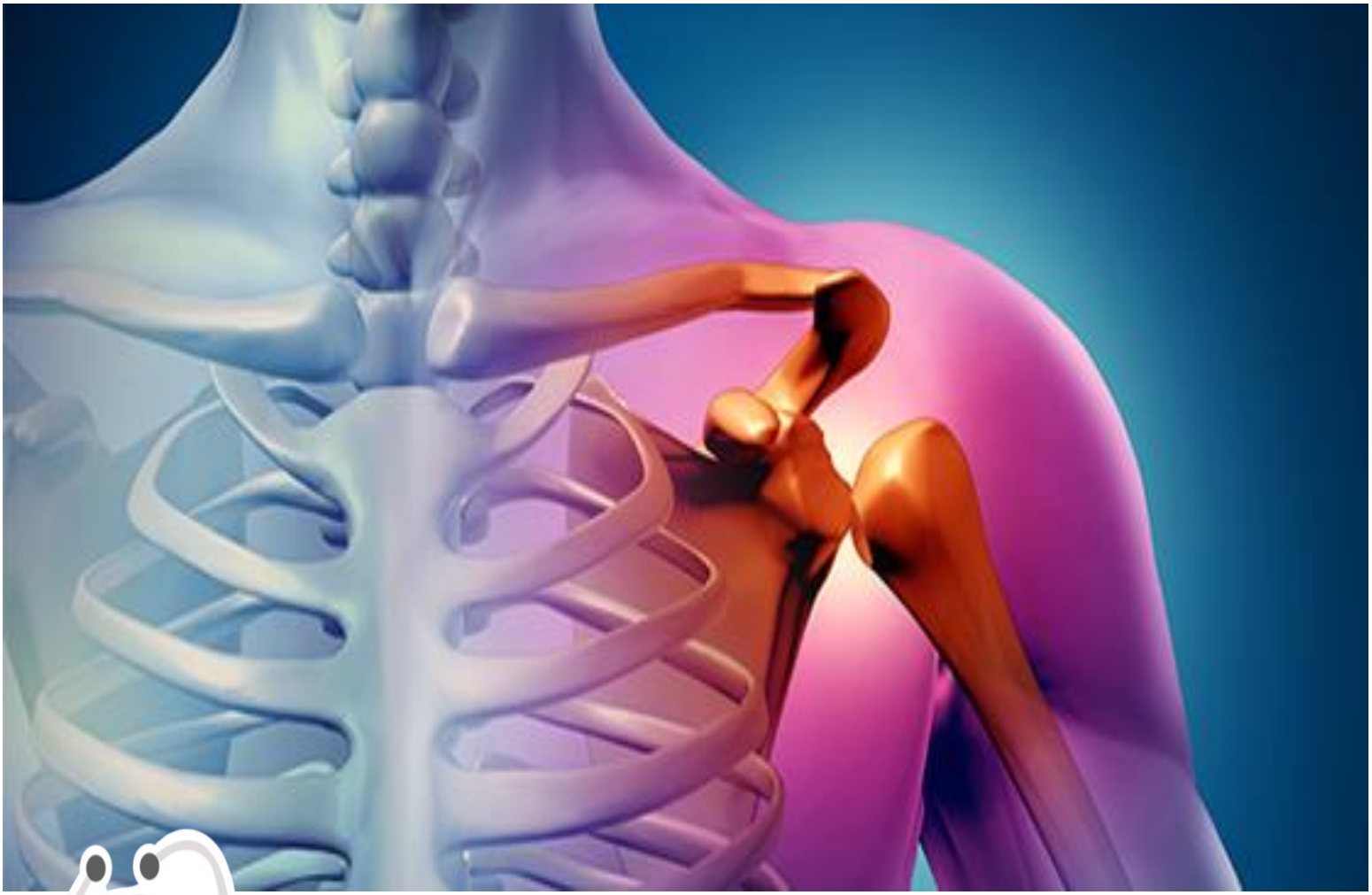
Orthopedics

5 Degenerative Joint Disease



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Color Code:

