

OSTEOARTHRITIS

435 medicine teamwork

[**Important** | **Notes** | Extra | **Editing file**]

lecture objectives:

⇒ Not given -_-

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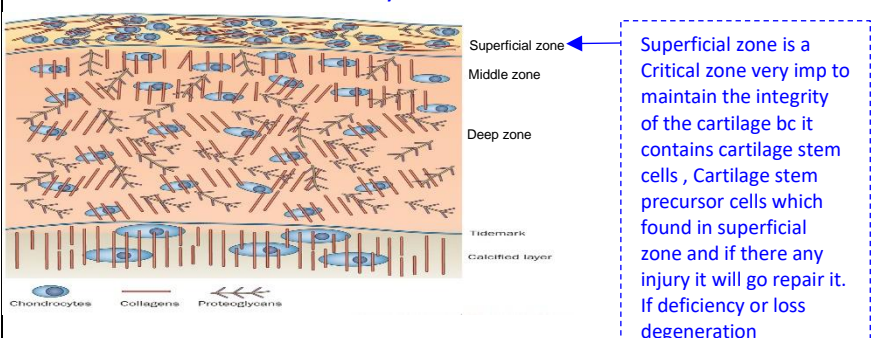
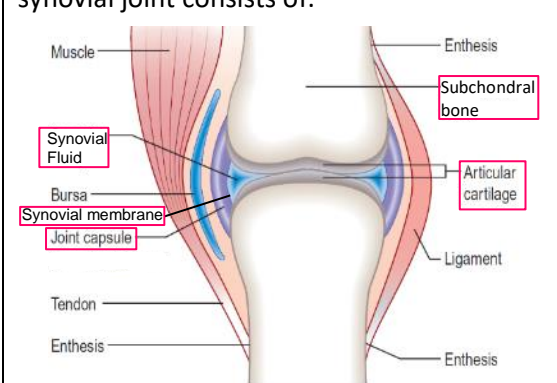
References:

Slides+Davidson+Kumar+Master The board

Basic Review of The Normal Joint

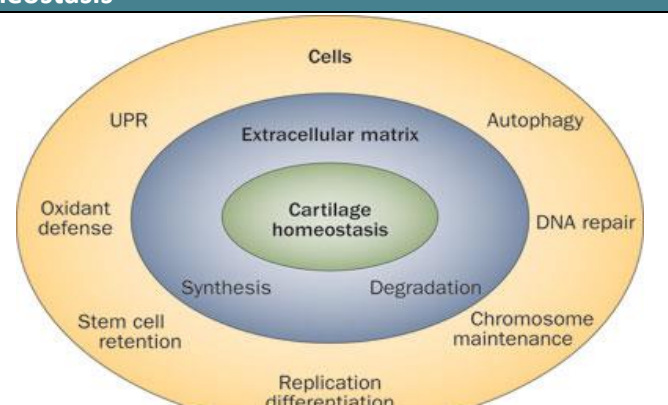
Joint Anatomy:

Joints classification					
Functional			structural		
Synarthroses (immovable)	Amphiarthroses (slightly moveable)	Diarthroses (freely moveable)	fibrous	cartilaginous	synovial
e.g.: Skull	e.g.: symphysis pubis, vertebral	e.g.: shoulder	e.g.: Skull	e.g.: symphysis pubis	e.g.: knee, elbow, shoulder

The normal articular surface of synovial joints	Synovial joint anatomy
<ul style="list-style-type: none"> ✓ articular cartilage (chondrocytes) surrounded by extracellular matrix includes: <u>proteoglycans</u> and <u>collagen</u>. ✓ The cartilage facilitates joint function and protects the underlying subchondral bone by distributing large loads, maintaining low contact stresses, and reducing friction at the joint. ✓ Hyaline cartilage forms the articular surface and is AVASCULAR. ✓ It relies on diffusion from synovial fluid for its nutrition.  <p>Superficial zone is a critical zone very imp to maintain the integrity of the cartilage bc it contains cartilage stem cells, Cartilage stem precursor cells which found in superficial zone and if there any injury it will go repair it. If deficiency or loss degeneration</p>	<p>synovial joint consists of:</p>  <p>Two bones articulating in this area of long bone And now what you are see is where they are articulate there is structure which called cartilage and it is surrounded by a cavity which is filled with synovial fluid which come from synovial membrane that lined the joint capsule</p> <p>Enthesis: Structure where must be muscle tendon attach to a bone</p>

