*Degenerative joint disease
*Wearing-tearing
*Commonest joint disorder
Is a **chronic** joint disorder in which there is progressive softening and disintegration of articular cartilage, accompanied by new growth of cartilage and bone at the joint margins (osteophytes), and capsular fibrosis.
CAUSES

The most obvious feature of OA is that it increases in frequency with age …..65.

It is defined as primary when no cause is obvious.

secondary when it follows a demonstrable abnormality: DM, marked obesity, hemophilia (hemarthrosis).

OA results from a disparity between the stress applied to articular cartilage and the ability of the cartilage to withstand that stress.
The cardinal features are:

1. progressive loss of articular cartilage thickness.
2. subarticular cyst formation and sclerosis.
3. Re-modelling of the bone ends and osteophyte formation, joint mice.
4. synovial irritation.
5. capsular fibrosis.
The earliest morphological change is softening of the articular cartilage.

The subarticular bone reacts to these changes in several ways: In the area of greatest stress, cysts appear and the surrounding trabeculae become thickened or sclerotic.

**Osteophytes:** reactive bony outgrowth develop at the margin of articular surface.

**Joint mice:** small fracture then displace pieces of cartilage and subcondral bone into joint
Bone spur (osteophyte)

Thinned cartilage

Cartilage fragments

Joint affected by osteoarthritis
OA is not a purely degenerative disorder, and the term ‘degenerative arthritis’ – which is often used as a synonym for OA – is a misnomer.

Although OA is not primarily an inflammatory disease, shedding of fragments from the fibrillated articular cartilage, as well as release of enzymes from damaged cells, may give rise to a low-grade synovitis. In the late stages, capsular fibrosis is common and may account for joint stiffness.
Patients usually present after middle age, although it is likely that cartilage changes start 10 or even 20 years before that. Sometimes – especially in younger patients – there is a history of some preceding joint disorder or injury. Joint involvement follows several different patterns either on one or two of the weightbearing joints (hip or knee), on the interphalangeal joints (especially in women) or on any joint that has suffered a previous affliction (e.g. congenital dysplasia, osteonecrosis or intra-articular fracture).
Pain is the usual presenting symptom. It is often quite widespread, or it may be referred to a distant site – for example, pain in the knee from OA of the hip. It starts insidiously and increases slowly over months or years. It is aggravated by exertion and relieved by rest. In the late stage the patient may have pain in bed at night.

Swelling may be intermittent (suggesting an effusion) or continuous (with capsular thickening or large osteophytes).

Deformity may result from capsular contracture or joint instability, but deformity may actually have preceded and contributed to the onset of OA.

Loss of function

In contrast to inflammatory joint disease, OA is unassociated with any systemic manifestations.
**SIGNS**

**Local tenderness** is common, and in superficial joints fluid, synovial thickening or osteophytes may be felt.

**Limited movement** in some directions but not others is usually a feature, and is sometimes associated with pain at the extremes of motion.

**Crepitus** may be felt over the joint (most obvious in the knee) during passive movements.

**Instability** is common in the late stages of articular destruction, but it may be detected much earlier by special testing. Instability can be due to loss of cartilage and bone, asymmetrical capsular contracture and/or muscle weakness.
CLINICAL VARIANT OF OA

1- Mono articular + pauciarticular OA:

dysfunction in 1 or 2 of large weight bearing joints + Obvious underlying abnormality

2- OA in unusual sites:

Uncommon in shoulder, elbow, wrist, ankle.

3- Polyarticular (generalized) OA: (most common form)

The typical pt is middle-aged women pain swelling + stiffness of the finger joints.

Most obvious in hands with interphalangeal joints become swollen + tender + appear to be inflamed.

Heberden’s nodes – DIP

Bouchard’s nodes – PIP
A KNEE WITH OA: X-RAY PROGRESSION

Doubtful (Grade 1)

Mild (Grade 2)

Moderate (Grade 3)
No signs of osteoarthritis

Joint space narrowing

Osteophyte
NORMAL KNEE

JIA/RA:
Symmetrical narrowing

OA:
asymmetrical narrowing

X-rays of two knees illustrating (left) rheumatoid arthritis and (right) degenerative osteoarthritis
Osteoarthritis in the metatarsophalangeal joint of the big toe. Joint space narrowing (black arrow) and bone spurs (white arrows) can be seen.