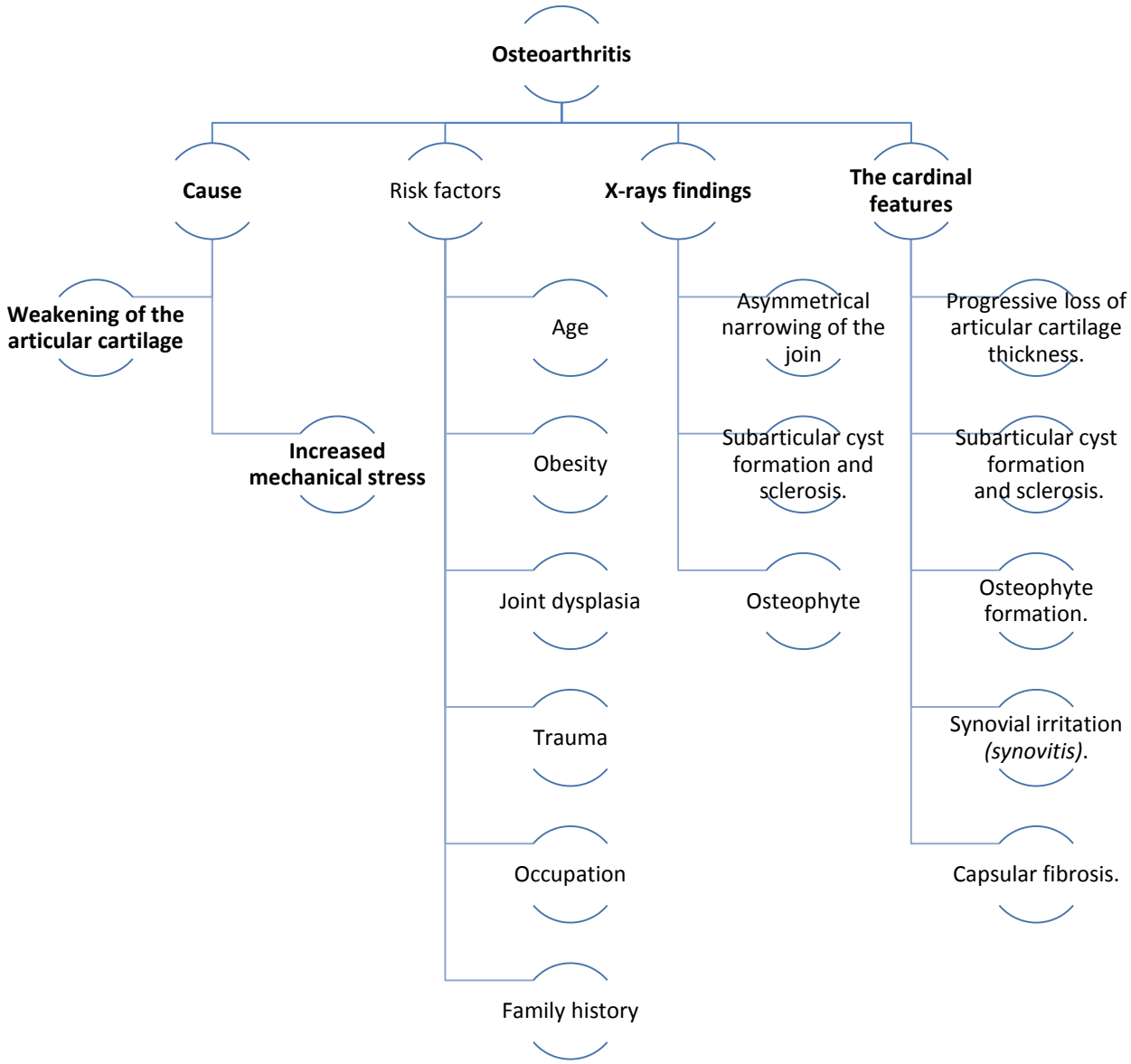
Slides	Doctor's Notes	Team Notes	Important
431 team work	Arabic Words	Books' notes	Other Sources

Objectives

To be able to specify the symptoms, signs, and predisposing factors, outline the assessment and appropriate investigation; propose a limited differential diagnosis and; outline the principles of management of a patient with:

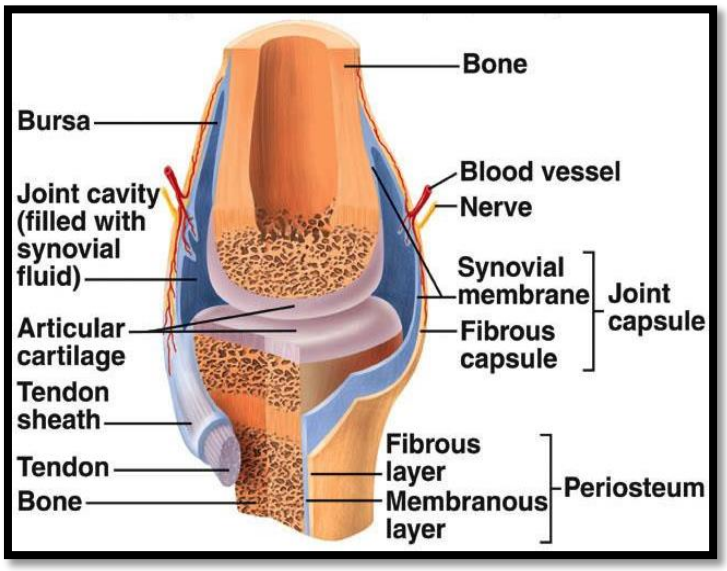
- **Degenerative OA**
- **Inflammatory arthritis** *(doctor didn't discuss it)*
  - Rheumatoid Arthritis
  - Gout
  - Seronegative spondyloarthropathy