Synovial Fluid
<p>Its synthesis:</p> <ul style="list-style-type: none"> → Synovial fluid is formed by (synoviocytes). → Synovial cells also manufacture hyaluronic acid (HA), also known as hyaluronate):a glycosaminoglycan that is the major noncellular component of synovial fluid. <p>Its Functions:</p> <ul style="list-style-type: none"> ✓ Synovial fluid supplies nutrients to the avascular articular cartilage; it also ✓ provides the viscosity needed to absorb shock from slow movements ✓ provides elasticity required to absorb shock from rapid movements

Joint Physiology:

Cartilage homeostasis	
<p>How can the cartilage maintain its integrity?</p> <p>balancing between syntheses and degradation</p> <p>Cartilage matrix is constantly turning over and in health there is a perfect balance between synthesis and degradation. Degradation of cartilage matrix is carried out by aggrecanases(a proteolytic enzymes) and matrix metalloproteinases, responsible for the breakdown of proteins and proteoglycans, and by glycosidases, responsible for the breakdown of GAGs.</p> <p>In inflammatory arthritis:</p> <p>Pro-inflammatory cytokines, such as interleukin-1 (IL-1) and tumour necrosis factor (TNF), stimulate production of aggrecanase and metalloproteinases, which contribute to cartilage degradation in inflammatory arthritis.</p>	

Osteoarthritis(OA)

General Characteristic of OA:

What is it:

- Heterogeneous group of conditions resulting in common histopathologic and radiologic changes **or at clinical level** involving Entire joint *organ*, including:
 - o the articular cartilage
 - o the subchondral bone and
 - o the synovium.
- It characterized by progressive destruction and loss of articular cartilage with an accompanying periarticular bone response.

Epidemiology:

- Internationally, osteoarthritis is the **most common articular disease**. Estimates of its frequency vary across different populations.
- The prevalence of OA increases with age, and most people over 60 years will have some radiological evidence of osteoarthritis although only a proportion of these have symptoms(Does not have necessary to be clinical evidence).
- the prevalence of osteoarthritis is higher among women than among men.
- **Interethnic** differences in the prevalence of osteoarthritis have been noted (e.g.: white people have chance to get knee OA than black..).

Involved Joints:

- **most commonly involved joints:**
 - o the distal interphalangeal joints (DIPJs) and first carpometacarpal joint of the hands
 - o first metatarsophalangeal joint(MTP) of the foot
 - o the weight-bearing joints (vertebrae, hips and **knees**).
- **rarely affected joints:** Elbows, wrists and ankles.

MCP is usually spared in OA while DIP can be affected those helping u to rule out RA

Aetiology:



