Lecture 16

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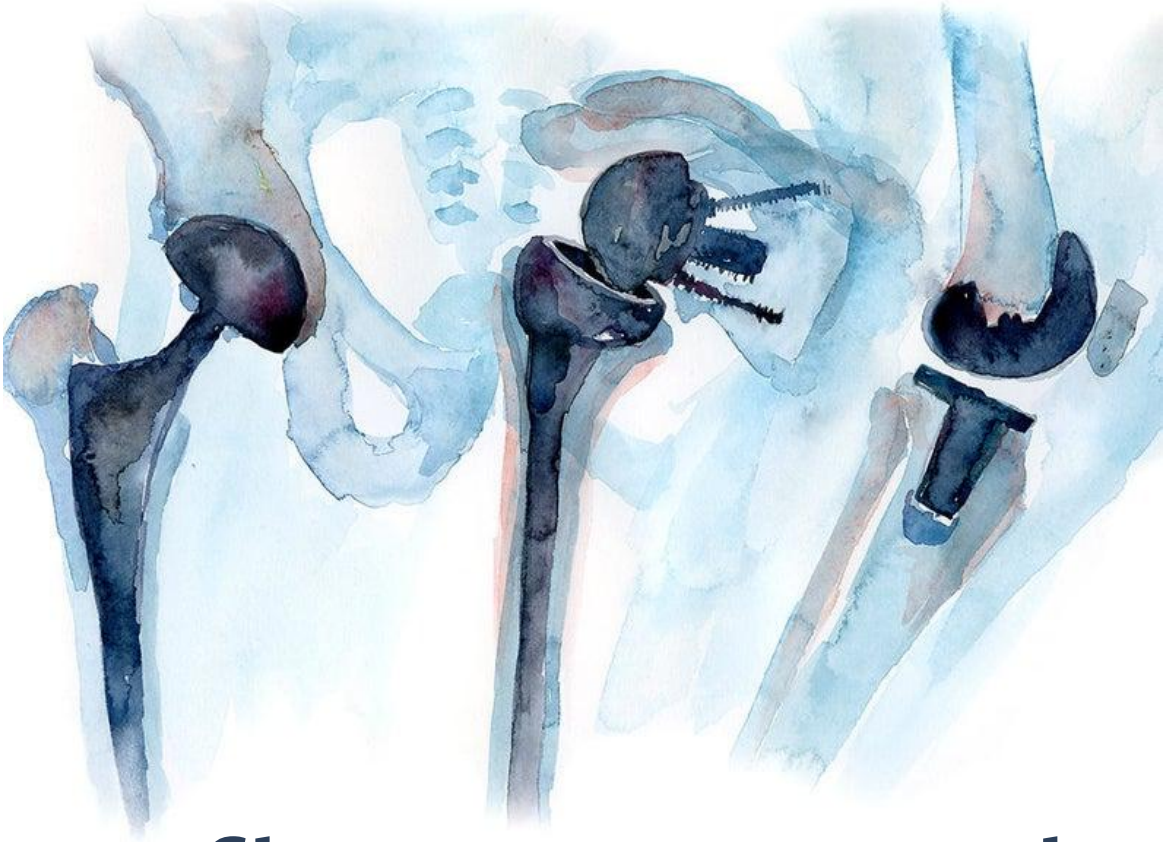


Inflammatory and Degenerative Joint Disorders

Dr. Ahmad Bin Nasser

Objectives:

➔ No Objectives were provided for this lecture.

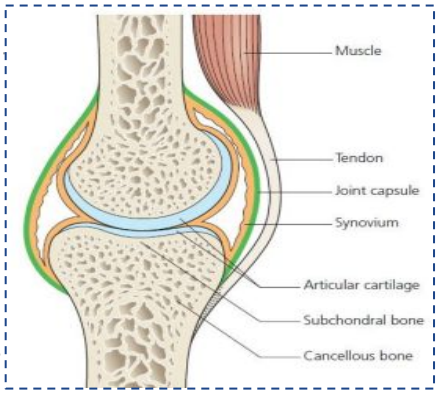
Color Index:

Original text | Doctor's notes | Text book
Important | Golden notes | Extra

Synovial Joints:

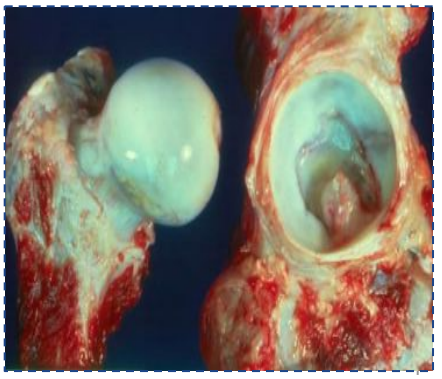
Introduction

- Synovial joints are the most common type of joint in the body. These joints are termed diarthroses, meaning they are freely mobile.
- The joint is made up of different structures that act together to move, lubricate, nourish and stabilize the joint.



❖ Components of Synovial Joints:

1- Articular Cartilage



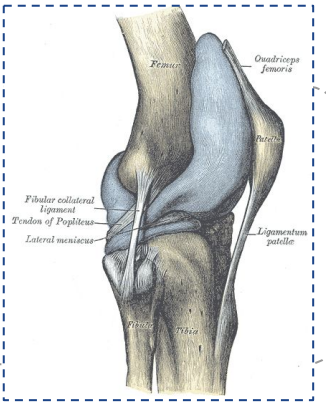
- Hyaline cartilage The hyaline cartilage is not replaceable, if damaged it's replaced by fibrocartilage.
- Viscoelastic material with variable load-bearing properties
- Decreases joint friction
- Avascular and aneural No nerve ending or vessels
- Chondrocytes have little capacity for cell division in vivo
- Direct damage to the articular surface is poorly repaired, or repaired only with fibrocartilage.
- Fibrocartilage has inferior biomechanical properties than hyaline.
- 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. [1]

Cartilage composition	
Water (60-80% net weight)	• Pumped in and out of cartilage depending on load. • Contributes to lubrication and nutrition.
Collagen (10-20% net weight)	• Secreted by chondrocytes • Mostly type-II collagen (90%) • Confers tensile strength to cartilage
Proteoglycans (10-15% net weight)	• Secreted by chondrocytes • Composed of GAG (aggrecan, chondroitin and keratin sulfate). • Negatively charged proteins hold water within the matrix • Provides compressive strength
Chondrocytes (5% net weight)	• The only cell type in cartilage.

