Osteoarthritis

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OA - Definition

- Heterogeneous group of conditions resulting in common histopathologic and radiologic changes involving
- Entire joint organ, including:
- the articular cartilage
- the subchondral bone and
- ▶ the synovium.

Epidemiology

- Internationally, osteoarthritis is the most common articular disease. Estimates of its frequency vary across different populations.
- 80-90% of individuals older than 65 years have evidence of radiographic osteoarthritis.
- ▶ the prevalence of osteoarthritis is higher among women than among men.
- Interethnic differences in the prevalence of osteoarthritis have been noted.

Involved joints

Weight-bearing joints, including:

the knees

the hips

cervical and lumbosacral spine

feet.

Non weight bearing joints:

the(DIP), the(PIP), and the(CMC) joints.

Joint classification

► Functional:

Synarthroses (immovable)(Skull)

Amphiarthroses (slightly moveable) (symphysis pubis, verebral)

Diarthroses (freely moveable)

Joint Classification

Structural

Synovial

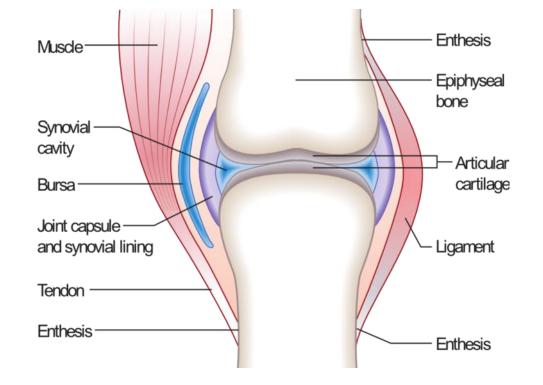
Fibrous

Cartilaginous

Synovial Joints

- Articular cartilage
- Subchondral bone
- ► Synovial membrane
- Synovial fluid
- Joint capsule

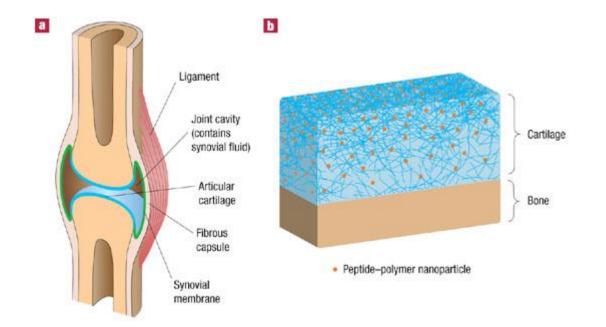
Synovial joint anatomy



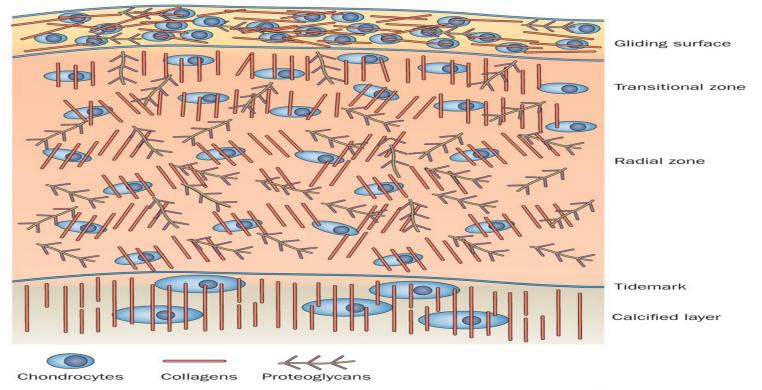
The normal articular surface of synovial joints

- articular cartilage (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, and
- reducing friction at the joint.

