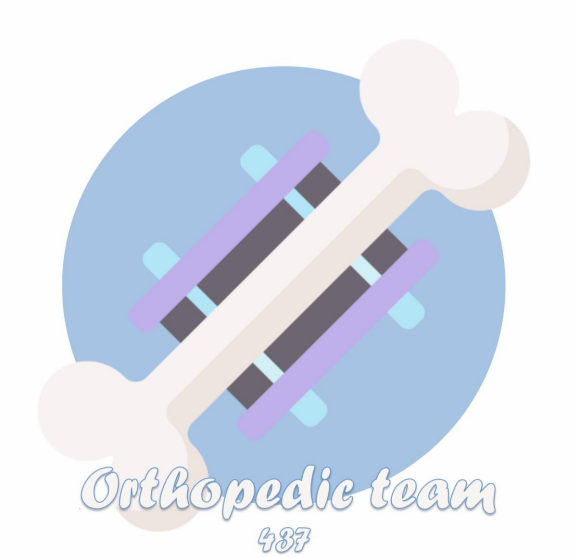
DEGENERATIVE JOINT DISORDERS



Lecture objectives:

Not given

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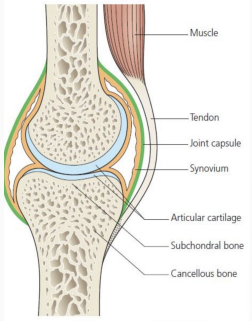
Revised by: Sondos AlHawamdeh
References: 436 team, Doctors notes

INTRODUCTION



Articular cartilage:

- It is avascular and aneural **hyaline cartilage.**
- **Viscoelastic material with variable load - bearing propertie;** decreases joint friction.

Cartilage composition:

1- Water (60% to 80% wet weight) most important part

- Pumped in and out of cartilage **depending on load**.
- **Contributes to lubrication and Nutrition.**

2- Collagen (10% to 20% wet weight)

- Secreted by chondrocytes.
- Mostly type 2 collagen (90%).
- Confers **tensile strength** to cartilage

3- Proteoglycans (10% to 15% wet weight)

- **Secreted by chondrocytes.**
- Composed of GAG (glycosaminoglycan) (aggrecan, chondroitin, and keratin sulfate)
- Negatively charged proteins holds water within matrix.
- Provides compressive strength.

4- Chondrocytes (5% wet weight)

- **Only cell type in cartilage, have little capacity for cell division in vivo[1].**

- So, when there is a direct damage to the articular surface, cartilage is poorly repaired or repaired only with (poor cartilage) **fibrocartilage** which has inferior biomechanical properties than hyaline cartilage and it is weaker than hyaline cartilage, and it wears out so much quicker.
- **If the collagen network is disrupted so water** comes to cartilage matrix **and the matrix becomes waterlogged and soft** look like sponge and be very friable, followed by loss of proteoglycans, cellular damage and splitting "fibrillation" of articular Cartilage and damaged chondrocytes begin to release matrix-degrading enzymes inside the joint cause further damage.

  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.

5- Capsule and ligaments:

- **Fibrous structure with tough condensations on its surface (ligaments).**
- Together with the overlying muscles, they help to provide stability. which is imp Ligaments provide stability to the joint (زي الوتد) when it's torn we lose a major stabilizing factor.

What is the difference between ligaments and tendons? We can't control ligaments but we can control tendons by muscle contraction

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[1] Studies are those in which the effects of various biological entities are tested on whole, living organisms.

6- Synovium:

- **Thin membrane, 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.

7- Synovial fluid:

- **Have several functions: Nourishes the avascular articular cartilage, plays an important part in reducing friction during movement** by type of fluid itself**, it also has slight adhesive properties which help in maintaining joint stability.** Very minimal role in stability
- The volume remains fairly constant, regardless of movement. Unless the joint got injured or infected it increases resulting in joint effusion = fluid accumulates and we feel it clinically as effusions. The excessive synovial fluid is not the pathology by itself, we have to know the underlying cause of the excessive production and treat accordingly
- **Role of synovial fluid: lubrication, nutrition, stability.**

