



Osteoarthritis

Objectives: No objectives .

Team Members: Abdulaziz alsalem+ Layla AlBreacan+ Trad Alwakeel+ Leena alwakeel

Team Leader: Haneen alsubki

Revised By: Yara Aldigi and Basel almeflh

Resources: 435 team + Davidson + doctor's slide

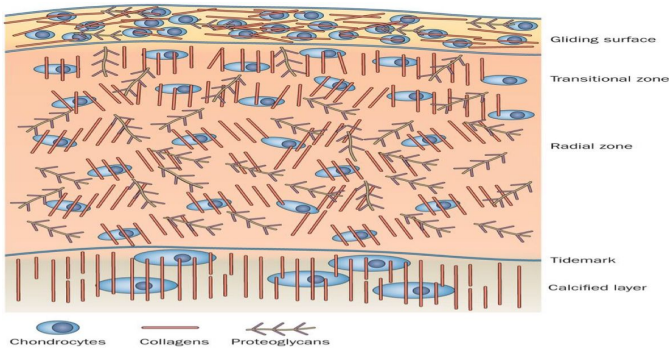
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Basic review of normal joint

Joint classification						
	Functional			structural depending on the material composing the joint .		
Type	Synarthroses -immovable -usually not affected by OA	Amphiarthrosis -slightly movable	Diarthroses -freely moveable	fibrous	cartilaginous	synovial - we will be talking about this type in the lecture
Example	Skull	symphysis pubis, vertebral	shoulder	Skull	symphysis pubis	knee, elbow, shoulder

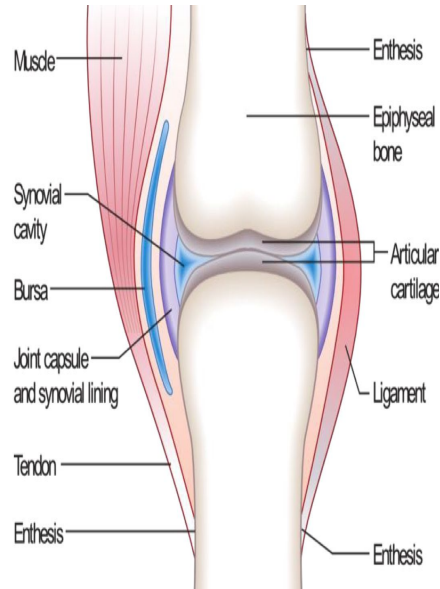
The normal articular surface of synovial joints

Articular cartilage aids the function of the joints (chondrocytes) surrounded by extracellular matrix includes: **proteoglycans** and **collagen**. The cartilage facilitates joint function and protects the underlying subchondral bone by: distributing large loads, maintaining low contact stresses- if the surface wasn't smooth the body weight on the weight bearing joints will be causing pain- , and reducing friction at the joint. As you can see in the picture below the distribution pattern of the matrix differs in the different layers of the cartilage . being the largest amount in parallel pattern in the gliding surface

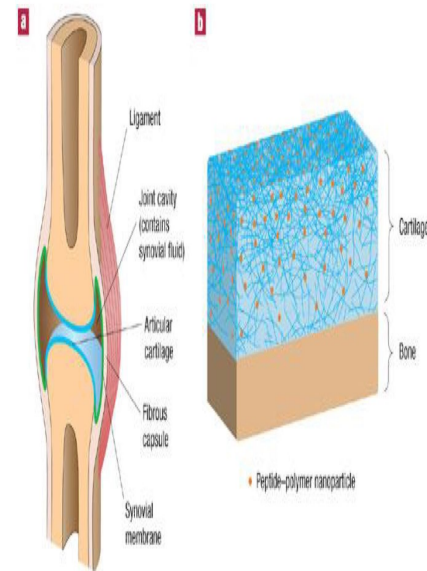


Synovial joint anatomy

synovial joint consists of:



Synovial cartilage



★ **Synovial Joints** consists of :

- articular joints
- subchondral the layer of bone below the cartilage bone
- synovial membrane
- joint capsule
- synovial fluid

★ **Synovial Fluid** is formed by synoviocytes.