Mind map



Quick Review of Joints Anatomy and Physiology

- **Articular Cartilage:**
  - Avascular, aneural hyaline cartilage that has some viscoelastic material with variable load bearing properties. It is essential in decreasing joints friction.
  - It is mainly composed of water (70%), type II collagen (15%) confers tensile strength to cartilage, proteoglycans (10%) negatively charged proteins hold water within the matrix and chondrocytes (5%) the only cell type in cartilage.
  - It has poor repairing properties, mostly repaired with fibrocartilage which is less effective than hyaline cartilage. If the collagen network is disrupted, the matrix becomes waterlogged and soft. Followed by loss of proteoglycans, cellular damage and splitting ('fibrillation') of the articular cartilage.
  - Damaged chondrocytes begin to release matrix-degrading enzymes in side the joint cause further damage.
- **Capsule:**
  - Fibrous structure with tough condensations on its surface *(ligaments)*. Together with the overlying muscles, they help to **provide stability**.
- **Synovium:**
  - Thin membrane that is **richly supplied with blood vessels, lymphatics and nerves.** It provides a non-adherent covering for the articular surface and produces synovial fluid. It is the target of autoimmune reactions in joint infections (septic arthritis) and autoimmune disorders such as rheumatoid arthritis and SLE.
- **Synovial fluid:**
  - Have several functions: Nourishes the avascular articular cartilage, plays an important part in reducing friction during movement, it also has slight adhesive properties, which help in maintaining joint stability.
  - The volume remains fairly **constant**, regardless of movement.
    Unless the joint got injured it **increases resulting in joint effusion**.



* Please watch this 5 minutes video before start studying the lecture. It will help to understand some points:
https://www.youtube.com/watch?v=41IMR_Dp5bs

Terminology

- **Osteoarthritis (OA)** is also known as degenerative arthritis or degenerative joint disease or osteoarthrosis.
- **Hemarthrosis:** bleeding into joint spaces.
- **CPPD:** Calcium pyrophosphate deposition disease.
- **Reiter's syndrome:** an inflammatory syndrome *(etiology unknown)* predominantly in males; characterized by arthritis, conjunctivitis and urethritis.
- **Tabes dorsalis:** a progressive deterioration of the spinal cord and spinal nerves associated with tertiary syphilis.
- **Loose bodies:** is a fragmented osteophyte.
- **Spondylolisthesis:** forward displacement of a vertebra over a lower segment.

Quick review:

Osteoarthritis x-ray findings mnemonic: LOSS	Rheumatoid arthritis x-ray findings mnemonic: LESS
L: Loss of joint space (Asymmetrical)	L: Loss of joint space (symmetrical)
O: Osteophyte formation	E : Erosion of joint
S : Subchondral sclerosis	S : Synovial thickening
S : Subchondral cysts	S : Subluxation and joint deformities

Osteoarthritis

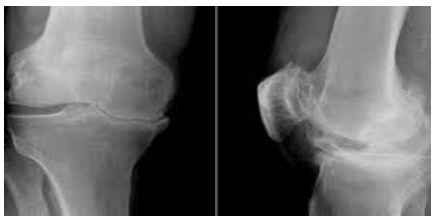
Osteoarthritis (OA) is a chronic, non-inflammatory, degenerative joint disorder in which there is progressive softening and disintegration of articular cartilage followed by new growth of cartilage and bone at the joint margins (osteophytes), because there is no cartilage any more bones expose to more pressure lead to Subchondral bone sclerosis and cyst formation, mild (compared to the inflammatory) synovitis and capsular fibrosis.

It has two types: **Primary** *(idiopathic)* and **secondary** (table 1).

Etiology:

OA results from a disparity between the stress applied to articular cartilage and the ability of the cartilage to withstand that stress. This could be due to one or a combination of two processes:

1) **Weakening of the articular cartilage** *(due to a genetic defect or enzyme activities).*
2) **Increased mechanical stress** in some part of the articular surface. Which can be caused by overuse or joint instability.



The initial trigger for OA appears to be the damage to the cartilage collagen network and loss of proteoglycans from the matrix, giving rise to deformation and gradual structural disintegration. **Varus deformity** of the knee. **Medial side** is the most affected.

Cause	Examples
Metabolic	Crystalline deposition disease *(Gout, CPPD),* Paget's disease
Inflammatory	RA, SLE, Reiter's syndrome
Neuropathic	DM, Tabes dorsalis
Hematologic	Sickle cell disease, Hemophilia *(due to recurrent hemarthrosis)*
Endocrine	DM, Acromegaly
Trauma	Osteochondral, Malunion, Sport injury
Congenital/developmental	Hip dyplasia, Multiple epiphyseal dysplasia
Necrosis an Infections	Perthe's disease, osteonecrosis, steroids

Prevalence:

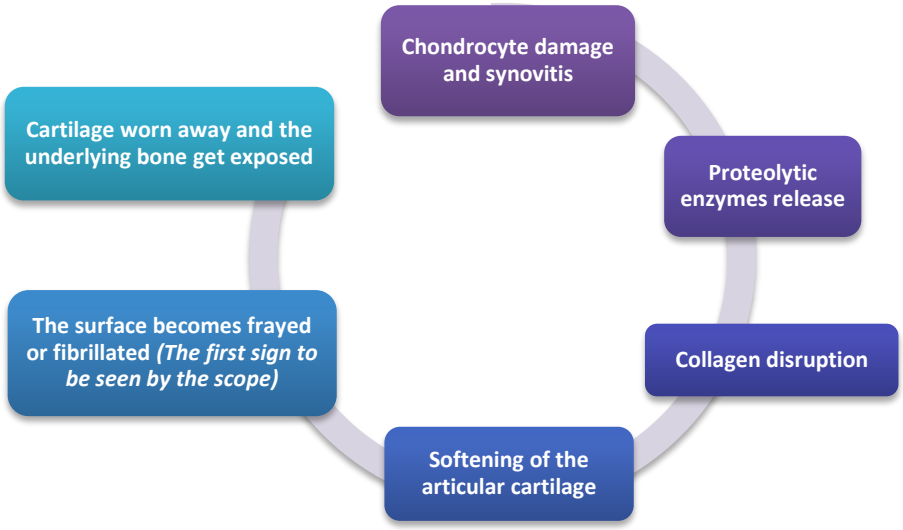
- Osteoarthritis is the commonest of all joint diseases.
- Osteoarthritis is much more common in some joints (hip, knee, spine and the fingers) than in others (the elbow, wrist and ankle).
- Commonest joints are knee, hip, Cervical spine & Lumbar Spine,1st Carpometacarpal,1st Metatarsophalangeal and Interphalangeal joints.
- Common in our community especially knees.
- Much more in females and more joints are affected in women.
- Presents earlier than in West.
- About 90% of those over 40 have asymptomatic degeneration of weight bearing joints.

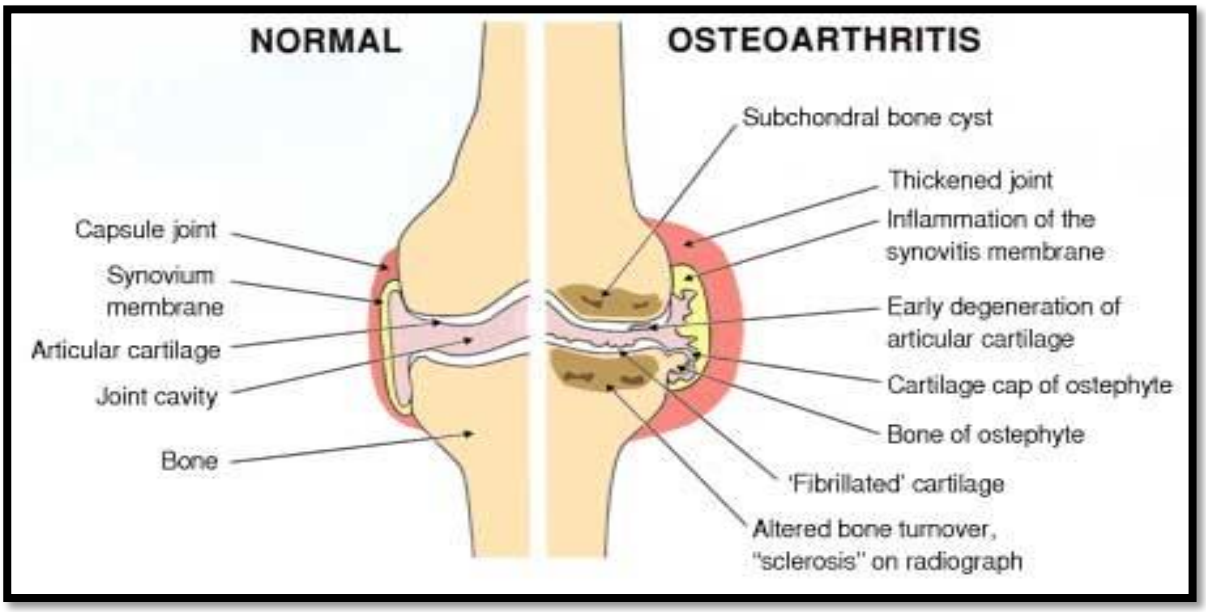
Pathology:

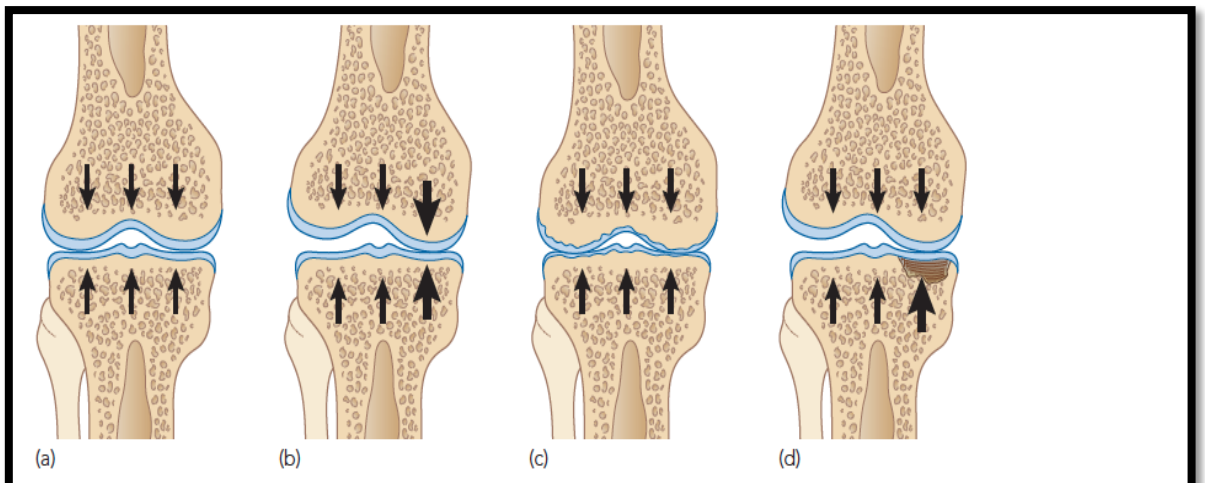
The cardinal *(major)* features are:

1) Progressive loss of articular cartilage thickness.
2) Subarticular cyst formation and sclerosis.
3) Osteophyte formation.
4) Synovial irritation *(synovitis)*.
5) Capsular fibrosis.

