Joint diseases

ARTHRITIS

Arthritis is inflammation of joints. The most common forms of arthritis are osteoarthritis and rheumatoid arthritis, which differ in their pathogenesis and clinical and pathologic manifestations . Other types of arthritis are caused by
immune reactions, infections, and crystal deposition.

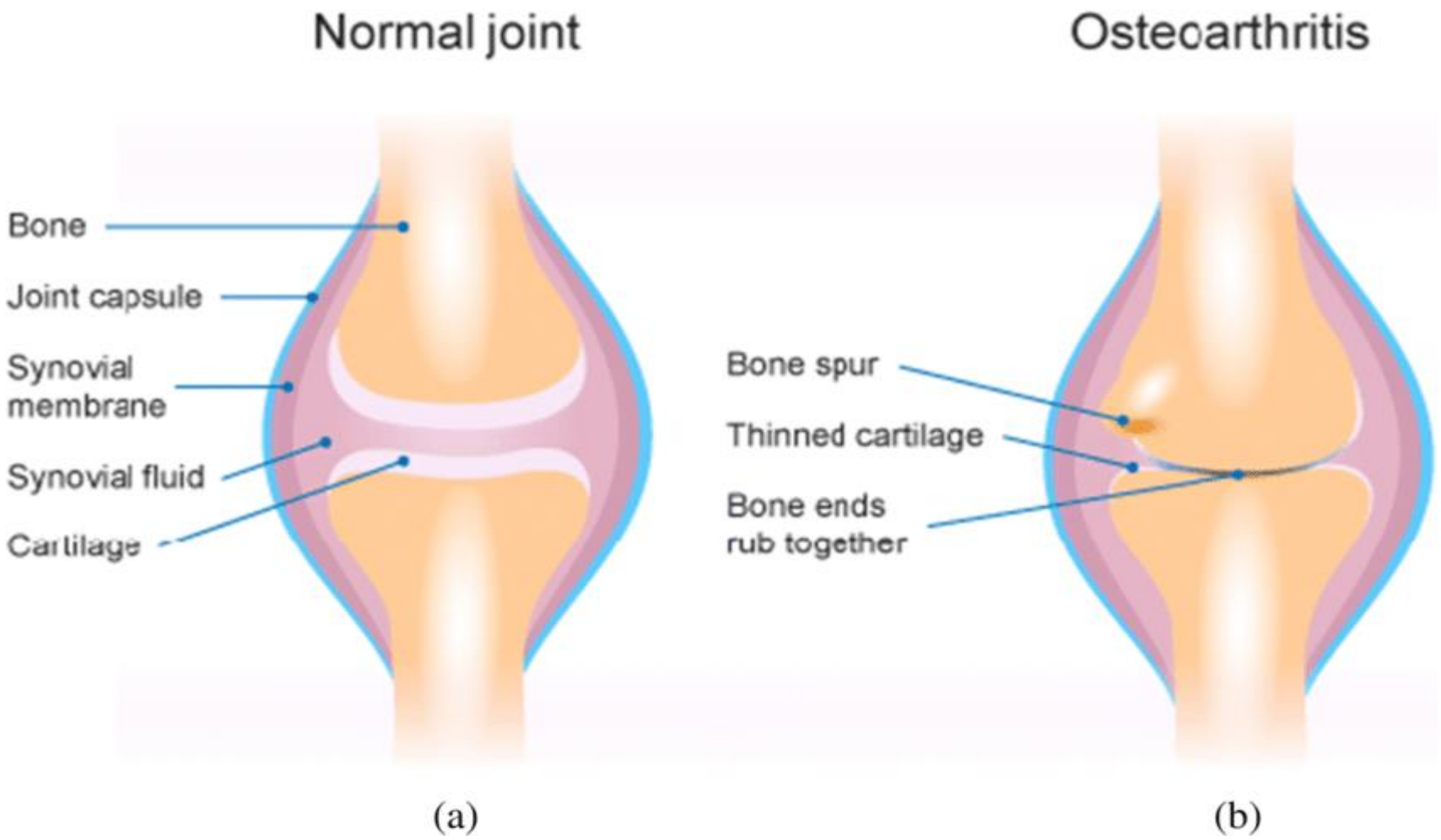
1-Degenerative Arthritis

Osteoarthritis (OA)

TYPES AND PATHOGENESIS OA occurs in 2 clinical forms:

Primary OA (osteoarthritis) occurs in the elderly, more commonly in women than in men. The process begins by the end of 4th decade and then progressively and steadily increases producing clinical symptoms. Probably, 1-wear and tear with repeated minor trauma, 2-heredity, 3-obesity and 4- ageing , all contribute to focal degenerative changes in the articular cartilage of the joints. Genetic factors favouring susceptibility to develop OA have been observed.

Secondary OA may appear at any age and is the result of any previous wear and tear phenomena involving the joint such as 1-previous injury, fracture, 2-inflammation, 2-loose bodies and 3-congenital dislocation of the hip.