- Synovial cells also manufacture **hyaluronic acid**(also known as hyaluronate): a glycosaminoglycan that is the major non cellular component of synovial fluid.

Its Functions:

- 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

★ **Cartilage homeostasis:**

How can the cartilage maintain its integrity? balancing between synthesis and degradation.

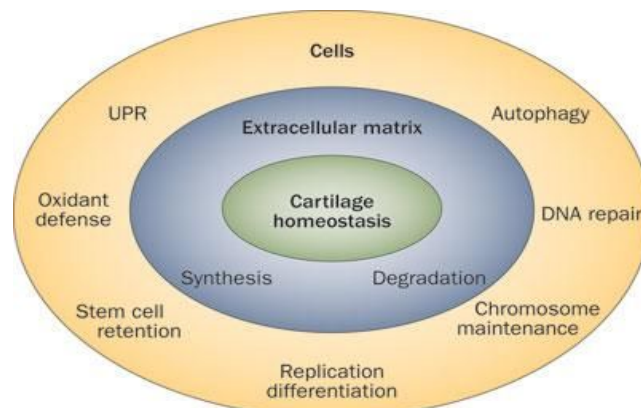
Cartilage and joints are avascular structure, so how can they regenerate? by a its cartilage homeostasis mechanism.

there's a balance between synthesis and degradation. maintained by stem cells and (in a specific concentration) and autophagy and DNA repair.

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.



Osteoarthritis

What is it:

-Heterogeneous group of conditions resulting in common histopathologic and radiologic changes involving Entire joint, including:

- the articular cartilage
- the subchondral bone
- 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 80-90% of individuals older than 65 years have evidence of radiographic osteoarthritis. although only a proportion of these have symptoms.
- the prevalence of osteoarthritis is higher among women than among men. **genetics, mechanical or biochemical reasons**.
- Interethnic differences in the prevalence of osteoarthritis have been noted. **Chinese, US indians**.

Involved Joints:

- **most commonly involved joints:**
- **usually in the weight bearing joints but non weight bearing joints may also be affected.**
- **type of joint affected is important because it can help us differentiate it from other diseases.**
- the weight-bearing joints: (knees, hips, feet, cervical and lumbosacral spine).
- non weight-bearing joints: **(DIP)¹ unlike RA which is sparing for DIP, (PIP)², and (CMC)³ joint of hands**
- first metatarsophalangeal joint (MTP) of the foot

Etiology:

- **Risk factors:** Age, obesity more weight in joints, trauma, hypogonadism, muscle weakness, repetitive use, infection (**septic arthritis**), crystal deposition, acromegaly, and previous inflammatory arthritis (burnt-out rheumatoid arthritis) **Genetics**
- **Heritable metabolic causes:** alkaptonuria, hemochromatosis, and wilson disease
- **Hemoglobinopathies:** sickle cell disease and thalassemia
- **Neuropathic disorders leading to a charcot joint:** syringomyelia, tabes dorsalis, and **uncontrolled diabetes, syphilis**
- **Underlying morphologic Risk factors:** congenital hip dislocation and slipped femoral capital epiphysis
- **Disorders of bone:** paget disease and avascular necrosis
- **Previous surgical procedures:** meniscectomy