The X-ray shows complete obliteration of the big toe joint in a case of severe arthritis.
are the basic diagnostic procedure for the disease.
(Mnemonic (loss))

- asymmetrical loss of cartilage (Narrowing of joint space)
- Osteophytes formation Subarticular cyst formation
- Subarticular sclerosis
- (evidence of previous disorders like old fractures, RA may be present)
A number of conditions may mimic OA, some presenting as a monoarthritis and some as a polyarthritis affecting the finger joint.

1- Avascular necrosis ‘Idiopathic’ osteonecrosis causes joint pain and local effusion

2- Inflammatory arthropathies Rheumatoid arthritis, ankylosing spondylitis and Reiter’s disease may start in one or two large joints

3- Polyaarthritis of the fingers

4- Diffuse idiopathic skeletal hyperostosis (DISH)
OSTEOARTHRITIS

DEGENERATIVE DISEASE

MORNING STIFFNESS LASTING LESS THAN 30 MINUTES

HEBERDEN'S NODES

ASYMMETRICAL

CARTILAGE LOSS

RHEUMATOID ARTHRITIS

AUTOIMMUNE DISEASE

MORNING STIFFNESS LASTING MORE THAN 30 MINUTES

INFLAMED SYNOVITUM

SYMETRICAL

EXTRA-ARTICULAR INVOLVEMENT
<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>OSTEARTHRITIS</th>
<th>RHEUMATOID ARTHRITIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Affects the elderly; has slow onset. Pain <strong>worsens with use.</strong></td>
<td>Affects the young; presents with morning stiffness that <strong>improves with use.</strong></td>
</tr>
<tr>
<td>Joint involvement</td>
<td>Affects the DIP, PIP, hips, and knees.</td>
<td>Affects the <strong>wrists, MCP, ankles, knees, shoulders, hips, and elbows.</strong> Has a <strong>symmetrical distribution.</strong></td>
</tr>
</tbody>
</table>
**Complication**

**Baker’s cyst** is flow of the synovial fluid into the gastrocnemius bursa. It can be asymptomatic or can cause restriction of movement. It’s painful when ruptured.

**Rotator cuff dysfunction** Osteoarthritis of the acromioclavicular joint may cause rotator cuff impingement, tendinitis, or cuff tears.

**Spinal stenosis** Longstanding hypertrophic OA of the lumbar apophyseal joints may give rise to acquired spinal stenosis. The abnormality is best demonstrated by CT and MRI.
There are three principles in the treatment of early OA:

1. **Relieve pain**; achieved by analgesics acetaminophen + NSAIDs Ibuprofen

2. **Increase movement**; Joint mobility can often be improved by physiotherapy exercise to improve muscle strength and induce condroplasts

3. **Reduce load** can be achieved by using a walking stick, wearing soft-soled shoes, avoiding prolonged, stressful activity and by weight reduction
- OA of knee: **debridement** of joint removal of interfering osteophytes, cartilage tags and loose bodies) can be performed arthroscopically.

- For both the hip and the knee, **realignment osteotomy** used to be popular, provided the joint was still stable and mobile pain relief was often dramatic, probably because it provided vascular decompression of the subchondral bone as well as redistribution of loading forces towards less damaged parts.

- Corrective osteotomy may prevent or delay progression of the cartilage damage.
- **Arthroplasty** (total joints replacement) is the *procedure of choice* for OA in patients with *severe symptoms*, *marked loss of function* and significant *restriction of daily activities*

- **Arthrodesis** (artificial induction of *joint ossification* between two *bones* by *surgery*) a reasonable choice if *stiffness* is not a drawback. This is most likely to apply to *small joints* that are prone to OA, can be done to large joints in rare conditions as failure of the replacement therapy or joint infection