DEGENERATIVE JOINT DISEASE

OSTEOARTHRITIS VIDEO, OSTEOARTHRITIS HIGH YIELD NOTES OSMOSIS, MAYO CLINIC OA

When you get the words Osteoarthritis/arthritis/osteoarthrosis (it meant to be degenerative type unless they specified (infective, inflammatory and so on)

1- Primary (idiopathic) Osteoarthritis OA: (خشونة المفاصل)

Chronic disorder in which there is:

1. **Progressive softening and disintegration of articular cartilage.**
2. **New growth of cartilage and bone at the joint margins (osteophytes).**
3. **Subchondral bone sclerosis and cyst formation.**
4. **Mild synovitis** effusion **and capsular fibrosis** due to not bending the joint because of pain this will lead to reduced range of motion

- ❏ Asymmetrically distributed, often localized to only **one** part of a joint for ex: if you have varus this leads to medial damages b /c most of the weight is in the medial side of joint results in faster wear out and tear, while having valgus leads to lateral damage.
- ❏ **Often associated with abnormal loading,** usually causes of OA is more mechanical (weight and loading); whatever part is loaded more will have early osteoartheritis like in obese patients patello-femoral part will be affected.
- ❏ **Un**accompanied by any systemic illness
- ❏ **Not primarily an inflammatory disorder** (although there are sometimes local signs of inflammation (reactive synovitis)).
- ❏ **Not a purely degenerative** (dynamic phenomenon: it shows features of both destruction and repair (osteophytes)). It is a misnomer because they have signs of degeneration and repair
- ❏ Keep in mind once degeneration starts it will not stop, it is progressive, we try to slow it down, change the patient's lifestyle to improve quality of life. We mainly depend on quality of life when treating a patient so we depend on it not the x-ray. REMEMBER it about the patients quality of life we don't treat x-rays we treat patients.

2- Secondary Osteoarthritis OA:

In secondary usually the **whole** joint is affected unlike primary osteoarthritis.

Trauma	Osteochondral, malunion, sport injury, dislocation.
Infection	
Metabolic	Crystal deposition disease (gout, CPPD[2]), Paget's disease.
Inflammatory	RA, SLE, Reiter's syndrome[3].
Congenital/developmental	Hip dysplasia, multiple epiphyseal dysplasia.
Necrosis	Perthes disease, osteonecrosis, steroids.
Neuropathic	DM, Tabes dorsalis.
Hematologic	Sickle cell anemia, hemophilia.
Endocrine	DM, Acromegaly.

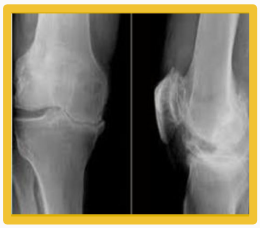
Etiology: OA has no single cause; rather, it is due to a variable combination of several risk factors.

- **Increased mechanical stress in some part of the articular cartilage,** This can be caused by overuse or joint instability.
- OA results from a disparity between the mechanical stress applied to articular cartilage and the ability of the cartilage to withstand that stress (which result in progressive deformity of the joint). This could be due to one or a combination of **two processes: Weakening of the articular cartilage** (due to a genetic defect or enzyme activities).
- **Varus deformity of the knee. Medial side is the most affected.** For example in the knee, the normal knee is in valgus with the inflammation and degenerative OA it becomes varus.
- **Other causes (risk factors): (primary OA)**

  OA is more of a process than a disease.

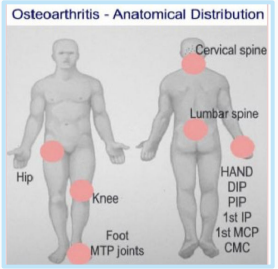
1. Increases in frequency with age. 2. **Family history** IMP factor 4. Poor sitting
3. Obesity (hips and knees take 3-4 times body weight with each step).

Remember we don't see cartilage in x-ray we see joint space. Usually the patient should be in a weight bearing position (standing) so we can properly interpret the image. (Centigram x-ray)



Prevalence:

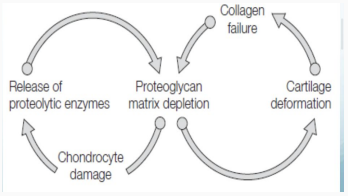
- Osteoarthritis is the commonest of all joint disease
- Much more common in some joints (knee, hip, spine (Cervical & Lumbar) and the fingers, 1st Carpometacarpal, 1st Metatarsophalangeal and Interphalangeal joints) than in others (the elbow, wrist and ankle).
- Females > males.
- Common in our community especially knees & presents earlier than in West
- About 90% of those over 40 have asymptomatic degeneration of weight bearing joints.