1- Mainly the loss of cartilage is progressive but sometimes the loss happens very quickly like in septic arthritis when the bacteria and the enzymes released by bacteria and phagocytosis cause direct damage to the joint.

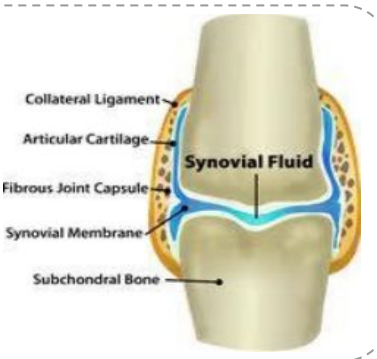
Synovial Joints:

2- Capsule and Ligaments



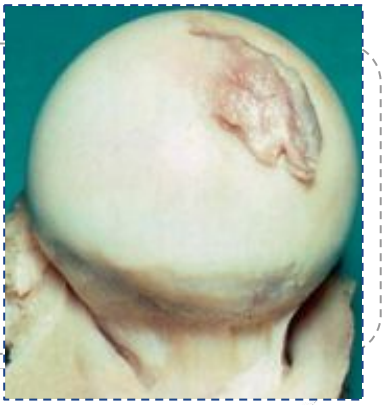
- Fibrous structures with tough condensations on its surface (ligaments).
- Together with the overlying muscles, help to provide with stability.
- Ligaments provide stability to the joint when it's torn we lose a major stabilizing factor.

3- Synovium

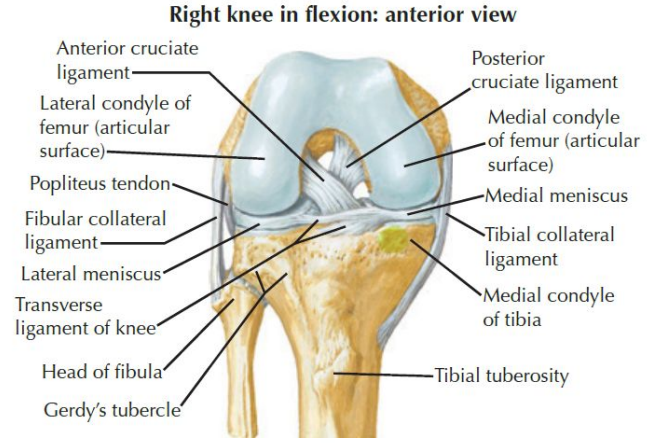


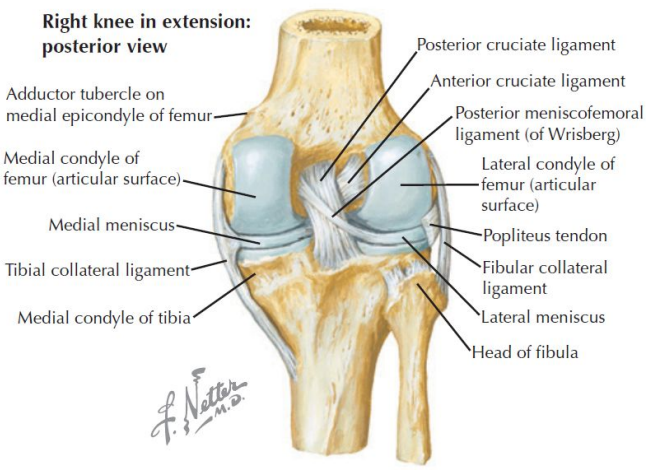
- Thin membrane
- Richly supplied with blood vessels, lymphatic and nerves
- Target tissue in joint infections and autoimmune disorders such as rheumatoid arthritis
- Provides a nonadherent covering for the articular surfaces