Box 11.4 Factors predisposing to osteoarthritis

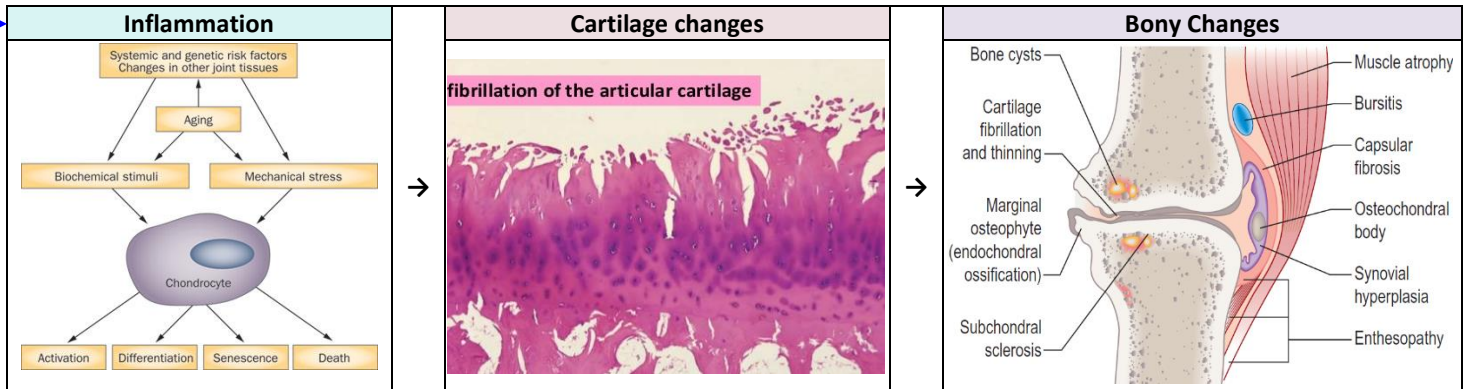
- **Obesity**
- **Heredity:** Familial tendency to develop nodal and generalized OA
- **Gender:** Polyarticular OA is more common in women; a higher prevalence after the menopause suggests a role for sex hormones
- **Hypermobility:** Increased range of joint motion and reduced stability lead to OA
- **Osteoporosis:** There is a **reduced risk** of OA
- **Diseases:** See Table 11.12→
- **Trauma:** A fracture through any joint. Meniscal and cruciate ligament tears قطع الرباط الصليبي cause OA of the knee
- **Congenital joint dysplasia:** Alters joint biomechanics and leads to OA. Mild acetabular dysplasia is common and leads to earlier onset of hip OA
- **Joint congruity:** Congenital dislocation of the hip or a slipped femoral epiphysis causes early-onset OA
- **Occupation:** Miners develop OA of the hip, knee and shoulder, cotton workers OA of the hand, and farmers OA of the hip
- **Sport:** Repetitive use and injury in some sports causes a high incidence of lower-limb OA.

Table 11.12 Causes of osteoarthritis

Primary OA	No known cause
Secondary OA	<p>Pre-existing joint damage:</p> <ul style="list-style-type: none"> Rheumatoid arthritis Gout Spondyloarthritis Septic arthritis Paget's disease Avascular necrosis, e.g. corticosteroid therapy <p>Metabolic disease:</p> <ul style="list-style-type: none"> Chondrocalcinosis <small>(calcium pyrophosphate deposition in the cartilage)</small> Hereditary haemochromatosis Acromegaly <p>Systemic diseases:</p> <ul style="list-style-type: none"> Haemophilia – recurrent haemarthrosis Haemoglobinopathies, e.g. sickle cell disease Neuropathies

Pathology and pathogenesis:



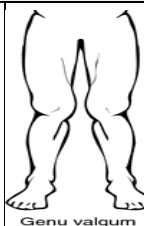
Pathogenesis :		
Inflammation	1	Inflammation occurs as cytokines and metalloproteinases* are released into the joint * Metalloproteinases, e.g. stromelysin and collagenase, secreted by chondrocytes degrade collagen and proteoglycans.
Cartilage changes	2	lead to breakdown of the cartilage matrix most imp thing to remember is there's degradation of Extracellular matrix (the level of proteoglycans eventually drops very low, the cartilage softens and lose elasticity and compromising joint surface integrity)
	3	Flaking and fibrillations (vertical clefts) develop along on the surface of an osteoarthritic joint. o Over time, the loss of cartilage results in loss of joint space
Bony Changes	4	The exposed subchondral bone responds with vascular invasion and increased cellularity, becoming thickened and dense (a process known as eburnation (sclerosis)) at areas of pressure
	5	Attempts at repair produce cartilaginous growths at the margins of the joint which later become calcified (osteophytes).
	6	subchondral bone undergo cystic degeneration. o Osteoarthritic cysts are also termed as: subchondral cysts, pseudocysts, geodes, or Egger cysts if it involved the acetabulum(hip) o Osteoarthritic cysts may range from 2 to 20 mm in diameter



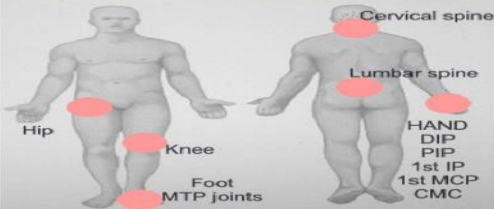


What is the different between the inflammation in RA and OA?