¹ Distal InterPhalangeal joints

² Proximal InterPhalangeal joints

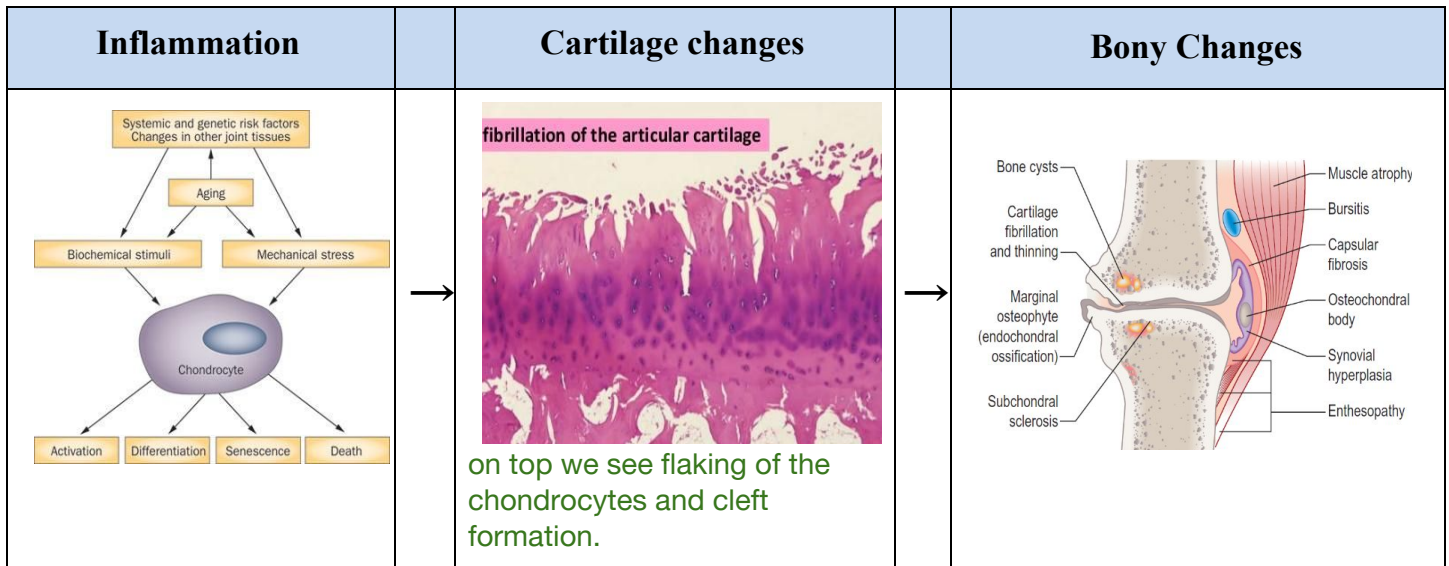
³ CarpoMetaCarpal Joints

Pathology & and Pathogenesis

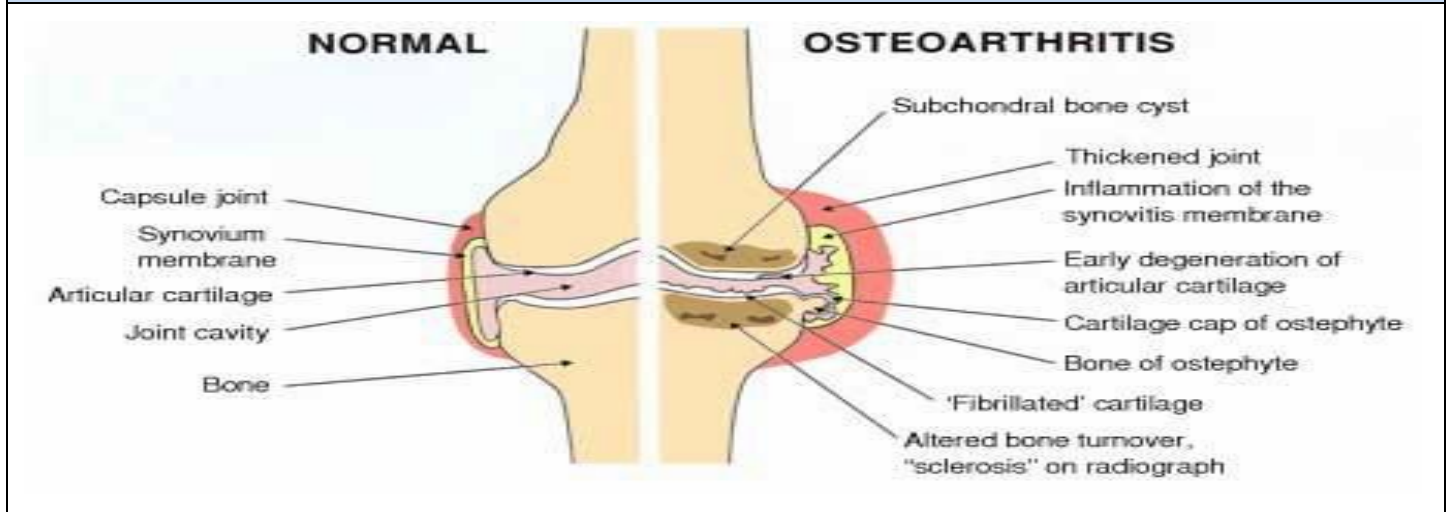
inflammation	1	Inflammation occurs as cytokines and metalloproteinases ⁴ are released into the joint. these agents are involved in the excessive matrix degradation that characterizes cartilage degeneration on OA. inflammation is present but it's not the initiating event like in rheumatoid arthritis. it comes later in the course of disease.
Cartilage changes	2	Swelling of the cartilage usually occurs the level of proteoglycans eventually drops very low, the cartilage softens and lose elasticity and compromises joint surface integrity
	3	Flaking and fibrillations zigzag asymmetrical surface(vertical clefts) develop along on the surface of an osteoarthritic joint. <ul style="list-style-type: none"> ● Over time, the loss of cartilage results in loss of joint space in the medial compartment of knee joint and that is the first sign recognized on x-ray . ● a greater loss of joint space occurs at those areas experiencing the highest loads
Bone Changes	4	bone denuded of its protective cartilage continues to articulate with the opposing surface the two bones will glide over each other without any protection! <ul style="list-style-type: none"> ● eventually, the increasing stresses exceed the biochemical yield strength of the bone
	5	The subchondral bone responds with vascular invasion and increased cellularity, becoming thickened and dense (a process known as eburnation “sclerosis”) at areas of pressure .
	6	subchondral bone undergo cystic degeneration . <ul style="list-style-type: none"> ● Osteoarthritic cysts are also referred to as subchondral cysts, pseudocysts, geodes, or Egger cysts if it involved the acetabulum hip. ● Osteoarthritic cysts may range from 2 to 20 mm in diameter
Joint changes	7	vascularization of subchondral marrow
	8	osseous metaplasia of synovial connective tissue Ossification happens .
	9	ossifying cartilaginous protrusion lead to irregular outgrowth of new bone (osteophyte)
	10	fragmentation of these osteophytes or of the articular cartilage itself results in the presence of intra-articular loose bodies (joint mice).

In brief : Tissue macrophages within the synovial membrane can be stimulated due to variety of reasons like trauma , deformities , obesity which will cause the release of cytokines . which will stimulate synoviocytes to secrete proteases and break protein cartilage . will also stimulate osteocytes as a repair mechanism to prevent the damage “osteoblasts” causing subchondral sclerosis which will appear as osteophytes .