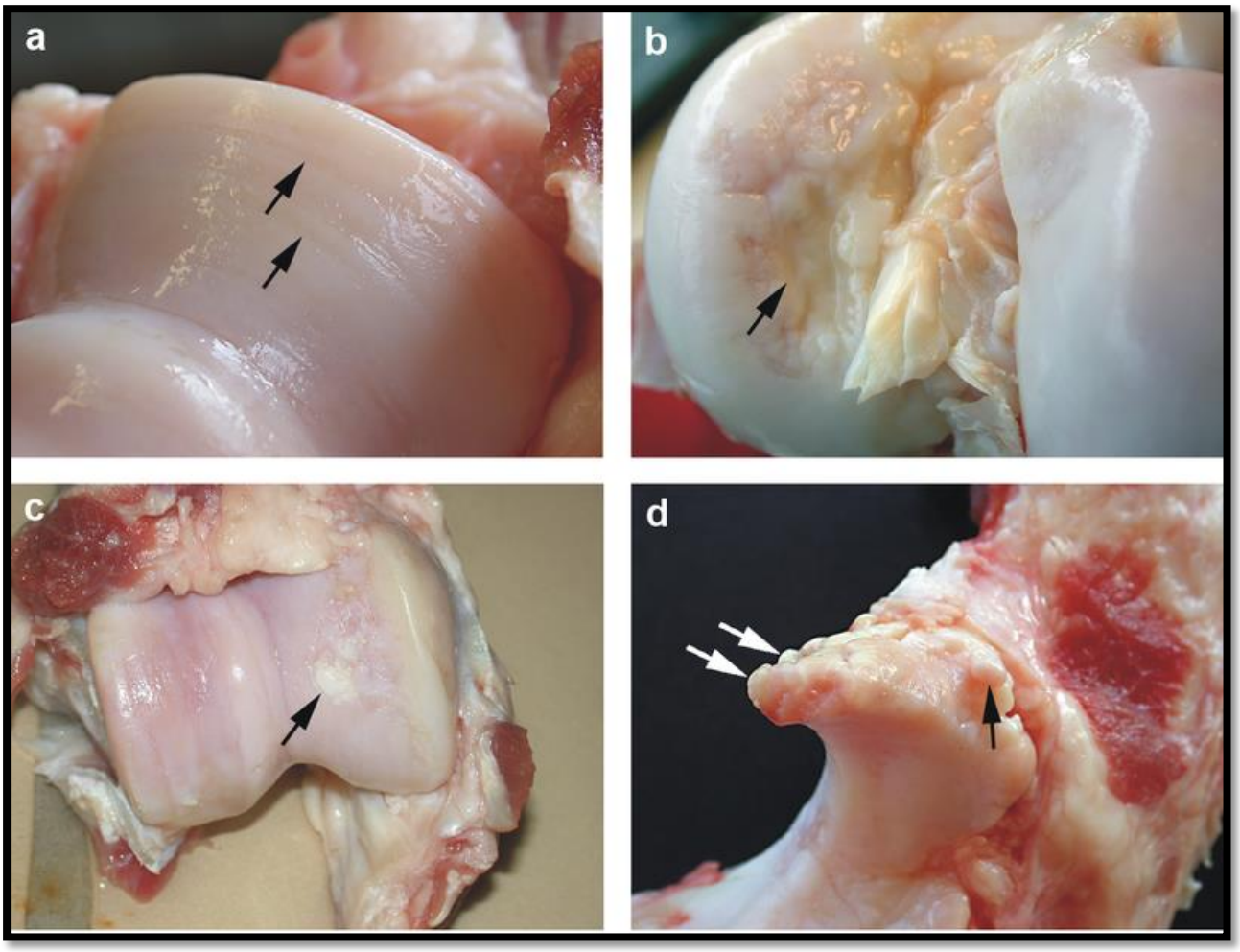
Subarticular cyst could arise from local areas of osteonecrosis or from the forceful pumping of synovial fluid through cracks in the subchondral bone plate.
Osteophytes arise from proliferation and remodeling of the adjacent cartilage at the edges followed by enchondral ossification of that cartilage.
OA at the beginning is asymmetrically distributed, often localized to only one part of a joint. In the area of greatest stress in the joint, cysts appear and the surrounding trabeculae become thickened or sclerotic. There are vascular congestion and the intraosseous pressure rises. Meanwhile, as the disease progresses, cartilage in peripheral, unstressed areas proliferate and ossifies, producing bony outgrowths *(osteophytes)*.
OA shows features of both destruction *(cartilage erosion)* and repair *(osteophytes)*. Although OA is not primarily an inflammatory disease, shedding of fragments from the fibrillated articular cartilage, as well as release of enzymes from damaged cells, may give rise to a low-grade synovitis that may produce local signs of inflammation. In the late stages, capsular fibrosis is common and may account for joint stiffness.