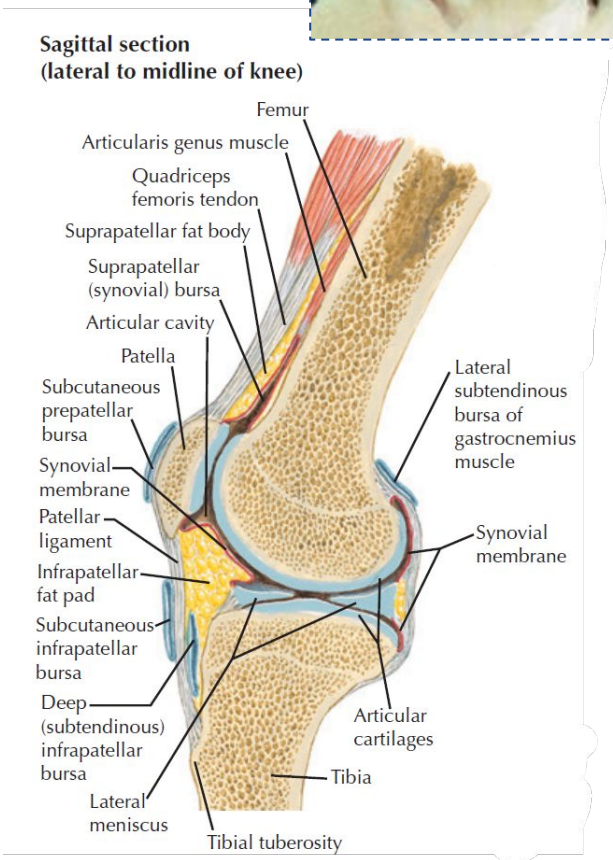
4- Synovial Fluid



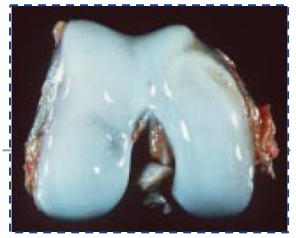
- Synovial fluid nourishes the avascular articular cartilage.
- Plays an important role in reducing friction during movement.
- Has slight adhesive properties which assist in maintaining joint stability.
- The volume remains fairly constant, regardless of movement.
- When a joint is injured fluid increases (joint effusion)







Osteoarthritis:



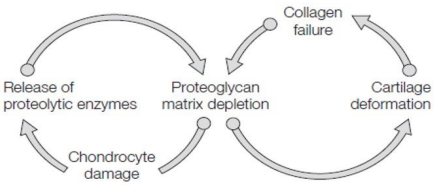
Prevalence

- 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).
- More joints are affected in women than in men.
- Common in our community especially knees (presents earlier than in West)
- About 90% of those over 40 have asymptomatic degeneration of weight bearing joints
- Commonest joints are knee, hip , Cervical spine & Lumbar spine, 1st Carpometacarpal, 1st Metatarsophalangeal and Interphalangeal joints

Pathology: 5 Cardinal Features

1- Progressive cartilage destruction

- Increase in water content which leads to swelling and softening of the cartilage
- This will lead later on to the depletion of proteoglycans
- Chondrocytes will die which will cause synovitis
  - ➔ This will release proteolytic enzymes → collagen disruption
- Fibrillation on weight bearing surfaces



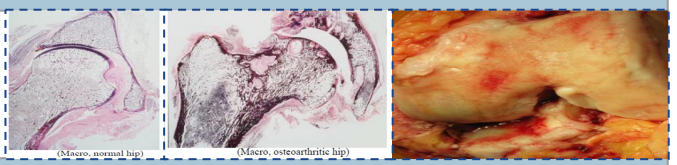
2- Subarticular cyst formation cyst (doesn't appear in X-rays except in the hip)

Arises from:

- Local areas of osteonecrosis
- Forceful pumping of synovial fluid through subchondral bony cracks

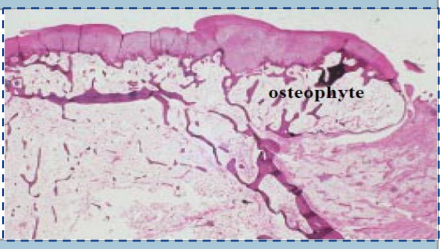
3- Sclerosis of the surrounding bone (seen in all X-rays)

- Bone becomes exposed (may be polished or burnished to ivory-like smoothness (eburnation)



4- Osteophyte formation (seen in all X-rays)

- Proliferation and remodeling of the cartilage at the edges
- This will be followed by endochondral ossification
  - ➔ The joint try to increase the surface area to distribute the stress so it forms a cartilage at the periphery of the joint which underwent ossification and became a bone



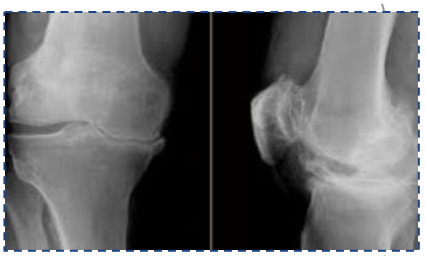
5- Capsular Fibrosis

- Marked vascularity and venous congestion of the subchondral bone (causes pain)
- The capsule and synovium are often thickened but cellular activity is slight
- Progressive bone erosion → **BONE COLLAPSE**
- Fragmented osteophyte → **LOOSE BODIES**
- Loss of height and ligamentous laxity → **MALALIGNMENT**

Osteoarthritis:

❖ Primary Idiopathic Osteoarthritis

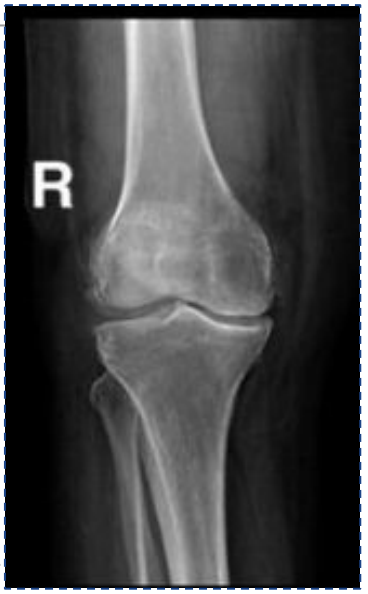
Definition



Chronic disorder, characterized by:

- Progresssive softening and disintegration of articular cartilage
- New growth of cartilage and bone at the joint margins (osteophytes)
  - ➔ Which leads to the loss of the congruency of the articular surface
- Subchondral bone sclerosis and cyst formation
- Mild synovitis and capsular fibrosis
  - ➔ Synovium becomes inflamed and produces more fluid as a protective mechanism [2]