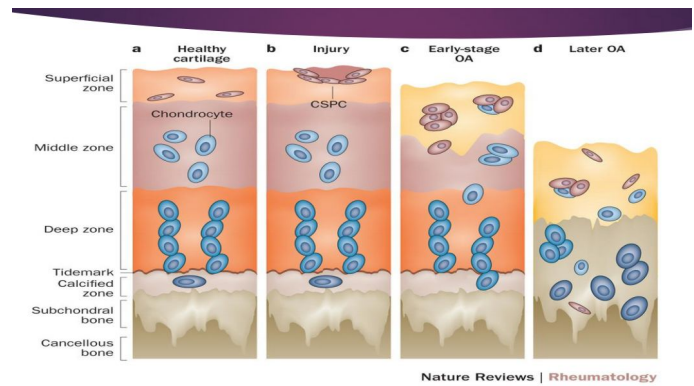
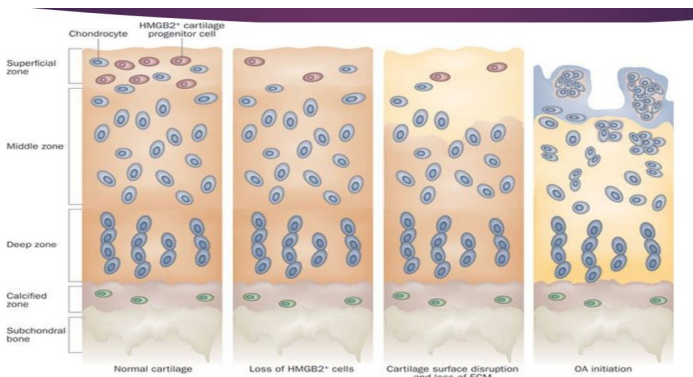
⁴ Metalloproteinases, e.g. stromelysin and collagenase, secreted by chondrocytes degrade collagen and proteoglycans.



Joint Changes



★ Cartilage disruption



Pic1:HMGB2(Highly mobility B2 cells) are responsible for production of chondrocytes. when their numbers go less this leads to loss of cartilage.

Pic2:CSPC (CARTILAGE SPECIFIC PRECURSOR CELL) distribution in normal articular cartilage and in OA. what happens in OA is that CSPC starts clumping and losses its function.

★ **Osteoarthritis progression:**

- **Stage 1:** breakdown of the cartilage matrix occurs
- **Stage 2:** involves the fibrillation and erosion of the cartilage surface
- **Stage 3:** a chronic inflammatory response in the synovium

Further progression: the above events alter the joint architecture, compensatory bone overgrowth occurs . joint architecture is changed , mechanical and inflammatory stress occurs on the articular surfaces , the disease progresses unchecked .

★ **Clinical Features:**

Presenting Symptoms: The main presenting symptoms are:

- joint pain and functional restriction in a patient over the age of 45, but more often over the age 60
- 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 morning stiffness(Less than 30 minutes).

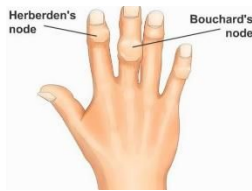
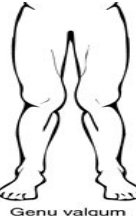
On Examination you will see :

- periarticular tenderness
- limited joint movement
- muscle wasting of surrounding muscle
- Crepitus (grating) is a common finding⁵
- Synovitis (mild or absent)



-the picture shows bowing of the left leg and curve appearance. the bottom X-ray shows complete loss of joint space. (dark area=joint) thus increasing the stress and articulation in the joint.

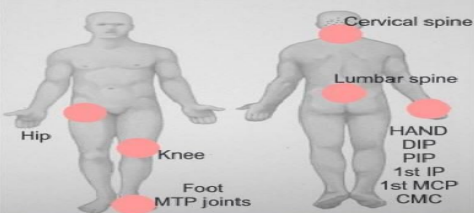


Deformity and Bony enlargement of the joints:

<p>Heberden's nodes are bony swellings at the(DIPJs)⁶</p> 	<p>Bouchard's nodes are bony swellings at the IPJs</p>	<p>varus deformity resulting from marked medial tibiofemoral osteoarthritis (bone twist is toward the center of the body)</p>	 <p>Valgus deformity Less commonly (bone twist is away from the center of the body)</p>
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⁵ also it is Palpable, sometimes audible when bending the joint, coarse crepitus due to rough articular surfaces.