The 5 cardinal (major) features are:

1- Progressive cartilage destruction seen in all X-rays

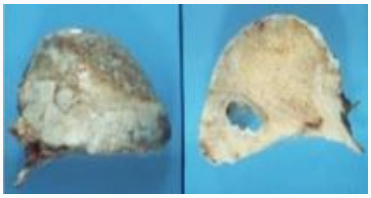
- **Increase** in water content leads to swelling and softening of the cartilage.
- Then later on there will be:
  1. Depletion of proteoglycans.
  2. Chondrocytes damage and synovitis > proteolytic enzymes > causes collagen disruption splitting of articular cartilage.
  3. Fibrillation on weight bearing surfaces and then complete destruction.

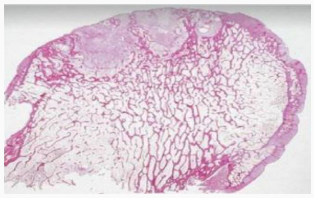


2- Subarticular cyst formation cyst doesn't usually appear in x-rays of knees but in hips is common to see it

Subarticular cyst could arise from:

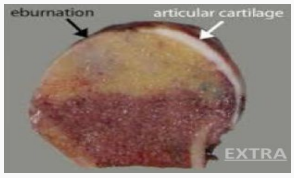
1. Local areas of osteonecrosis the bone get necrosed and die **Or**
2. From the forceful pumping of synovial fluid through cracks in the subchondral bone plate. From the fibrillation (crack in the cartilage) (with pressure there will be pumping of fluid which will accumulate under the sub-articular cartilage to form the cyst.

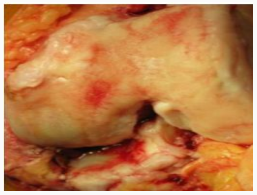


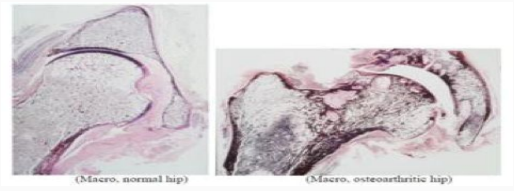


3- Sclerosis of the surrounding bone seen in all X-rays

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

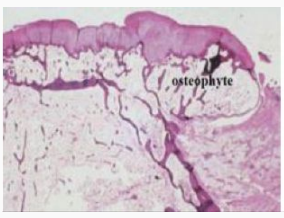


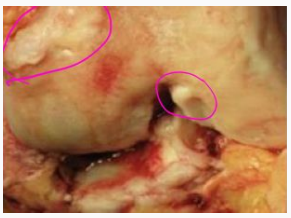




4- Osteophyte formation seen in all X-rays

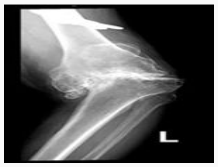
- Proliferation and remodeling of the adjacent cartilage at the edges. followed by
- Endochondral ossification of that cartilage. The joint try to increase the surface area to distribute the stress so it forms a cartilage at the periphery of the joint and this cartilage underwent endochondral ossification and becomes a **bone**.





5- Capsular fibrosis

- Marked vascularity and venous congestion of the subchondral bone. Cause of pain (in medial side), Consider it in the management plan.
- **The capsule and synovium are often thickened but cellular activity is slight.**
- Progressive bone erosion > **BONE COLLAPSE**.
- Fragmented osteophyte > **LOOSE BODIES**. That could block or latch the joint
- Loss of height and ligamentous laxity > **MALALIGNMENT** like in severe varus LCL will stretch out a lot and that will cause ligament loss



Bold are Signs of severe or recurrent OA, if patient presents with night pain or pain at rest (End stage disease)

★ **Key pathological features of OA (EXTRA FYI from apley):**

Pathology	Radiographic correlates
Focal areas of loss of articular cartilage	Joint space narrowing (if loss is extensive)
Bone growth at the joint margins	Osteophytes
Sclerosis of underlying bone	Sclerosis of subchondral bone
Cyst formation in underlying bone	Bone cyst
Loss of bone	Bone attrition
Varying degree of synovial inflammation	Effusions may be apparent
Fibrosis and thickening of the joint capsule	Not visible on radiograph