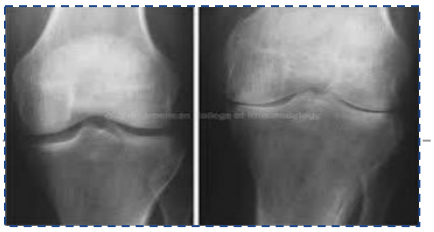
Characteristics



- Asymmetrically distributed, often localized to only one part of a joint
  - ➔ The place where the highest pressure is located at. On the hip it will affect the top more, if it's fully damaged search for another reason
- Often associated with abnormal loading
  - ➔ Usually causes of OA is more mechanical (weight and loading); whatever part is loaded more will have early osteoarthritis like. In obese patients patello-femoral part will be affected.
- Unaccompanied by systemic illness
- Not primarily an inflammatory disorder although there are sometimes local signs of inflammation
- Not purely degenerative; dynamic phenomenon; it shows features of both destruction and repair

Etiology

1. **Disparity between the mechanical stress** to which the articular cartilage is exposed
2. Increased mechanical stress in some part of the articular surface
3. Varus deformity of the knee (that's why the medial side is usually only affected)
4. Obesity (hips and knees take 3-4x body weight with each step)
5. Increase in frequency with age
6. Family Hx
7. More of a process than a disease

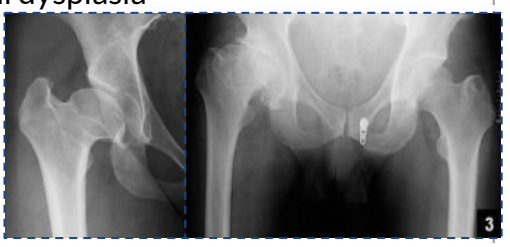


❖ Secondary Osteoarthritis

Etiology

Main difference is total joint involvement (not only medial) ↑

1. **Trauma**: malunion, osteochondral, sport injury, dislocation
2. **Congenital/developmental:** hip dysplasia, multiple epiphyseal dysplasia
3. **Infection**
4. **Necrosis**: Perthes disease, osteonecrosis, steroids
5. **Endocrine**: DM, acromegaly
6. **Hematologic**: Sickle cell disease, hemophilia
7. **Neuropathic**: DM, tabes dorsalis
8. **Inflammatory**: Rheumatoid arthritis, SLE, Reiter's syndrome
9. **Metabolic**: crystalline deposition disease (gout, CPPD), Paget's disease



Osteoarthritis:

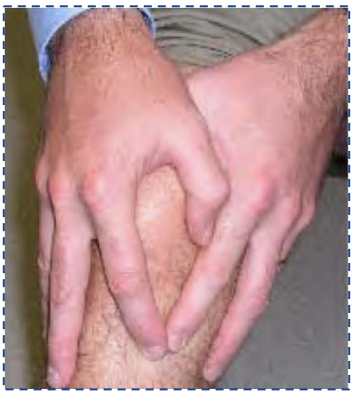
Clinical Features

- Intermittent course, with periods of remission lasting for months
- One or two of the weight-bearing joints(hip or knee) might be affected
- In our community patients having degenerative knee will have degenerative lumbosacral & cervical spine. So, in a knee surgery check spine and vice versa
- Pain of OA starts in one of two ways either suddenly after an event (I did something I'm not used to (Marathon) or progressively (day after day)

Symptoms

1- Pain (fluctuating and not continuous especially during the extremities of motion)

- Localized or rarely referred to distant site (e.g: pain in the knee from hip osteoarthritis)
- Insidious in onset
- Aggravated by exertion and relieved by rest
- In advanced stages there will be night pain or pain at rest



Causes of Pain:

- Bone pressure due to vascular congestion and intraosseous hypertension; **most important**.
- Mild synovial inflammation
- Capsular fibrosis with pain on stretching the shrunken tissue.
- Muscular fatigue [1]

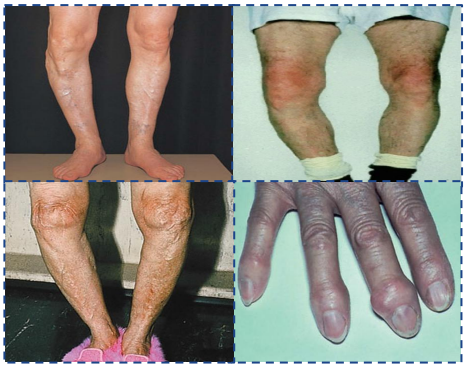
2- Stiffness

- Initially after periods of inactivity, but later on it will be constant and progressive
- The worst advice you give the patient is to tell him/her not to use the affected joint, as a result the patient will develop stiffness, weight gain, muscle loss and the symptoms will be worse!

3- Loss of Function

Signs

- Swelling: Intermittent = effusions, Continuous = large osteophytes
- Deformity → mal-alignment (Primary OA = Varus)
- Tenderness
- Limited range of movement.
- Crepitus
  - ➔ Joint crepitation is not an indication of OA unless it is accompanied by pain or swelling, or limited ROM
- Instability
  - ➔ Due to loss of cartilage and bone, asymmetrical capsular contracture and/or muscle weakness.



1- Might be muscle pain, the joint is weak so the muscles contract for a long time, causing pain and fatigue. So the patient avoids fully flexing and extending , so they give up and don't try to move that's why physiotherapy is important, it also increases the capacity of the muscle and strengthens it to support the joint, so more movement before feeling pain

Osteoarthritis:

❖ Imaging:[1]

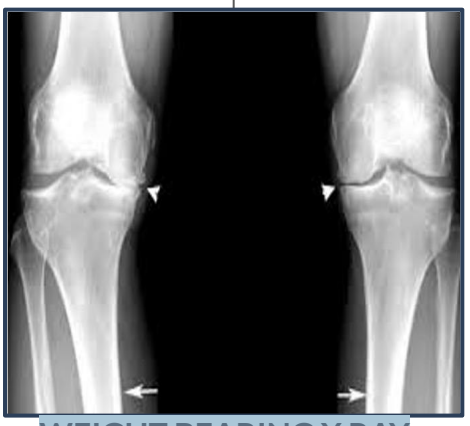
Used to confirm the diagnosis and rule out other causes (NOT to treat)

