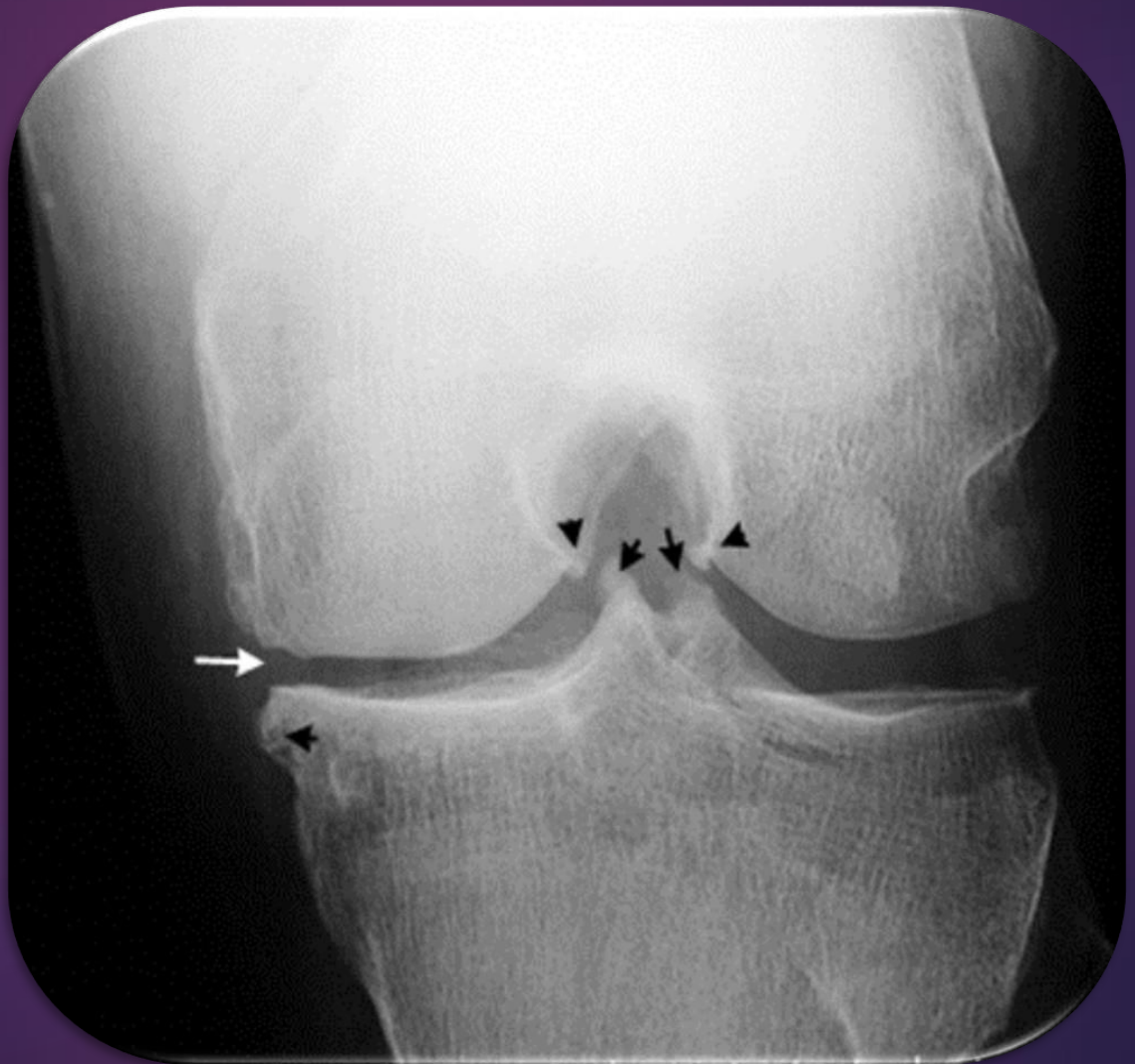
FEATURES	OSTEOARTHRITIS	RHUEMATOID ARTHRITIS	GOUT
PRESENCE OF SYMPTOMS AFFECTING THE WHOLE BODY:	Systemic symptoms are not present.	Frequent fatigue and a general feeling of being ill are present	Chills and a mild fever along with a general feeling of malaise may also accompany the severe pain and inflammation
DURATION OF MORNING STIFFNESS:	Morning stiffness lasts less than 30-60 mins;	Morning stiffness lasts longer than 1 hour.	Not seen
NODULES:	Heberden's & bouchard's nodes	Heberden's nodes are absent.	
PAIN WITH MOVEMENT:	Movement increases pain	Movement decreases pain	
AGE OF ONSET:	Most commonly occurs in individuals over the age of 50.	Usual ac[illegible]t is 20-	Usually over 35 yrs of age in men and after menopause in females
LAB FINDINGS:	Ra factor & anti-ccp antibody negative. Normal esr & c-reac[illegible] protein.	Ra factor & anti-ccp antibody positive. Esr & c-reactive protein elevated.	Joint fluid microscopy is diagnostic.