⁶ distal interphalangeal joints

★ Subtypes of Primary Osteoarthritis

Three Subtypes:		
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 MTP⁷, hip, and intervertebral(spondylosis). <p>Osteoarthritis - Anatomical Distribution</p>  <p>hand joints are most commonly affected in OA. can be symmetrical and lacks signs of acute inflammation.</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. • Radiologically, there is marked osteolysis. Destructive phases are followed by phases of remodelling.  <p>peripheral erosion in the PIP joint. MCP joints are spared and that another way to differentiate it from rheumatoid arthritis.</p>	<p>is a condition where the cartilage on the undersurface of the patella deteriorates and softens.</p> 

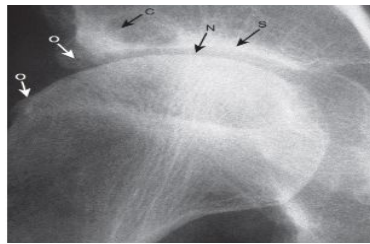
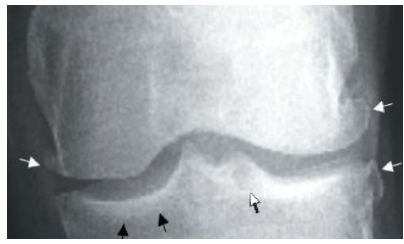
★ Differential Diagnosis

- 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 DIP may mimic OA.
- Septic arthritis or post infectious 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

⁷ metatarsophalangeal joint

★ Investigations

- X-rays is the most accurate **DIAGNOSTIC** test : are only abnormal in advanced disease and show narrowing of the joint space (resulting from loss of cartilage), osteophytes, subchondral sclerosis and cyst formation. **plain radiography is goldstone for initial workup.**
- MRI demonstrates **early** cartilage changes. (It is not necessary for most patients with suggestive symptoms and typical plain X-ray features).
- 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.
- CT scan and ultrasonography
- bone Scintigraphy if we suspect infection
- arthrocentesis aspiration of synovial fluid to look for inflammation, usually WBC count is less (up to 200) compared to inflammatory arthritis (thousands). in infections it can be several thousands WBCs. arthrocentesis also helps to look for crystals.

Radiographic Changes	
	
<p>X ray of hip showing changes of osteoarthritis. Note the superior joint space narrowing (N), subchondral sclerosis (S), marginal osteophytes (white arrows) and Egger cysts (C).</p>	<p>X ray of knee joint affected by OA, showing osteophytes at joint margin (white arrows), subchondral sclerosis (black arrows) and subchondral cyst (open arrow).</p>

★ **Management** treating inflammatory arthritis is easier than treating OA.

Non Pharmacologic	1	<ul style="list-style-type: none"> • Lifestyle modification, physical and rehab therapy; Obese patients should be encouraged to lose weight, particularly if weight-bearing joints are affected. • Physical measures are the keystone of OA treatment.⁸
Pharmacotherapy	2	<p>Medication:</p> <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 be given topically. • Intraarticular corticosteroid injections produce short-term improvement when there is a painful joint effusion; systemic corticosteroids are not used. • muscle relaxants, injectable hyaluronic acid.
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

How to differentiate between osteoarthritis and rheumatoid arthritis?

- Rheumatoid arthritis presents with inflammatory signs like morning stiffness. (in OA it lasts for 5-15 mins and then gets better, while in RA it lasts longer.
- DIP joints are spared in RA but affected in OA
- CMC joints are not affected in OA unless patient is in a profession where they're exhausting them.
- Wrist joints and elbow joints are usually not affected in OA.
- MTP joints are not affected by OA
- heat and warmth with significant synovial swelling in RA. (can be seen in OA so we confirm with synovial fluid)
- inflammatory pain is worse at rest, relieve by activity (RA), where as OA pain gets better with rest and worse with activity.