Early

- Asymmetrical loss of cartilage

(narrowing of the ('joint space')

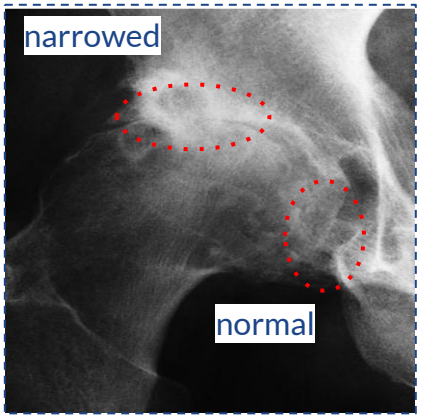
- Subchondral bone sclerosis
- Cysts close to the articular surface
- Osteophytes at the margin of the joint

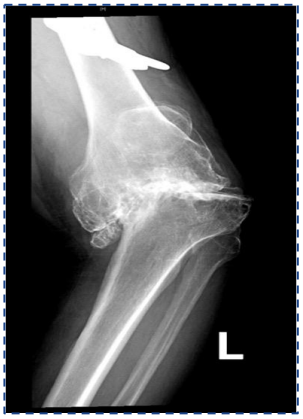


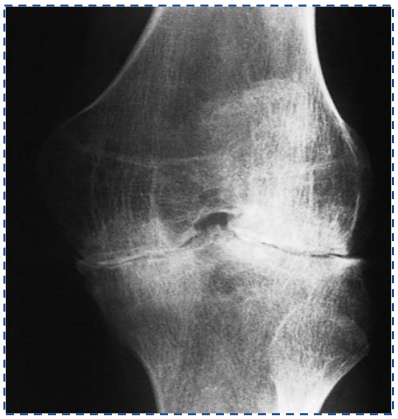
WEIGHT BEARING X RAY

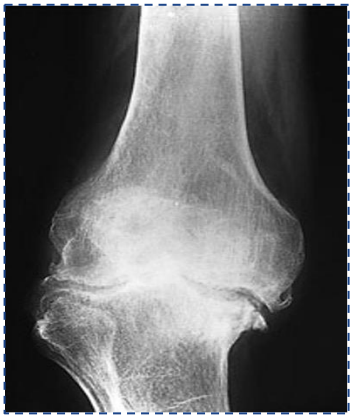
Late

- Malalignment
- Joint subluxation
- Bone loss
- Loose bodies
- Signs of other disorders
- Symmetric narrowing in inflammatory OA e.g. Rheumatoid Arthritis







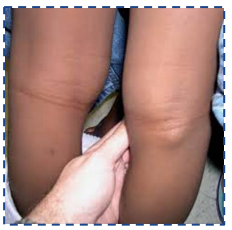


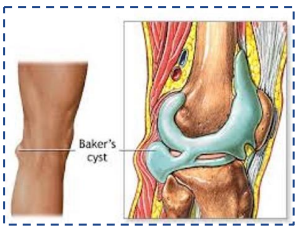
Grade 4 is bone on bone , while grade 1 is only seen on a MRI, X-Ray is normal

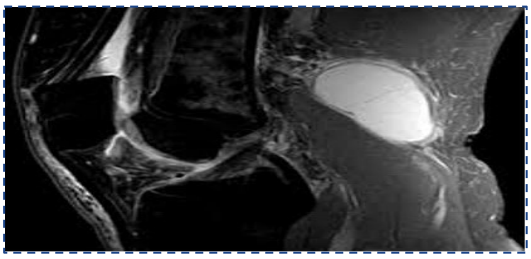
❖ Complications:

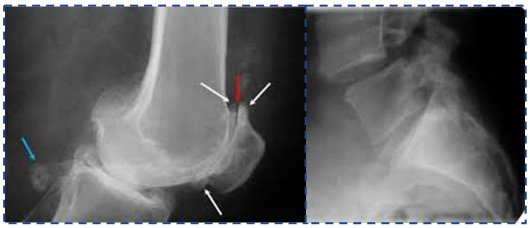
Complications

- Capsular herniation: knee OA; marked effusion and herniation of the posterior capsule (Baker's cyst)
  - ➔ Very common, synovial fluid finds a weak area and goes there
- Loose bodies
  - ➔ Arthroscopy is done if there are mechanical symptoms (Loose bodies)
  - ➔ Can't move because pebble like structures are preventing it from moving
- Rotator cuff dysfunction in acromioclavicular joint OA
- Spinal Stenosis
- Spondylolisthesis
  - ➔ Severe segmental disability; at L4/L5









1- Imaging is X-Ray and weight bearing (standing) X-Ray Standing x-ray is always better. If you are searching for thinning of cartilage do weight bearing x-ray to indirectly see the cartilage (you will see the space occupied by the cartilage). MRI is indicated if x-ray is normal.

Osteoarthritis:

Management

Early treatment:

- Maintain movement and muscle strength
- Protect the joint from 'overload'
- Relieve pain: Analgesics
- Modify daily activities (If obese lose weight, avoid carrying heavy weight, strengthen muscles)

Conservative Management

1- Maintain movement and muscle strength by physiotherapy

This will help in:

- Pain relief through massage or application of warmth
- Prevent contractures
- Muscle strengthening
- Range of motion

2- Load reduction: Never advice the patient to stop moving or to be immboile

- Weight reduction (if the patient obese)
- Shock-absorbing shoes.
- Walking stick (carry the stick using the hand on the unaffected side)
- Unloading brace. (if the knee is in varus, it pushes knee to valgus)
  - ➔ Failed to show any benefits.
- Not bearing heavy object.