Synovial cartilage

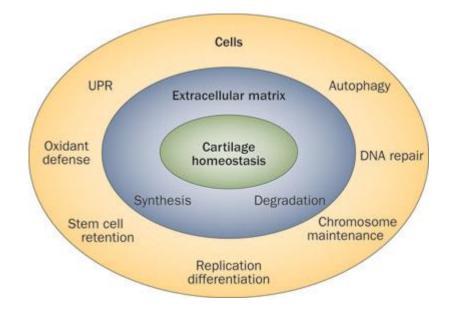


Synovial cartilage

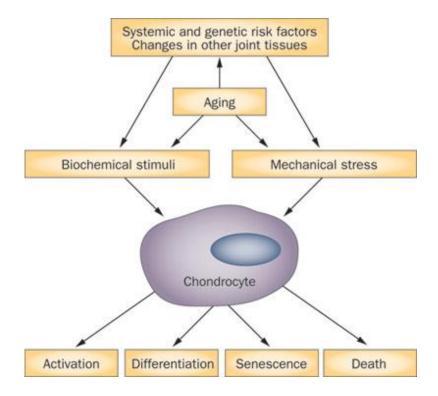


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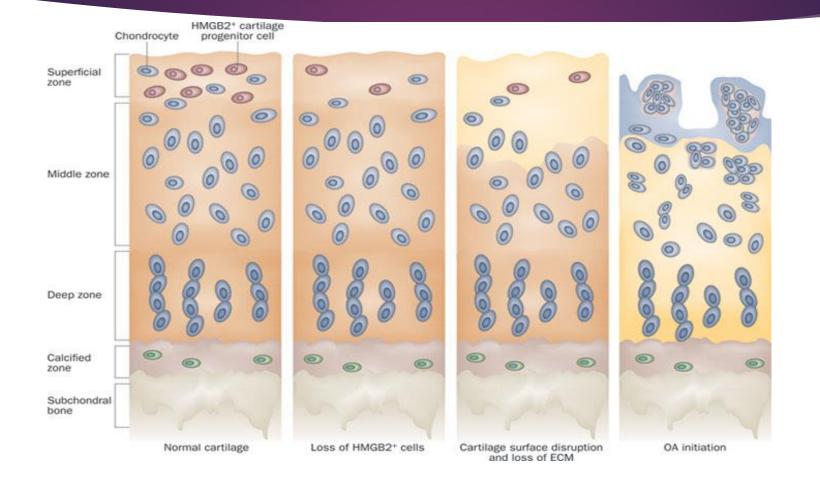
Cartilage homeostasis



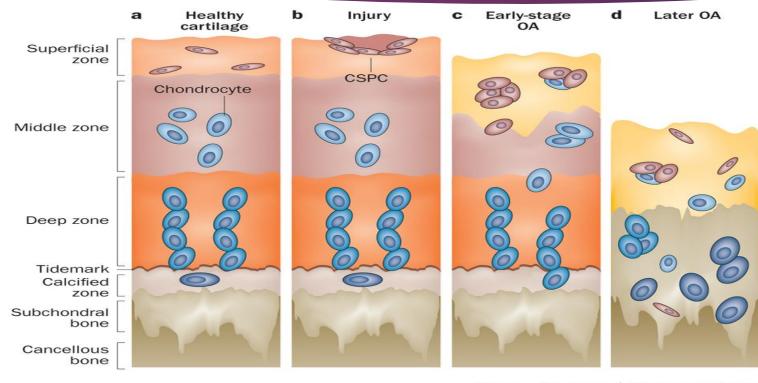
Pathogenesis Pathways in OA



Cartilage Disruption



CSPC distribution in normal articular cartilage and, OA pathogenesis.



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Synovial Fluid

- Synovial fluid is formed by (synoviocytes).
- Synovial cells also manufacture hyaluronic acid (HA, also known as hyaluronate), a glycosaminoglycan that is the major noncellular component of synovial fluid.
- 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.

There is Inflammation in OA

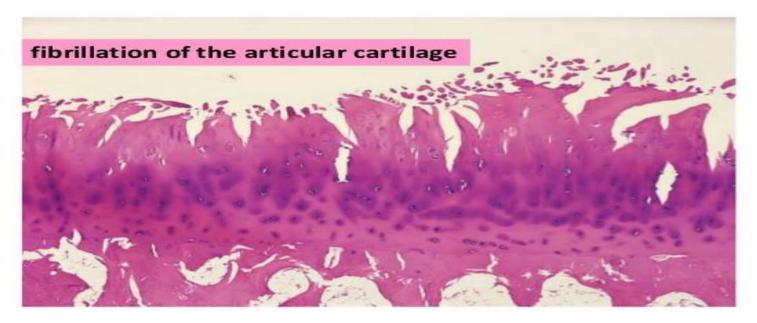
Inflammation occurs as cytokines and metalloproteinases are released into the joint. These agents are involved in the excessive matrix degradation that characterizes cartilage degeneration in osteoarthritis.

Pathogenesis

- Swelling of the cartilage usually occurs
- the level of proteoglycans eventually drops very low, the cartilage softens and lose elasticity and compromising joint surface integrity.
- Flaking and fibrillations (vertical clefts) develop along on the surface of an osteoarthritic joint. Over time, the loss of cartilage results in loss of joint space.
- a greater loss of joint space occurs at those areas experiencing the highest loads.

Cartilage changes

MORPHOLOGY



Clinical and Radiological



Bone changes

- Bone denuded of its protective cartilage continues to articulate with the opposing surface.
- Eventually, the increasing stresses exceed the biomechanical yield strength of the bone.
- The subchondral bone responds with vascular invasion and increased cellularity, becoming thickened and dense (a process known as eburnation) at areas of pressure.