Clinical features:

It has an **intermittent** course, with period of remission sometimes lasting for months, affecting one or two of the weight bearing joints (hip or knee) in our community patients having degenerative **knee** will have degenerative **lumbosacral & cervical** spine, So in the surgery for knees check spine and vice versa. Pain of OA started in one of two ways either **suddenly** after an event (I did something I'm not used to (Marathon)) or **progressively** (day after day). Pain usually with activity relieved by rest. So? **Mechanical pain!** Pain not always there + not always the same.

Symptoms: The classic symptoms of osteoarthritis: Pain, Limitation of movement, swelling.

- **Pain:**

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 and in advance stage there will be night pain or pain at rest.

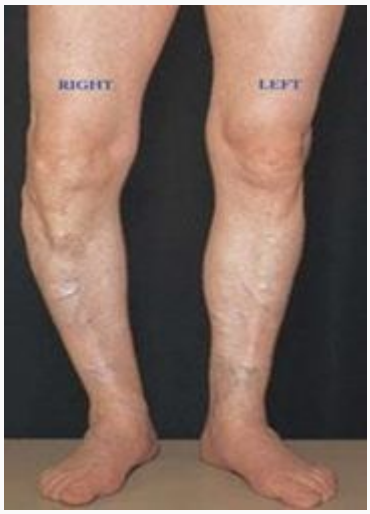
➢ The pain is never steady somedas it is worse than the others

Causes of the pain:

1. Bone pressure due to vascular congestion and intraosseous hypertension **(most important)** osteotomy done in young patient is based on this theory in order to relieve the vascular congestion and intraosseous hypertension.
2. Mild synovial inflammation.
3. Capsular fibrosis with pain on stretching the shrunken tissue.
4. Muscular fatigue. In advanced cases this is a major cause of pain, when patients do massage the pain resolve temporary but eventually the muscle will spasm again and the pain will return

- **Stiffness:** initially after periods of inactivity, but later on it will be constant and progressive.
- The worst advice you give the patient is telling 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!

- **Loss of function.**

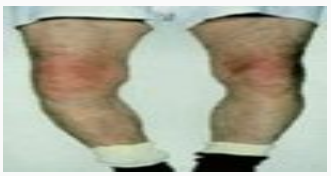


BOTH knees are affected by varus, but right knee is severely affected and left Is moderately affected, progressive OA in medial part of right knee is resulting in progressive varus of knee.

Normal valgus of knees (5-7 degree), 7 in female because they have hip and ligament laxity.

Signs:

- **Swelling**: Intermittent (→effusions), Continuous (→large osteophytes).
- **Deformity**; mal-alignment.
- **Tenderness.**
- **Limited** range of **movement.**
- **Crepitus**[2]. Joint crepitation is not an indication of OA unless it is accompanied by pain or swelling
- **Instability** because of Loss of cartilage and bone, asymmetrical capsular contracture and/or muscle weakness.



Heberden's nodes

[2] Rubbing of bone against bone, you can both hear and feel.

- **Natural history of the disease:**

A patient walks 5k usually, whenever he walks 6 km he'll get pain and effusion once a year, the next year it happens more and the swelling lasts longer. Later it will be monthly. He may use a cane and pray on a chair; thus he will not move a lot and will eventually develop stiffness.

➔ Imaging:

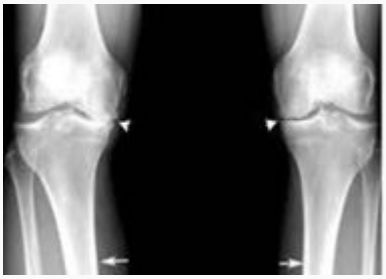
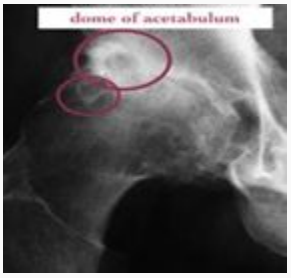
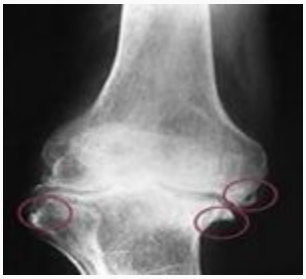
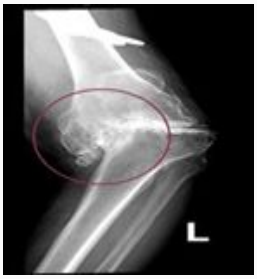
➔ Standing X-ray (while weight bearing). X-ray shows joint space and it should be equal in all join parts (medial and lateral)