⁸ Local strengthening and aerobic exercises improve local muscle strength, improve the mobility of weight-bearing joints and improve general aerobic fitness

Summary

★ **Osteoarthritis : It characterized by progressive destruction and loss of articular cartilage with an accompanying periarticular bone response** , affects Entire joint, including:

- the articular cartilage
- the subchondral bone
- the synovium

★ **Involved joints :**

- the weight-bearing joints: (knees, hips, feet, cervical and lumbosacral spine).
- non weight-bearing joints: (DIP), (PIP), and (CMC) joint of hands

★ **Risk factor of osteoarthritis:** aging , obesity , trauma.

★ **Pathogenesis :**

-Inflammation : occurs as cytokines and metalloproteinase are released into the joint.

-Cartilage:

- Swelling caused by low proteoglycans
- fibrillations(vertical clefts) develop

- Bone:

- Eburnation: vascular invasion and increased cellularity
- Subchondral cysts
- Osteophyte : irregular outgrowth of new bone

-Synovium :

- Metaplasia of synovial

★ **Investigation :**

- X-rays
- MRI
- Laboratory tests
- CT scan and ultrasonography
- bone Scintigraphy arthrocentesis

★ **Management :**

- Non pharmacological : Lifestyle modification , physical and rehab therapy
- Pharmacotherapy:Arthroscopy , osteotomy , Arthroplasty , Fusion and joint lavage stem cell therapy

Questions

Q1: In osteoarthritis, which one of these joints is most likely affected?

- A. DIP joint
- B. MTP joints
- C. CMC joins
- D. Wrist joint

Q2: A 40 Y.O lady complaining of morning stiffness lasting for 1 hour and relieved by movement, her MCP joints are swollen and red. Which one of the following is the diagnosis?

- A. osteoarthritis.
- B. Erosive osteoarthritis.
- C. Rheumatoid arthritis.
- D. gouty arthritis.

Q3: Which one of these findings are most compatible with osteoarthritis?

- A. osteophytes.
- B. crystals formation.
- C. swan neck deformity
- D. ulnar deviation

Q4: A 55 year old patient presented with pain, stiffness and swelling in her DIP joints. you're considering osteoarthritis. What is the best initial investigation to do?

- A. CT
- B. MRI
- C. X-ray
- D. Lab tests

Q5: Which of the following can be considered as a risk factor for osteoarthritis ?

- A. A 42 y/o male patient with BMI = 25
- B. A 51 y/o female with osteoporosis
- C. A 23 y/o male with a history of greenstick fracture
- D. A 40 y/o male with enlarged hands, feet and facial features

Q6 : How can an X-ray help a healthcare provider to diagnose osteoarthritis?

- A. It can show cartilage loss
- B. It can show bone damage
- C. It can show bone spurs
- D. All of the above

Q7 : A 64 y/o female patient presented to the hospital complaining of joints pain and stiffness after rest. The examination showed muscle wasting and crepitus. after investigations, she was diagnosed with osteoarthritis. Which of the following is the best initial management plan for relieving her pain ?

- A. Lifestyle modification
- B. Paracetamol
- C. Arthroscopy
- D. Intrarticular corticosteroids injection

Q8 : Which of the following bone deformities can be seen in DIP ?

- A. Bouchard's node
- B. Heberdens's node
- C. Varus deformity
- D. Valgus deformity

Q9 : An otherwise healthy 44-year-old man with no prior medical history has had increasing back pain and right hip pain for the past decade. The pain is worse at the end of the day. On physical examination 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 with narrowing of the joint space at the right acetabulum seen on a radiograph of the pelvis. Which of the following diseases is he most likely to have?

- A. Rheumatoid Arthritis
- B. Osteomyelitis
- C. Osteoarthritis
- D. Gout

Answers :

1)A , 2)C , 3)A , 4)C , 5)D , 6)D 7)B , 8)B , 9)B