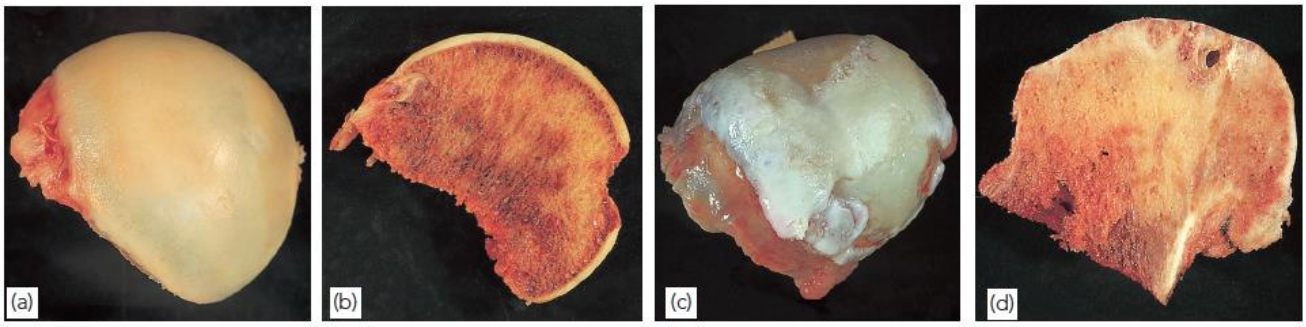


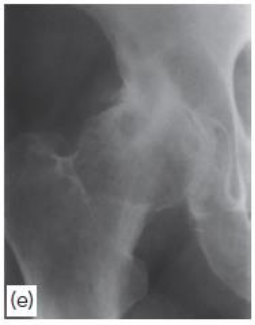


5.1 Osteoarthritis – pathogenesis In a normal joint (a), the loading forces are evenly distributed. Cartilage damage results from: (b) increased stress on some part of the articular surface; (c) a preceding inflammatory disorder which weakens the cartilage and renders it unable to bear even normal loads; or (d) abnormality of the subarticular bone which alters its ability to support the cartilage appropriately.



Damaged cartilage from sows. (a) cartilage erosion (b) cartilage ulceration (c) cartilage repair (d) osteophyte (bone spur) formation.





5.2 Osteoarthritis – pathology (a) Normal ageing causes slight degeneration of the articular surface, but the coronal section (b) shows that the cartilage thickness is well preserved. By contrast, in progressive osteoarthritis the weightbearing area suffers increasing damage. In the femoral head specimen (c), the superior surface is completely denuded of cartilage and there are large osteophytes around the periphery. In the coronal section (d), the subarticular cysts are clearly revealed; the x-ray (e) shows that the superolateral joint space (cartilage) has virtually disappeared and there are cysts in the underlying bone.

Clinical features:

Cartilage changes start 10 or even 20 years before symptoms. OA has two clinical variants;

1) **Monarticular and pauciarticular OA:** characterized by pain *(due to* Bone pressure caused by vascular congestion and intraosseous hypertension most important, mild synovial inflammation and *capsular fibrosis)* and dysfunction in one or two of the large weight-bearing joints.
2) **Polyarticular (generalized) OA:** the most common form,
   - Characterized by pain, swelling and stiffness of the distal finger joints.
   - The first carpometacarpal, big toe metatarsophalangeal joints, knees and lumbar facet joints, may be affected as well.
   - Over a period of years osteophytes and soft-tissue swelling produce a characteristic knobbly appearance of the distal interphalangeal joints *(Heberden's nodes)* and, less often, the proximal interphalangeal joints *(Bouchard's nodes)*
   - Pain may disappear but stiffness and deformity can be disturbing.

OA pain is starts insidiously and increases slowly over months or years. It is aggravated by exertion and relieved by rest. Stiffness is worst after periods of rest. Typically symptoms follow an intermittent course, with periods of remission sometimes lasting for months. Night pain or pain at rest, swelling, deformity, tenderness, crepitus on movement, loss of mobility, muscle wasting, and joint instability (because the tissues contracted on one side and they are stretched on the other side and the muscles are fatigued) are features of advanced disease. OA is unassociated with any systemic manifestations.

OA is uncommon in the shoulder, elbow, wrist and ankle. If any of these joints is affected you should suspect a secondary cause or previous injury to that joint.