3- Modify activities and Sitting Habits

- Modify activities and avoid others (pray using a chair for example)
- Change sitting habits (avoid sitting on the floor for example)

4- Medication: pain relief only as needed

- **Oral**: paracetamol, NSAIDs, muscle relaxants,narcotics, supplements and herbs.
  - ➔ Supplements has no effect on the disease (placebo effect)
- **Injections**: (Local) **not** recommended in general.
  1- Steroids: it is used for patients who can't use NSAIDs either for kidney disease or peptic ulcer
  2- Hyaluronic acid injection (oil injection/ filler): the goal is to relief pain it might be an option for patients with early disease or those who cant take medications
  3- Plasma: take blood from pt and separate plasma limited effect on pain relief
  4- Stem cells: no proof of effectiveness

  Remember injection is not a part of standard treatment of OA

Osteoarthritis:

Management

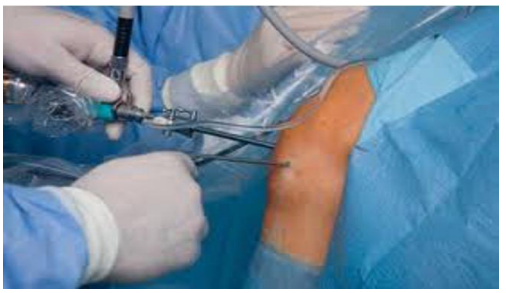
- If conservative treatment fails and the patient is in pain, we can proceed to surgical management.
- We shouldn't proceed to surgical management based on radiological features alone even if there's severe OA. The only exception to this is if there's extensive bone resorption along with severe malalignment.

Surgical Management

1- Joint Debridement (Arthroscopy): Honeymoon surgery

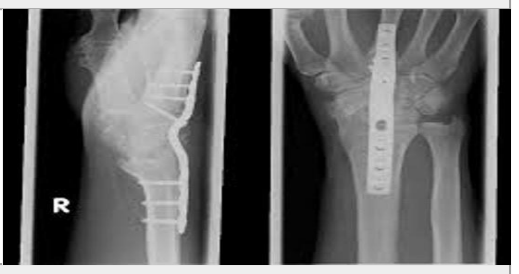
Not common procedure, done under GA, done commonly in private hospital, not indicated and will not correct the disease, used only if there is indication for it (blocking or latching of the joint).

- Removal of loose bodies
- Removal of meniscal or labral tears
- For mechanical symptoms



2- Arthrodesis

- Transfer from painful stiff into painless stiff joint (leads to loss of motion)
- Small joints; hand (wrist joint), foot and spine (1st CMC)

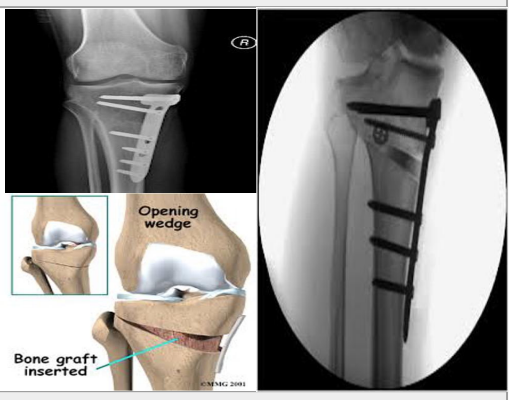


3- Corrective Osteotomy

- Realign axis and distribute weight
- We offload the arthritic part, and we load the uninjured part
- Knee and hip are common sites
- Candidate: Young, active, mild OA

Minor role in pain relief

- Vascular decompression of the subchondral bone
- Redistribution of loading forces towards less damaged parts of the joint

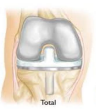


4- Joint Replacement (Arthroplasty)

- Nowadays then procedure of choice for advanced OA

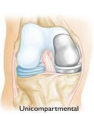
Total joint replacement

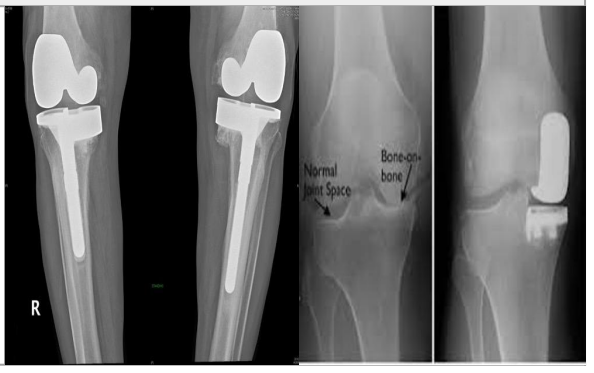
- Knee, hip, shoulder, ankle and elbow
- **Candidate:** Painful, deformed stiff joint, old patient



Partial joint replacement

- Knee
- **Candidate:** Same as for osteotomy (Young, active, mild OA)





Extra:

Degenerative Arthritis: Osteoarthritis

- see Family Medicine, FM42

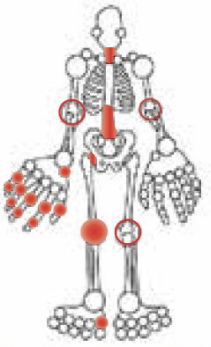
Definition
- progressive deterioration of articular cartilage and surrounding joint structures caused by genetic, metabolic, biochemical, and biomechanical factors with secondary components of inflammation