- RA: there is initial inflammation which is going to cause inflammatory response and damage.
- In OA: secondary inflammation occurring to whatever damage e.g. genetic or environmental which lead to inflammation and different mediatory mediator

Clinical features:

Presenting Symptoms				
<ul style="list-style-type: none"> - The main presenting symptoms are joint pain and functional restriction: <ul style="list-style-type: none"> o in a patient over the age of 45, but more often over 60 years. o Joint pain made worse by movement and relieved by rest - Stiffness occurs after rest ('gelling') and in contrast to inflammatory arthritis there is only transient (Less than 30 minutes) morning stiffness. 				
On Examination				
periarticular tenderness	limited joint movement	muscle wasting of surrounding muscle	Crepitus (grating) is a common finding and it is Palpable, sometimes audible when bending the joint, coarse crepitus due to rough articular surfaces.	Synovitis mild or absent
deformity and bony enlargement of the joints:				
Heberden's nodes are bony swellings at the DIPJs 	Bouchard's nodes are bony swellings at the IPJs	varus deformity resulting from marked medial tibio-femoral osteoarthritis (bone twist is toward the center of the body) 	Valgus deformity Less commonly (bone twist is away from the center of the body) 	

Subtypes of Primary OA:

Three Suptypes:		
Primary generalized OA	Erosive osteoarthritis	Chondromalacia Patellae(Knee OA)
<ul style="list-style-type: none"> - common form of OA - This is usually seen in combination with nodal OA(Generalized nodal osteoarthritis) - Its onset is often sudden and severe. - There is a female preponderance and a strong familial tendency. - The other joints affected are the knees, first MTP,hip, and intervertebral (spondylosis). <p>Osteoarthritis - Anatomical Distribution</p> 	<ul style="list-style-type: none"> - This is rare. - The DIPs and PIPs are inflamed, and equally affected and the functional outcome is poor. - MCP is spared - Radiologically, there is marked osteolysis. - Destructive phases are followed by phases of remodelling. 	<p>is a condition where the cartilage on the undersurface of the patella deteriorates and softens.</p> <p>Degeneration of the cartilage underneath the patella and this is more seen in the runner and younger people</p> 

Differential diagnosis:

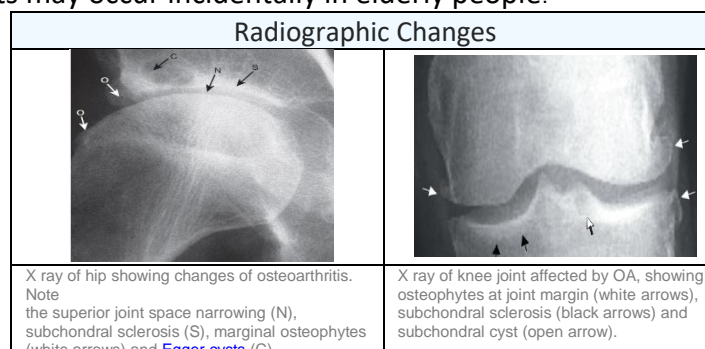
- **Crystalline arthropathies (ie, gout and pseudogout):** Examination of synovial fluid using compensated polarized microscopy will demonstrate crystals
- **Inflammatory arthritis (eg, rheumatoid arthritis):**OA is differentiated from RA by the pattern of joint involvement and the absence of the systemic features and marked early morning stiffness that occur in RA.
- **Seronegative spondyloarthropathies (eg, psoriatic arthritis and reactive arthritis):**affecting the DIPJs may mimic OA.
- **Septic arthritis or postinfectious arthropathy**
- **Fibromyalgia:**The main presenting feature of fibromyalgia is widespread pain, which is often worst in the neck and back ,The pain is characteristically diffuse and unresponsive to analgesics
- **Tendonitis**