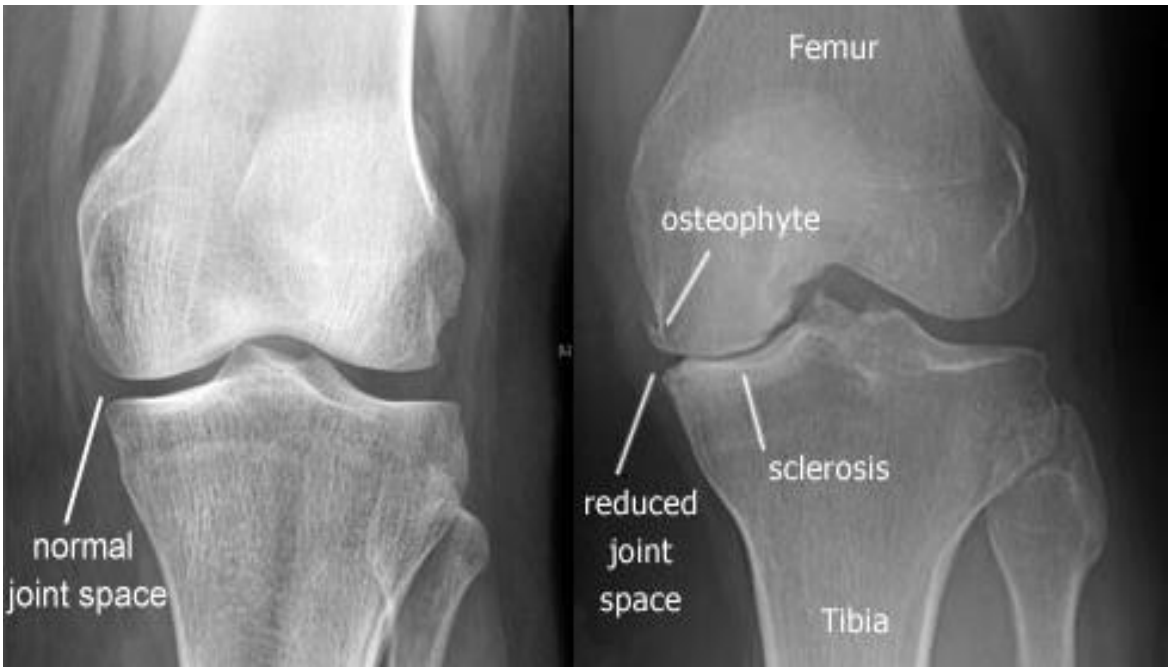
Muscular fatigue, Mild synovial inflammation

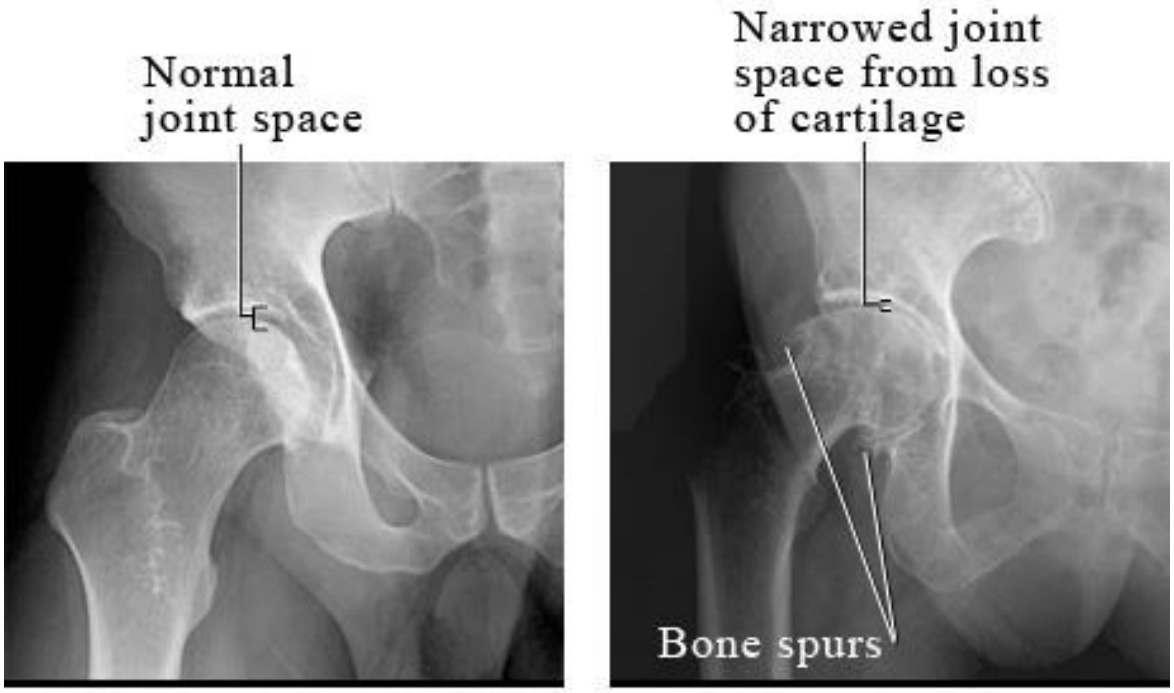
Risk factors:

- **Age** (increase with age).
- **Obesity** *(hips and knees take 3-4 body weight with each step).*
- **Joint dysplasia:** Congenital acetabular dysplasia, Perthes' disease and slipped upper femoral epiphysis.
- **Trauma:** fractures involving the articular surface, and joint instability.
- **Occupation:** continuous knee-bending activities.
- **Family history.**

X-rays findings:

- Asymmetrical narrowing of the joint space *(Symmetric narrowing indicate inflammatory OA like RA).*
- Subarticular cyst formation and sclerosis.
- Osteophyte formation at the margins.
- Evidence of previous disorders that may have increased OA risk.
- **Late features:** Malalignment, Joint subluxation, Bone loss, Loose bodies.