➔ OA x-ray finding Mnemonic: **LOSS** (Subchondral cyst)

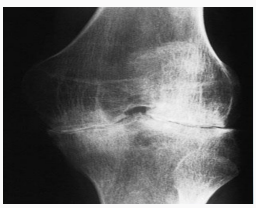
➔ **Asymmetrical Loss of cartilage (narrowing of the 'joint space').**

- **Osteophytes at the margins of the joint.**
- **Subchondral bone Sclerosis.**
- **Cysts close to the articular surface** in knee most likely → medial side, hip → dome (superior-lateral).

➔ **Late features: Malalignment, Joint subluxation, Bone loss, Loose bodies.**

Subchondral bone sclerosis	Subchondral cyst	Osteophyte formation	Bone loss
			
White sclerotic lesion in the medial side of knee and complete loss of cartilage. Notice the narrowing of joint space. Primary OA	Cysts at the dome of acetabulum (superior-lateral part). Loss of cartilage at the femoral head with acetabulum.	Malalignment, Osteophyte, joint subluxation, bone loss, loose bodies. This is classic pic of systematic cause of arthritis, most likely advanced secondary OA b/c whole joint is affected.	

Look for signs of other disorder: **Symmetric** narrowing in **inflammatory OA** (such as RA).



EXTRA: Rheumatoid arthritis or sepsis X-ray finding Mnemonic: **LESS**

L: loss of joint space (Symmetric), E: erosion of joint, S: synovial thickening, S: subluxation and joint deformities.

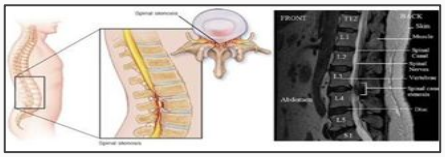
Complications:

1. Loose bodies.

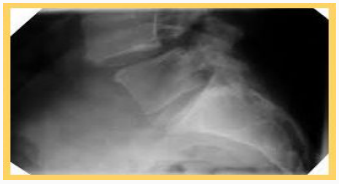
1. Rotator cuff dysfunction: Acromioclavicular (AC) joint OA.

Osteophytes compress the joint and overlying rotator cuff causing impingement of rotator cuff and tendonitis and later on tear.

1. **Spinal stenosis,** very dangerous.



1. Spondylolisthesis: Severe segmental instability at L4/ L5.



1. Capsular herniation: Knee OA; marked effusion and herniation of the posterior capsule (Baker's cyst) filled with synovial fluid. Useless to remove (will happen again).

Management: Depends on several factors:

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

Secondary treat underlying cause,

Early treatment:

- Maintain movement and muscle strength. Strengthening the muscle & improving the ROM is an important part in extending the life of a joint.
- **Protect the joint from overload.** Lose weight in obesity.
- Relieve pain. Via analgesics.
- Modify daily activities.

Conservative treatment:

Maintain movement and muscle strength: by **physiotherapy** which will help in:

1. Pain relief (either by massage or application of warmth) or by other different types of physiotherapy
2. Prevents contractures.
3. Muscle strengthening.
4. Enhancing the range of motion.

Protect the joint from overload (Load reduction): Never advice the patient to stop moving or to be immboile

1. Weight reduction. (if the patient obese)
2. Shock-absorbing shoes.
3. Walking stick. الألم في الركبة اليمين يمسك العصا في اليد العكس اليسار
4. Unloading brace. if has varus knee, unloading brace pushes knee to valgus لما يمشي يحمل على الجزء اللي فيه كارتلج ، but **Doesn't** change anything, fails to show any benefits.
5. Not bearing heavy object.