INVESTIGATIONS

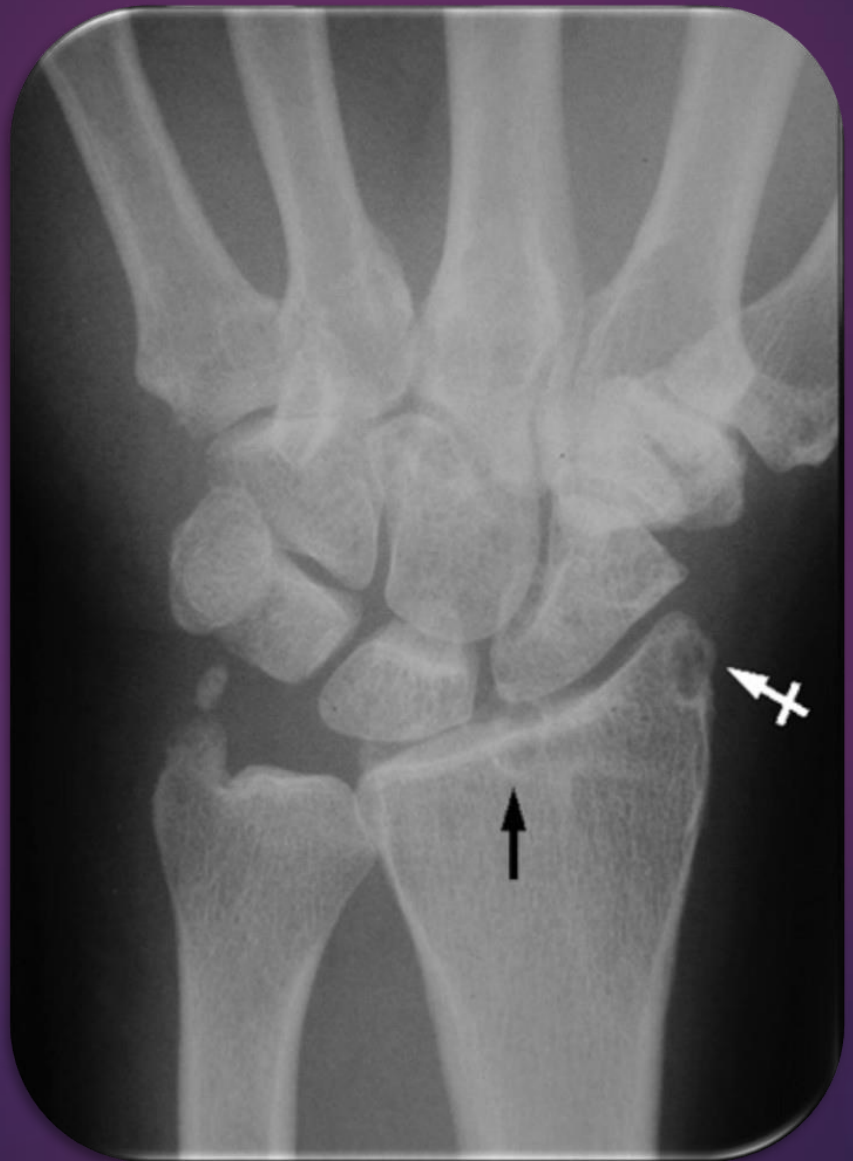
- **Plain X-Ray:**
  - **Joint space narrowing**
  - **Subchondral sclerosis**



MARGINAL OSTEOPHYTES



SUBCHONDRAL CYST



Complications

- Restricted functionality
- Spasms
- Atrophy
- Neurological deficits


THANK
YOU