Normal joint
Osteoarthritis
Bone
Joint capsule
Synovial membrane
Synovial fluid
Cartilage
Bone spur
Thinned cartilage
Bone ends rub together
(a)
(b)

MORPHOLOGIC FEATURES :

As mentioned above, the weight-bearing joints such as hips, knee and vertebrae are most commonly involved but interphalangeal joints of fingers may also be affected.

1. Articular cartilages , The regressive changes are most marked in the weight bearing regions of articular cartilages. Initially, there is loss of cartilaginous matrix . Further progression of the process causes loosening, flaking and fissuring of the articular cartilage resulting in breaking off of pieces of cartilage exposing subchondral bone.

2. Bone , The denuded subchondral bone appears like polished ivory. There is death of superficial osteocytes and increased osteoclastic activity causing rarefaction, microcyst formation and occasionally microfractures of the subjacent bone. The margins of the joints respond to cartilage damage by osteophyte or spur formation. Loosened and fragmented osteophytes may form free loose bodies.

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3. Synovium , Initially, there are no pathologic changes in the synovium but in advanced cases there is low-grade chronic synovitis and villous hypertrophy.

The manifestations of OA are most conspicuous in large joints such as hips, knee and back. In symptomatic cases, clinical manifestations are joint stiffness, diminished mobility, discomfort and pain.

The difference between O.A and R.A

	Osteoarthritis	Rheumatoid arthritis
Primary pathogenic abnormality	**Mechanical injury to articular cartilage**	**Autoimmunity**
Joints involved	**Primarily weight bearing (knees, hips)**	**Often begins with small joints of fingers ; progression leads to involvement of multiple joints**
Pathology	**Cartilage degeneration and fragmentation, bone spurs, subchondral cysts; minimal inflammation**	**inflammatory pannus invading and destroying cartilage; severe chronic inflammation; joint fusion (ankylosis)**
Serum antibodies	**None**	**Various, including ACPA, rheumatoid factor**
Involvement of other organs	**No**	**Yes (lungs, heart, other organs)**

Gout:

is marked by transient attacks of acute arthritis initiated by urate crystals deposited within and around joints. Gout, whether primary (90% of cases) or secondary to underlying disease, is characterized by abnormally high levels of uric acid in tissues and body fluids.

Pathogenesis: Hyperuricemia (plasma urate level above 6.8 mg/dL) is necessary, but not sufficient, for the development of gout. Elevated uric acid can result from overproduction, reduced excretion, or both

In primary gout, elevated uric acid most commonly results from reduced excretion, the basis of which is unknown in most patients.

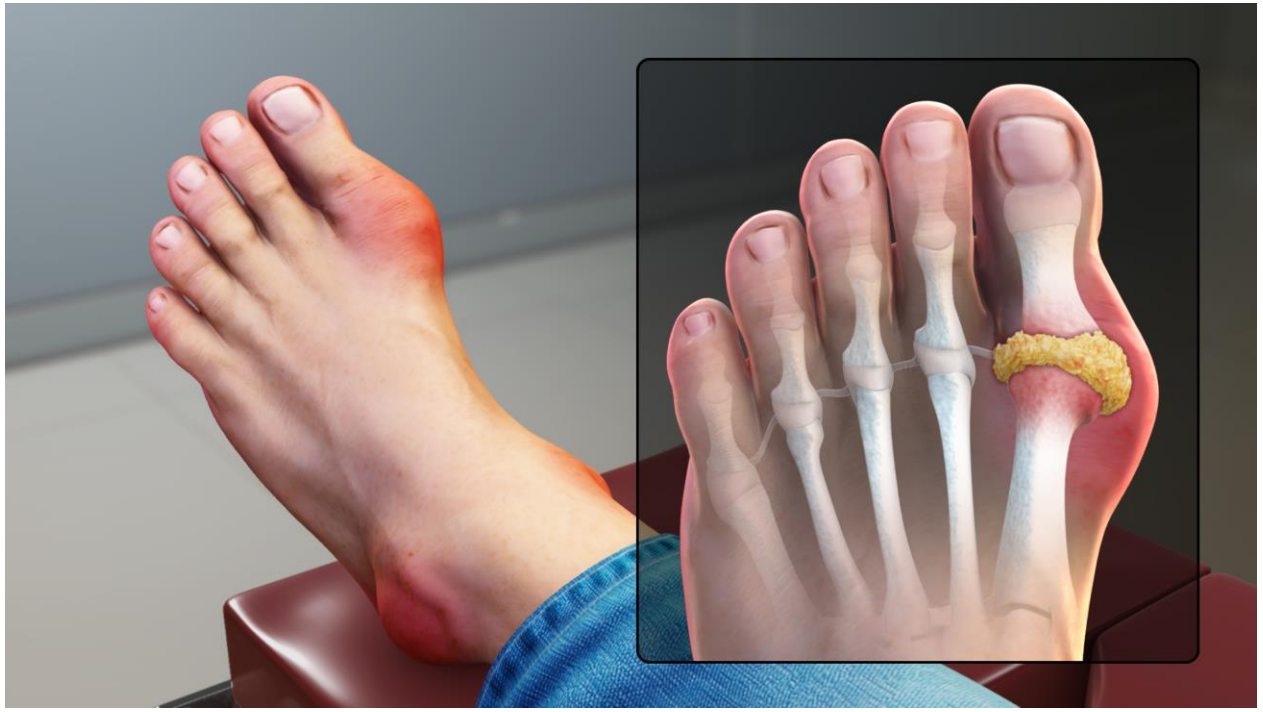
Secondary gout, is associated with medications or conditions that cause hyperuricemia