Modify activity and sitting habits:

Pray in chair instead

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, relief pain

4- Sem cell: no proof of effectiveness

Remember injection is not a part of standard treatment of OA

Surgical treatment: after failing of conservative treatment

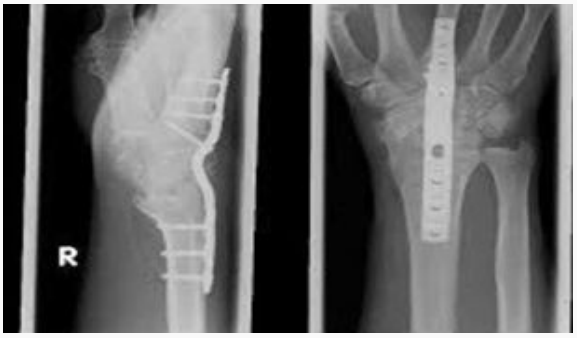
1- Joint Debridement (Arthroscopy) تنظيف الركبة Honeymoon surgery

- Removal of: Loose bodies, Meniscal tear or labral tears.
- For mechanical symptoms.
-

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)

2- Arthrodesis لحم المفصل

- Transfer from painful stiff into painless stiff joint. Leads to loss of motion.
- Small joints; hand (wrist joint), foot and spine.

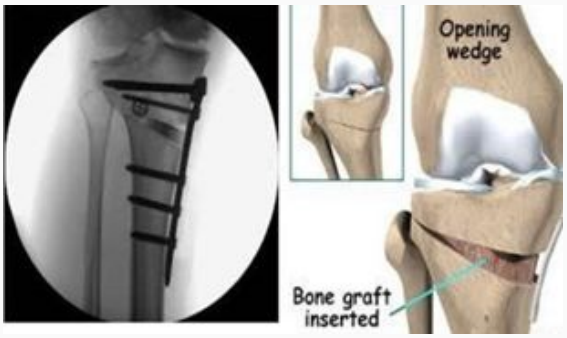


3- Corrective osteotomy القطع العظمي التصحيحي / الخزوع العظمية التصحيحية (good for deformities in general)

- Realign axis and redistributes weight.
- Knee; hip.

Has a role in pain relief by:

1. Vascular decompression of the subchondral bone.
2. Redistribution of loading forces toward less damaged parts of the joint.



Usual candidates are: 3

1. **Young** less than 50, after 50 is better to do total knee replacement.
2. **Active** patients.
3. **Mild OA affecting only one part of the knee**

4- Arthroplasty (Joint replacement) عملية تبديل المفصل

Nowadays; the **procedure of choice** for advanced OA. not first line it is done when conservitive treatment fails

#**Total** joint replacement:

- Knee, hip, shoulder, ankle and elbow.
- Painful, deformed stiff joint, old patients.

#**Partial** joint replacement:

- Knee joint.
- Same patient as for osteotomy. (young, active, mild OA)

EXTRA: SYMPTOMS AND SIGNS AT DIFFERENT JOINT SITES: (435 team)

#HIP:

- Pain is usually felt in the groin, laterally over the hip and radiates down the anterolateral aspect of the thigh to the knee.
    - Occasionally the pain can radiate beyond the knee. Referred pain felt only in the knee is not uncommon, and clinicians should always consider hip OA as a cause of isolated knee pain.
- Pain is worse on exercise and walking distance is reduced. Pain at rest and night pain can be particularly troublesome.
- Stiffness is usually experienced first thing in the morning and after having sat still for a while, but it quickly resolves on movement to be replaced by pain. Complex movements, such as getting in and out of a motorcar or putting on socks, which involve deep flexion combined with rotation, are often difficult or impossible to perform. Patients struggle with stairs and in the absence of a banister may only manage stairs on all fours.

EXAMINATION:

- ➢ **Antalgic gait**, characterized by an uneven cadence, in which less time is spent in the stance phase of the painful limb.
- ➢ There is a **globally reduced range of movement** with internal rotation often restricted early in the disease progression.
- ➢ Joint movement is **limited by pain** at the extremes of movement.