Complications:

- **Capsular herniation:** Knee OA, marked effusion and herniation of the posterior capsule *(Baker's cyst)*.
- **Rotator cuff dysfunction:** acromioclavicular *(AC)* joint OA.
- **Spinal stenosis.**
- **Loose bodies.**
- **Spondylolisthesis:** severe segmental instability; at L4/5.

Treatment:

Depends on several factors:

- Joint *(or joints)* involved.
- Stage of the disorder.
- Severity of the symptoms
- Age of the patient.
- Functional need.

❖ **Early stage of the disease:** **There are three principles:**

(1) Relieve pain; by *Acetaminophen, NSAIDs, rest periods and modification of activities (avoiding activities like climbing stairs, squatting and praying on the floor), application of warmth, massage.*

(2) Increase joint mobility; by *physiotherapy, even a small increase in range and power will reduce pain, prevent contractures and improve function.*

(3) Reduce load; by *using a walking stick, unloading brace, wearing Shock-absorbing shoes, avoiding prolonged stressful activity and by weight reduction.*

❖ **Intermediate stage of the disease:**

Arthroscopic joint debridement *(removal of interfering osteophytes, meniscal or labral tears and loose bodies that cause blocking of the movement or cracking)*, for OA of the knee

Corrective osteotomy *(redistribution of loading forces towards less damaged parts)*, it provides a significant pain relive because it provided vascular decompression, usual candidates are young, active patients with mild OA (you still have articular cartilages that you want to save). Nowadays the advances in joint replacement surgery have superseded it especially partial joint replacement.

❖ **Late stage of the disease:**

Joint replacement (Arthroplasty) is the procedure of choice for OA in patients with severe and advanced symptoms. Can be total for old patients with painful deformed stiff joint in or partial for young active patients with mild OA.

Arthrodesis *(Fusion of the two ends of the bones)* is sometimes indicated for joints in which permanent stiffness is not a drawback. Relive only pain and cause permanent stiffness *(in small joints e.g. in hand, foot and spine).*

For those whom interested this is a 10 minutes video of a total knee arthroplasty:
https://www.youtube.com/watch?v=Nmb5-e3cwBw

Summary

- **OA** is common and present earlier in our region compared to the west.
- **Commonest joints to be involved** are Knee, Hip, Cervical & Lumbar Spine, 1st Carpometacarpal, 1st Metatarsophalangeal *(called **hallux stiffness**)* and interphalangeal joints.
- Swelling either intermittent (effusion) or continuous (large osteophytes).
- **Locally applied medication** are not recommended.
- **Progressive bone erosion** will lead to bone collapse.
- Loss of height and ligamentous laxity will lead to **malalignment**.
- **Varus deformity** can be seen in a knee joint with OA.
- **Indications of surgery:** Pain *(most common)*, Sub-laxation, and severe bone erosion.
- It's important to ask about the **Activities Of daily Living "ADL"** whenever the patient is complaining of pain.
- Deformity alone by itself is NOT an indication of surgical Rx.
- **Varus mal-alignment and fixed flexion** are common in OA, while in Rheumatoid it's more common to have **valgus and hyperextension**.
- In synovial analysis for OA both **protein level and glucose level** are normal.
- **X-ray** is the best modality for investigation with AP & lateral standing views + Skyline "Axial" view in case of hip or knee OA to show the patella-femoral joint.
- **Hip disease** can transfer to the knee and vice versa.
- **Arthroscopic lavage** is the same as arthroscopic joint debridement.
- **Steroid injections** has NO effect on OA as a main method of treatment, it's only beneficial in case of acute presentation of OA because its duration of effect = 3 weeks.
- **Hyaluronic acid injection** has almost similar results of arthroscopy, so it's preferred as it doesn't require anesthesia.

(Cont.)

- Normal Anterior cruciate ligament, knee stability, and range of motion should be considered before performing **Osteotomy**.
- **Partial arthroplasty** is usually indicated for trauma *(e.g. femur neck, hip and knee fractures).* It is contraindicated in rheumatoid arthritis.

MCQ's

Q1) Which of the following is the first sign to be seen on x-ray in a patient with OA?

A. Osteophyte
B. Subarticular cyst
C. Subarticular sclerosis
D. Asymmetrical narrowing of the joint space

Q2) Which of the following will NOT significantly affect your decision when managing a patient with OA?

A. The joint involved
B. Stage of the disorder
C. Severity of the symptoms
D. Age of the patient
E. Functional needs
F. Number of children

Q3) What is the procedure of choice for OA in patients with severe and advanced symptoms?

A. Arthrodesis
B. Arthroplasty
C. Corrective osteotomy
D. Arthroscopic joint debridement

Q4) Which of the following is NOT one of the main principals of management of early OA?

A. Relieve pain
B. Increase joint mobility
C. Reduce load
D. Reduce swelling

Q1= D

Q2= F

Q3= B

Q4= D