Investigations:

- 1) **The most accurate test(DIAGNOSTIC) is X-rays:** are only abnormal in advanced disease and show narrowing of the joint space (resulting from loss of cartilage), osteophytes, subchondral sclerosis and cyst formation.x ray won't show the cartilage
- 2) MRI demonstrates early cartilage changes. (**It is not necessary** for most patients with suggestive symptoms and typical plain X-ray features).**MRI looks for tendon ,muscle any soft structure**
- 3) Laboratory tests are normal:Full blood count and ESR are normal. Rheumatoid factor is negative, but positive low-titre tests may occur incidentally in elderly people.

► Notes:

Arthrocentesis(aspiration of synovial fluid) always indicated when an infected or crystal induced arthritis is suspected, particularly a monoarthritis



MCQ!!! X ray shows joint space narrowing ,osteophytes & subchondral cyst? Your mind always should think of OA

Management:

Non pharmacologic	1	Life style modification, physical and rehab therapy I can't emphasize this enough <ul style="list-style-type: none">- Obese patients should be encouraged to lose weight, particularly if weight-bearing joints are affected.- Physical measures are the keystone of OA treatment. Local strengthening and aerobic exercises improve local muscle strength, improve the mobility of weight-bearing joints and improve general aerobic fitness
Pharmacotherapy	2	Medication: <ul style="list-style-type: none">- Paracetamol is the initial drug of choice for pain relief,- NSAIDs are used in patients who do not respond to simple analgesia and should be used in short courses rather than a continuous basis. NSAIDs can also be given topically.- Intraarticular corticosteroid injections produce short-term improvement when there is a painful joint effusion; systemic corticosteroids are not used.
Surgical	3	<ul style="list-style-type: none">- Arthroscopy: look at the damages ,if there any problem with the tendon it can repair it.- Osteotomy: Bone cutting to Correct bone Deformity- Arthroplasty :Total joint replacement has transformed the management of severe symptomatic OA- Fusion and joint Lavage: Joint washing enables ridding the enzymes that are responsible for damage to the cartilage- Stem cell therapy? not approved

► **Notes:**

Treatment should focus on the symptoms and disability, not the radiological appearances

MCQs

1)50 y\o male diabetic patient,presents with an acutely painful right knee for 5 days.He denied history of trauma.on examination his temperature is 37.8 C with a hot swollen right knee.investigations revealed a white cell count of 12.6×10^9 and a knee x ray shows reduced joint space.which one of the following could be the most appropriate step?

- arthrocentesis
- Blood culture
- MRI of the right knee
- Request rheumatoid factor

2)A 45 y\o lady complains of gradual development of distal interphalangeal joints swelling and pain. She denied presence of oral ulcers, skin rash and morning stiffness. On examination the joints involved were 3rd and 4th DIP joints bilaterally. What is the most likely diagnosis?

- Gouty arthritis
- Rheumatoid arthritis
- Osteoarthritis
- SLE

3) What is the treatment of choice of osteoarthritis?

- Colchicine
- Paracetamol
- Prednisone
- Propranolol

4)80 y\o female came to the hospital with low back pain after carrying heavy bag. What is the most appropriate investigation you will order at the moment?

- MRI
- Bone scan
- X-ray for fracture
- Electrolytes levels

5)An otherwise healthy middle aged man with no prior medical history has had increase back pain and right hip pain for the past 10 years . the pain is worse at the end of the day . He has bony enlargement of the distal interphalangeal joints . A radiograph of the spine reveals the presence of prominent osteophytes involving the vertebral bodies .There is sclerosis and narrowing of the joint space at the right acetabulum seen on radiograph of the pelvis .which of the following pathological process is the most likely in the patient ?

- gout
- reiter's disease
- osteoarthritis
- rheumatoid arthritis

Answer key:

1 (A) | 2 (C) | 3(B) | 4 (C) | 5(C)