Management: Weight reduction + pain management + physiotherapy

.#KNEE:

- Knee osteoarthritis occurs most commonly in the **medial tibiofemoral joint** but can occur in all three compartments and is often tricompartmental. Isolated patellofemoral OA is probably due to altered biomechanics of the extensor mechanism.
- Pain is felt globally over the knee and the proximal tibia. In isolated patellofemoral OA the pain is felt anteriorly over the knee and is often worst when ascending or descending stairs as the patella is compressed against the femur.
- As in the hip, the pain is a deep-seated aching sensation related to exercise. Rest pain and night pain develop in the later stages.
- Patients sometimes report **audible crepitus** (crackling or grating sounds) coming from the knee as well as **symptoms of instability** (a feeling that the knee is going to give way).
- They may notice gradual deformity of the knee, in **particular varus deformity**, but less commonly valgus deformity. **Fixed flexion deformity** means that the knees cannot lock in full extension and thus patients cannot stand comfortably for prolonged periods due to muscle fatigue. Loss of flexion beyond 90 degrees makes standing from a sitting position difficult as patients cannot move their centre of gravity anterior to their mid-coronal plane. Swelling and stiffness are common features.

EXAMINATION:

- ➢ **Antalgic gait, wasting of quadriceps muscles, joint effusion.**
- ➢ **Joint deformity** the joint deformity may be passively correctable. Deformity is towards the compartment most severely affected, usually varus deformity with predominantly medial compartment OA.
- ➢ **Crepitus palpable and sometimes audible on movement.**
- ➢ **Tenderness along the joint line and palpable osteophytes that can be tender.**

#HAND:

- Most commonly affecting **DIPs and the thumb base** (both the radiocarpal and scaphotrapezoid joints), less commonly, PIPs joints and metacarpophalangeal joints.
- OA of the hand is strongly associated with OA at other joint sites, especially the knee, and with genetic predisposition. It is far more common in **women** starts relatively abruptly around the time of the **menopause** (sometimes called 'menopausal OA') with painful inflammation in DIPs joints over time(years) the inflammation settles and the joint is left with the typical pathological features of OA.
- **Erosions** can occur ('erosive OA'), and cysts containing hyaluronan that protrude at the margins of the joints are not uncommon.
- Distal interphalangeal joint OA is not generally a major problem in terms of function, but thumb base OA can be, as it leads to instability and difficulty with pinch grip.

#Other joints:

Almost any joint can be affected by OA, particularly if it is damaged by severe trauma.

However, there are peculiarities to the phenotype of the condition at different sites. For example, elbow OA is almost always asymptomatic (just causing loss of full extension of the elbow), while shoulder OA is more likely to result in severe bone destruction (a condition sometimes called 'Milwaukee shoulder') than is OA at other joint sites.

Degenerative Arthritis: Osteoarthritis

• see Family Medicine, FM40

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
• now 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 yr olds, 85% of 80 yr olds)

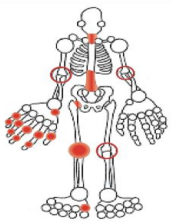
Risk Factors
• genetic predisposition, advanced age, obesity (for knee and hand OA), female, trauma

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

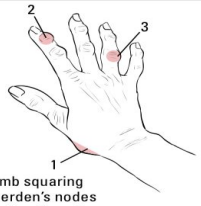
OA of MCP joints can be seen in hemochromatosis or CPPD-related disease (chondrocalcinosis)

ESR can also be elevated in anemia, end-stage renal disease, females, increased age, and obesity



• 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

Figure 3. Common sites of joint involvement in OA



1. Thumb squaring
2. Heberden's nodes
3. Bouchard's nodes

Figure 4. Hand findings in OA

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)

Differential Diagnosis of Elevated ESR
• Systemic inflammatory diseases
• Localized inflammatory diseases
• Malignancy
• Trauma
• Infection
• Tissue injury/ischemia

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)

The Radiographic Hallmarks of OA
• Joint space narrowing
• Subchondral sclerosis
• Subchondral cysts
• Osteophytes