Classification (Based on Etiology)
- primary (idiopathic)
  - most common, unknown etiology
- secondary
  - post-traumatic or mechanical
  - post-inflammatory (e.g. RA) or post-infectious
  - heritable skeletal disorders (e.g. scoliosis)
  - endocrine disorders (e.g. acromegaly, hyperparathyroidism, hypothyroidism)
  - metabolic disorders (e.g. gout, pseudogout, hemochromatosis, Wilson's disease, ochronosis)
  - neuropathic (e.g. Charcot joints), atypical joint trauma due to peripheral neuropathy (e.g. DM, syphilis)
  - avascular necrosis (AVN)
  - other (e.g. congenital malformation)

Pathophysiology
- the process appears to be initiated by abnormalities in biomechanical forces and/or, less often, in cartilage
- elevated production of pro-inflammatory cytokines is important in OA progression
- tissue catabolism > repair
- contributing factors (mechanisms unknown): genetics, alignment (bow-legged, knock-kneed), joint deformity (hip dysplasia), joint injury (meniscal or ligament tears), obesity, environmental, mechanical loading, age, and gender
- considered to be a systemic musculoskeletal disorder rather than a focal disorder of synovial joints

Epidemiology
- most common arthropathy (accounts for ~75% of all arthritis)
- increased prevalence with increasing age (35% of 30 y/o, 85% of 80 y/o)

OA of MCP joints can be seen in hemochromatosis or CPPD-related disease (chondrocalcinosis)



- Hand (DIP, PIP, 1st CMC)
- Hip
- Knee
- 1st MTP
- L-spine (L4-L5, L5-S1)
- C-spine
- Uncommon: ankle, shoulder, elbow, MCP, rest of wrist

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Figure 3. Common sites of joint involvement in OA

Table 8. Synovial Fluid Analysis

Parameter	Normal	Non-Inflammatory	Inflammatory	Septic	Hemorrhagic
Colour	Pale yellow	Pale yellow	Pale yellow	Yellow to white	Red/brown
Clarity	Clear	Clear	Opaque	Opaque/purulent	Sanguinous
WBC/mm^3	<200	<2000	≥2000 (crystal induced arthritis – often much higher than 2000)	>50000	Variable
% PMN	<25%	<25%	≥50%	>75%	Variable
Culture/Gram Stain	–	–	–	Usually positive	–
Examples		Trauma OA Neuropathy Hypertrophic – arthropathy	Seropositive Seronegative Crystal arthropathies	*S. aureus* Gram negative GC → difficult to culture (may have a low WBC)	Trauma Hemophilia

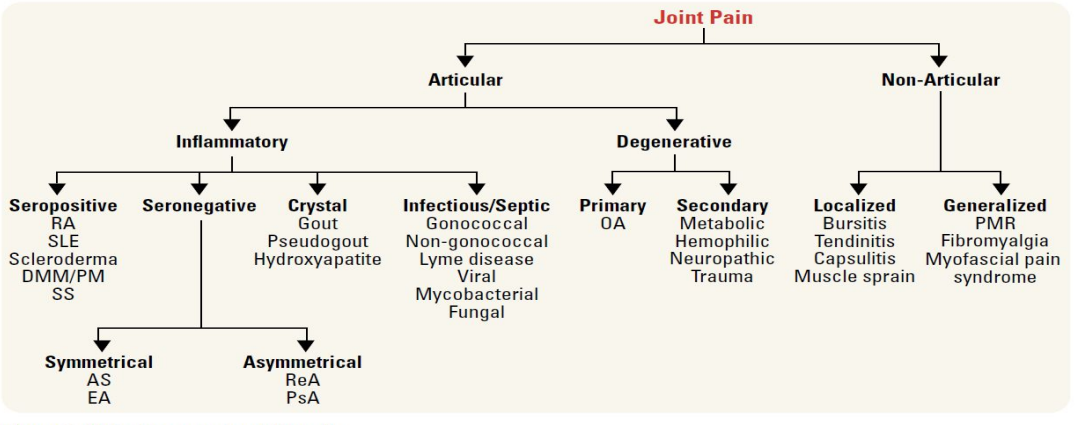

Figure 2. Clinical approach to joint pain

Extra:

Risk Factors

- genetic predisposition, advanced age, obesity (for knee and hand OA), female, trauma

Table 9. Signs and Symptoms of OA

Signs	Symptoms
Joint line tenderness; stress pain ± joint effusion	Joint pain with motion; relieved with rest
Bony enlargement at affected joints	Short duration of stiffness (<1/2 h) after immobility, called gelling
Malalignment/deformity (angulation)	Joint instability/buckling (often due to ligamentous instability)
Limited ROM	Joint locking due to "joint mouse" (bone or cartilage fragment)
Crepitus on passive ROM	Loss of function (e.g. meniscal tear or other internal derangements)
Inflammation (mild if present)	Insidious onset of pain, localized to affected joints
Periarticular muscle atrophy	Fatigue, poor sleep, impact on mood

Joint Involvement

- generalized osteoarthritis: 3+ joint groups
- asymmetric (knees usually affected bilaterally)
- hand
  - DIP (Heberden's nodes = osteophytes → enlargement of joints)
  - PIP (Bouchard's nodes)
  - CMC (usually thumb squaring)
  - 1st MCP (other MCPs are usually spared)
- hip
  - usually presents as groin pain ± dull or sharp pain in the trochanteric area, internal rotation and abduction are lost first
  - pain can radiate to the anterior thigh, but generally does not go below the knee
- knee
  - initial narrowing of one compartment, medial > lateral; seen on standing x-rays, often patellar-femoral joint involved
- foot
  - common in first MTP and midfoot
- lumbar spine
  - very common, especially L4-L5, L5-S1
  - degeneration of intervertebral discs and facet joints
  - reactive bone growth can contribute to neurological impingement (e.g. sciatica, neurogenic claudication) or spondylolisthesis (forward or backward movement of one vertebra over another)
- cervical spine
  - commonly presents with neck pain that radiates to scapula, especially in mid-lower cervical area (C5 and C6)