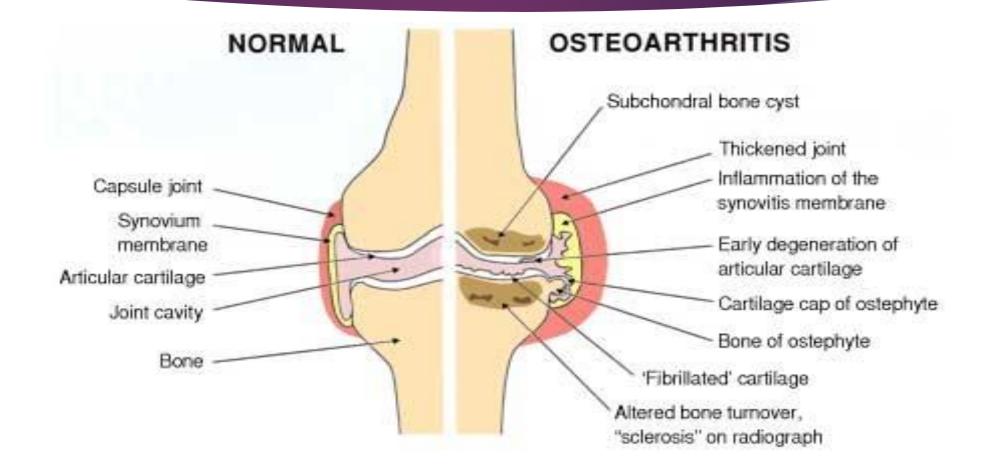
Bone changes

- subchondral bone undergo cystic degeneration.
- Osteoarthritic cysts are also referred to as subchondral cysts, pseudocysts, or geodes and may range from 2 to 20 mm in diameter.
- Osteoarthritic cysts in the acetabulum are termed Egger cysts.

Joint changes

- vascularization of subchondral marrow,
- osseous metaplasia of synovial connective tissue, and
- ossifying cartilaginous protrusions lead to irregular outgrowth of new bone (osteophytes).
- Fragmentation of these osteophytes or of the articular cartilage itself results in the presence of intra-articular loose bodies (joint mice).

Joint changes



Etiology

Risk factors-

- Age, obesity,trauma, genetics, hypogonadism, muscle weakness, repetitive use, Infection, crystal deposition, acromegaly, previous inflammatory arthritis (burnt-out rheumatoid arthritis)
- Heritable metabolic causes (alkaptonuria, hemochromatosis, Wilson disease)Hemoglobinopathies (sickle cell disease and thalassemia)Neuropathic disorders leading to a Charcot joint (syringomyelia, tabes dorsalis, and diabetes)Underlying morphologic risk factors (congenital hip dislocation and slipped femoral capital epiphysis)
- Disorders of bone (Paget disease and avascular necrosis)Previous surgical procedures (meniscectomy)Diabetes mellitus [44]

Radiographic changes



Radiological changes



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.

Primary Osteoarthritis

Three subtypes

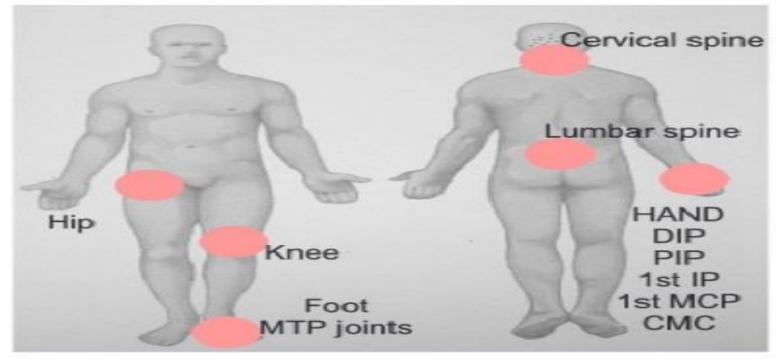
Primary generalized OA

Erosive osteoarthritis

Chondromalacia Patellae

PGOA

Osteoarthritis - Anatomical Distribution



Erosive OA



Chonromalcia Patellae



Differential Diagnosis

- Crystalline arthropathies (ie, gout and pseudogout)
- Inflammatory arthritis (eg, rheumatoid arthritis)
- Seronegative spondyloarthropathies (eg, psoriatic arthritis and reactive arthritis)
- Septic arthritis or postinfectious arthropathy
- Fibromyalgia
- Tendonitis

Work Up

- Laboratory
- Plain Radiography
- CT scan, MRI scan, ultrasonography
- Bone scintigraphy
- Arthrocentesis

Treatment

Non pharmacologic-

Life style modification, physical and rehab therapy

Pharmacotherapy

Arthroscopy

Osteotomy

Arthroplasty

- Fusion and joint Lavage
- Stem cell therapy

References

1.1.Hunter, W. Of the structure and diseases of articulating cartilages. *Phil. Trans. Royal Soc.* 470, 514–521 (1743).
2.2.National Collaborating Centre for Chronic Conditions (UK).
3.National Clinical Guideline for Care and Management in Adults(Royal College of Physicians of London, 2008).
3.3.Felson, D. T. An update on the pathogenesis and epidemiology of osteoarthritis. *Radiol. Clin. North Am.* 42, 1–9 (2004).
4.4.WHO. The World Health Report 2002: reducing risks, promoting healthy life (WHO, 2002).
5.5.Centers for Disease Control and Prevention (CDC). Prevalence of doctor-diagnosed arthritis and arthritis-attributable activity limitation—United States, 2010–2012. *MMWR Morb. Mortal. Wkly Rep.* 62, 869–873 (2013).