Treatment
• presently no treatment alters the natural history of OA
• prevention: prevent injury, weight management, maintenance of muscle strength
• **non-pharmacological therapy**
  ▪ weight loss (minimum 5-10 lb loss) if overweight
  ▪ physiotherapy: heat/cold, low impact exercise programs
  ▪ occupational therapy: aids, splints, cane, walker, bracing
• **pharmacological therapy** (see Table 34, RH29)
  ▪ 1st line with few joints affected, knee, hand – topical: transdermal NSAIDs preparations, capsaicin
  ▪ 1st line with multiple joints, hip – oral: acetaminophen/NSAIDs
  ▪ treat neuropathic pain if present (anti-depressants, anti-epileptics, etc.)
  ▪ joint injections: corticosteroid (sparse use, effective for short-term treatment), hyaluronic acid (little evidence of benefits)
  ▪ glucosamine ± chondroitin (efficacy not supported)
• **surgical treatment**
  ▪ total and/or partial joint replacement, joint debridement (not shown to be effective), osteotomy, fusion

Osteoarthritis

• see Rheumatology, RH5

Epidemiology
- most common form of arthritis seen in primary care
- prevalence is 10-12% and increases with age
- results in long-term disability in 2-3% of patients with OA
- almost everyone over the age of 65 shows signs of OA on x-ray, but only 33% of these individuals will be symptomatic

Clinical Features
- joint pain with activity, improved with rest, morning stiffness or gelling <30 min
- deformity, bony enlargement, crepitus, limitation of movement, peri-articular muscle atrophy
- usually affects distal joints of hands, spine, hips, and knees

Investigations
- no laboratory tests for the diagnosis of OA
- hallmark radiographic features: joint space narrowing, subchondral sclerosis, subchondral cysts, osteophytes

Management
- goals: relieve pain, preserve joint motion and function, prevent further injury
- conservative
  - patient education, weight loss, low-impact exercise (OT/PT), assistive devices (e.g. canes, orthotics)
- pharmacological
  - consider comorbidities such as PUD, HTN, IHD, hepatic disease, and renal disease
  - medications do not alter natural course of OA
  - 1st line: acetaminophen up to 4 g/d (OA is not an inflammatory disorder)
  - 2nd line: NSAIDs (COX-2 selective) in low doses for short durations
  - 3rd line: combination analgesics (e.g. acetaminophen and codeine)
  - other pharmacological adjuncts:
    - intra-articular corticosteroid or hyaluronic acid injections
    - topical NSAIDS (diclofenac)
    - capsaicin cream
    - oral glucosamine
- surgery
  - consider if persistent significant pain and functional impairment despite optimal pharmacotherapy (e.g. debridement, osteotomy, total joint arthroplasty)

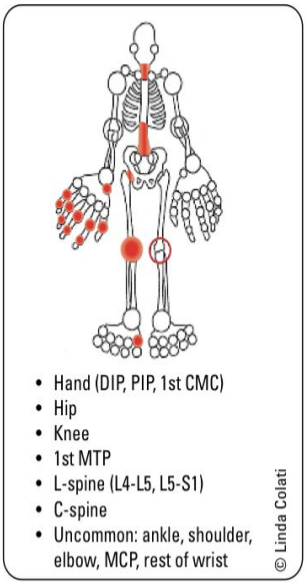


Figure 14. Common sites of involvement in OA

QUESTIONS

1- Recurrence of Baker's cyst should make the surgeon suspect:

A. Neoplastic change.
B. Undiagnosed pathology within knee.
C. Incomplete removal of the cyst.
D. The communication to the joint is persisting.

Ans: B

2- Localized Bone Sclerosis may be due to:

A. Syphilis.
B. Sclerosing Osteoperiostitis.
C. Osteoarthritis.
D. Bone tumors.
E. All of the above.

Ans: E

3- Rheumatoid Arthritis primarily involves the:

A. Articular cartilage.
B. Subchondral bone.
C. Synovial membrane.
D. Capsule.
E. Ligaments.

Ans: C

4- Complications of Rheumatoid Arthritis in the hands include:

A. Tenosynovitis.
B. Rupture of extensor tendons.
C. Carpal tunnel syndrome.
D. Ulnar deviation at the metacarpophalangeal joints.
E. Bony ankylosis of affected joints.

Ans :D

5- A 57-year-old lady with osteoarthritis in both knees. They plan to do total knee replacement surgery. What are the imaging findings?

A. Osteophytes.
B. periarticular erosions.
C. Varus deformity.

Ans: A

6- Case of osteoarthritis booked for knee replacement which one of the following clinical manifestations represents advanced disease?

A. Rest pain
B. Stiffness
C. Effusion
D. Tenderness

Ans: A