Investigations

- blood work
  - normal CBC and ESR, CRP
  - negative RF and ANA
- radiology: 4 hallmark findings, see sidebar
- synovial fluid: non-inflammatory (see Table 8, RH4)

Treatment

- presently no treatment alters the natural history of OA
- prevention: prevent injury, weight management, physical activity (maintenance of muscle strength)
- **non-pharmacological therapy**
  - weight loss (minimum 5-10 lb loss) if overweight
  - exercise: more effective if supervised, often by physiotherapists or in a class setting; Tai chi is strongly recommended for hip/knee OA
  - self-efficacy and self-management programs (goal-setting, positive thinking, education on the disease)
  - thermal intervention: heat or cold
  - occupational therapy: aids, splints, cane, walker, bracing
- **pharmacological therapy** (see Table 34, RH30)
  - stepped approach to therapy (local → systemic therapy)
  - local therapy:
    - topical NSAIDs, topical capsaicin (knee, hand OA)
    - injections: intra-articular glucocorticoids (knee, hip OA)
  - systemic therapy:
    - acetaminophen, oral NSAIDs
    - centrally acting agents (e.g. duloxetine)
  - evidence is inconclusive on the use of: intra-articular hylauronates, platelet-rich plasma and stem cell therapy for hip or knee OA
  - there is insufficient evidence to support use of: chondroitin, glucosamine herbal remedies, and supplements
  - the following are not recommended: opioids and medical cannabinoids (for pain)
- **surgical treatment**
  - total and/or partial joint replacement, joint debridement (not shown to be effective), osteotomy, fusion

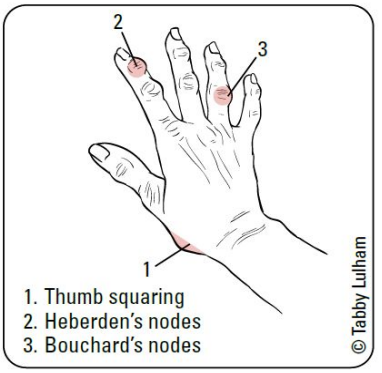

Figure 4. Hand findings in OA

Differential Diagnosis of Elevated ESR

- Systemic inflammatory diseases
- Localized inflammatory diseases
- Malignancy
- Trauma
- Infection
- Tissue injury/ischemia

The Radiographic Hallmarks of OA

- Joint space narrowing
- Subchondral sclerosis
- Subchondral cysts
- Osteophytes

Exercise for Osteoarthritis of the Knee: A Cochrane Systematic Review

Br J Sports Med 2015;49:1554-1557

Purpose: To determine if land-based therapeutic exercise is beneficial for people with knee OA in reducing pain, improving physical function, and improving quality of life.

Methods: Five databases searched for randomized clinical trials comparing therapeutic exercise with a non-exericise control.

Results: 54 studies identified. Results from 44 trials indicate that exercise significantly reduced pain (12 points/100; 95% CI 10 to 15) and improved physical function (10 points/100; 95% CI 8 to 13) after treatment. 13 studies showed improved quality of life with exercise. 12 studies showed reduced knee pain (6 points/100; 95% CI 3 to 19) and 10 studies showed improved physical function (3 points/100;95% CI 1 to 5) with exercise.

Conclusions: In people with knee OA, land-based therapeutic exercise provides short term benefit that is sustained a few months after treatment.

Quiz

MCQs

Q1: Which of the following non-operative treatments for osteoarthritis has the best evidence to support its use?

A. Combination of supervised and home exercise programs
B. Hyaluronic acid injections
C. Paracetamol
D. Lateral heel wedge

Q2: According to the latest recommendations made for the treatment of osteoarthritis (OA) of the knee, which of the following nonoperative treatment modalities has the weakest supporting evidence for the treatment for knee osteoarthritis?

A. Weight loss
B. Activity modification
C. Intra-articular corticosteroids injection
D. Intra-articular hyaluronic acid injection

Q3: A 61-year-old man with progressive left hip pain comes to the physician for a follow-up examination. One year ago, he was diagnosed with osteoarthritis of the left hip. Since then, he has had an 8-kg (18-lb) weight loss after changing to a vegetarian diet, regular swimming, and physical therapy. The pain worsens when he climbs stairs, which makes it increasingly difficult for him to reach his apartment located on the second floor. Over the last few weeks, he gradually increased the frequency of diclofenac intake but says that even a daily intake does not provide complete pain relief. He asks if there is a treatment that will lead to a long-term improvement of his symptoms. He has no history of major medical illness. His only other medication is pantoprazole. He does not smoke or drink alcohol. He is 179 cm (5 ft 10 in) tall and weighs 80 kg (176 lb); BMI is 25 kg/m2. Physical examination of the left hip shows crepitus, a limited internal rotation, and pain with full flexion and extension. An x-ray of the left hip shows joint space narrowing, osteophytes, and subchondral sclerosis and cysts. Which of the following is the most appropriate next step in management?

A. Refer for arthroscopic hip debridement
B. Perform intra-articular glucocorticoid injections
C. Prescribe walking aids
D. Refer for total hip arthroplasty

SAQs

Case

1. What advice would you give a patient recently diagnosed with Osteoarthritis to slow down the progression of disease and improve his symptoms?
   Maintain movement and muscle strength, lose weight.
2. Please describe the role of radiological investigations in the diagnosis and management of degenerative joint diseases, and mention any relevant technical aspects when requesting these investigations.
   Imaging is used for confirmation, not for diagnosis. We can use it to differentiate between primary and secondary osteoarthritis.
   Request images on weight-bearing.

Answers

Q1	Q2	Q3
A	D	D

THANK YOU

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