. In a small minority of cases primary gout is caused by uric acid overproduction as a result of enzymatic defects

The arthritis in gout is triggered by precipitation of urate crystals in the joints, stimulating the production of mediators that recruit leukocytes . Resident macrophages in the synovium phagocytose the crystals, Only about 10% of patients with hyperuricemia develop gout.

Other factors that contribute to the development of symptomatic gout include:

- Age of the individual and duration of the hyperuricemia. Gout usually appears after 20 to 30 years of hyperuricemia
- Genetic predisposition.
- Alcohol consumption
- Drugs (e.g., thiazides) that reduce excretion of urate



MORPHOLOGY:

Acute gouty arthritis is characterized by an intense inflammatory infiltrate rich in neutrophils that permeates the synovium and synovial fluid. Urate crystals are frequently found in the cytoplasm of the neutrophils in aspirated joint fluid and are arranged in small clusters in the synovium. Tophi in the articular cartilage, ligaments, tendons, and bursae are pathognomonic of gout. They are formed by large aggregates of urate crystals surrounded by an intense foreign body giant cell reaction .

Gouty nephropathy refers to renal complications (e.g., uric acid nephrolithias [kidney stones], pyelonephritis) caused by the deposition of urate crystals or tophi in the renal medullary interstitium or tubules.

Clinical Features. Four clinical stages are recognized:

- Asymptomatic hyperuricemia begins around puberty in men and after menopause in women.
- Acute arthritis presents with sudden onset, excruciating joint pain, localized hyperemia, and warmth. Most first attacks are monoarticular and 50% occur in the metatarsophalangeal joint of the big toe.
- Asymptomatic intercritical period is the symptom-free interval following resolution of acute arthritis.
- Chronic tophaceous gout develops on average about 10 years after the initial acute attack .

Treatment

Treatment of gout aims at lifestyle modification (e.g., weight loss, alcohol reduction, dietary changes to reduce purine intake) and medication to reduce inflammation (e.g., NSAIDs, colchicine) and lower serum urate levels (e.g., xanthine oxidase inhibitors). Uricosuric drugs that increase renal uric acid excretion can also be used.

Generally, gout does not shorten life span but can significantly affect quality of life.

Calcium Pyrophosphate Crystal Deposition Disease (Pseudogout) :

Calcium pyrophosphate crystal deposition disease (CPPD), also known as pseudogout, usually occurs in individuals older than 50 years and becomes more common with increasing age.

CPPD is divided into sporadic (idiopathic), hereditary, and secondary types. An autosomal dominant variant caused by germline mutations in the pyrophosphate transport channel results in crystal deposition and arthritis relatively early in life. Various disorders, including

1- previous joint damage,
2- hyperparathyroidism,
3-hemochromatosis,
4- hypothyroidism,
4-and diabetes, predispose to secondary CPPD.

MORPHOLOGY: The crystals first develop in the articular cartilage, menisci, and intervertebral discs, and as the deposits enlarge they may rupture and seed the joint.

Clinical Features.

CPPD is frequently asymptomatic. However, it may produce acute, subacute, or chronic arthritis that can be confused clinically with OA or RA. The joint involvement may last from several days to weeks and may be monoarticular or polyarticular; .

Ultimately, approximately 50% of affected individuals experience significant joint damage.

Therapy is supportive: there is no known treatment that prevents or slows crystal formation

Infectious Arthritis

Joints can become infected from hematogenous dissemination, from direct inoculation through the skin, or from contiguous spread from a soft tissue 7 abscess or osteomyelitis. Infectious arthritis is potentially serious because it can cause rapid, permanent joint destruction

Suppurative Arthritis

Bacteria that cause acute suppurative arthritis usually enter the joints by hematogenous spread. As with osteomyelitis, the etiologic agent depends on the anatomic location and clinical setting (e.g., trauma, intravenous drug use). Staphylococcus aureus is the most common pathogen in adults and children; in neonates, group B Streptococcus may be responsible. Infection with gram-negative bacilli and Pseudomonas is generally seen in patients who are immunocompromised and in people who use intravenous drugs .

The classic presentation is the sudden development of an acutely painful, warm, and swollen joint with a restricted range of motion. Fever, leukocytosis, and elevated C-reactive protein are common. The infection usually involves only a single joint, most commonly the knee, hip, shoulder and elbow . The axial joints are more often involved in individuals who inject drugs. Joint aspiration is diagnostic if it yields purulent fluid in which the causative agent can be identified.

Prompt recognition and effective antimicrobial therapy can prevent joint destruction.