Pathology Team

Non-infectious arthritis

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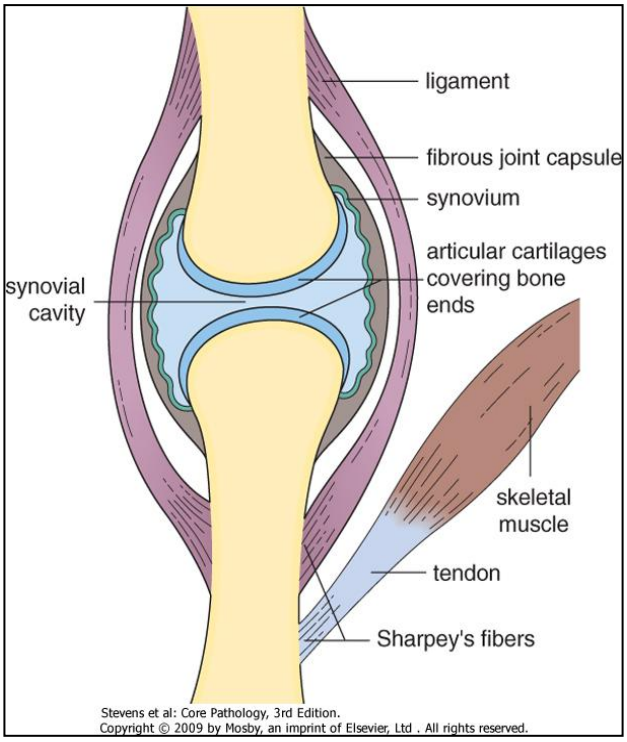
Bone Pathology

JOINTS/TYPES



Synovial joints

*Also called diarthroses.

*Joint space bone ends are covered by hyaline cartilage.

*Strengthened by dense fibrous capsule.

*This is reinforced by ligaments and muscles.

* The presence of joint space allows wide range of motion.

Non-synovial joints:

*Also called solid joint or synarthrosis.

*No joint space present.

*Provides structural integrity and minimal movement.

Arthritis

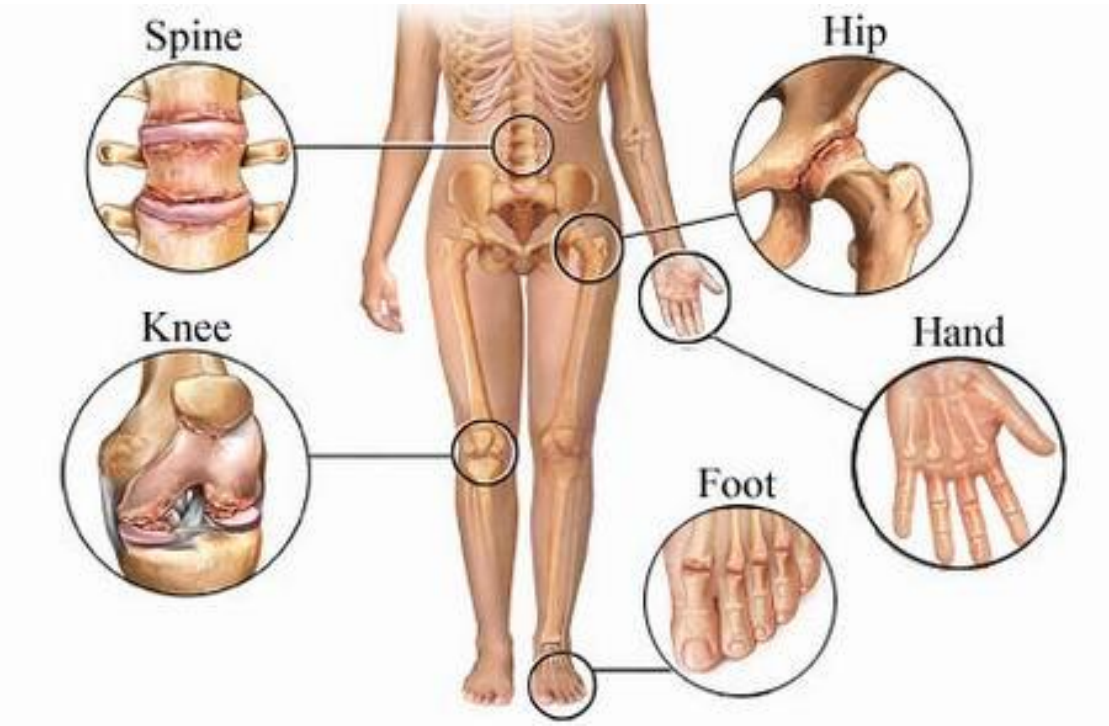
- INFECTOUS (SUPPURATIVE) ARTHRITIS.
- TUBERCULOUS ARTHRITIS.
- OSTEOARTHRITIS.
- RHEUMATOID ARTHRITIS.

degenerative joint disease

(osteoarthritis)

- Non-neoplastic disorder of progressive erosion of articular cartilage associated with aging, trauma, occupational injury.
- Usually age 50+ years (present in 80% at age 65 years).
- Cartilage degradation may be mediated by IL-1.

Osteoarthritis Common site



usually one joint or same joint bilaterally

Gender has some influence; knees and hands are more commonly affected in women.

whereas hips are more commonly affected in men.

Osteoarthritis

Types

Primary osteoarthritis:

- Appears insidiously with age and without apparent initiating cause.
- Usually affecting only a few joints.

Secondary osteoarthritis:

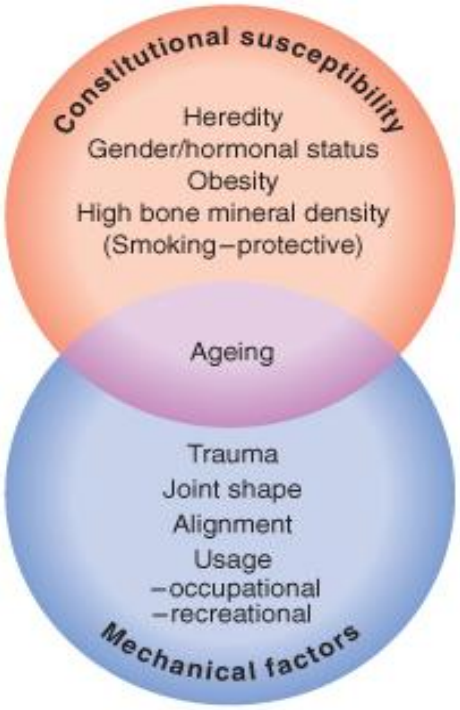
- Some predisposing condition, such as previous traumatic injury, developmental deformity, or underlying systemic disease such as diabetes, ochronosis, hemochromatosis, or marked obesity.
- Secondary osteoarthritis affect young.
- Often involves one or several predisposed joints.
- Less than 5% of cases.

Note :

1- Hemochromatosis=abnormal metabolism of iron.

2- Ochronosis is the syndrome caused by the accumulation of homogentisic acid in connective tissues.

Risk factors for the development of osteoarthritis:



Osteoarthritis

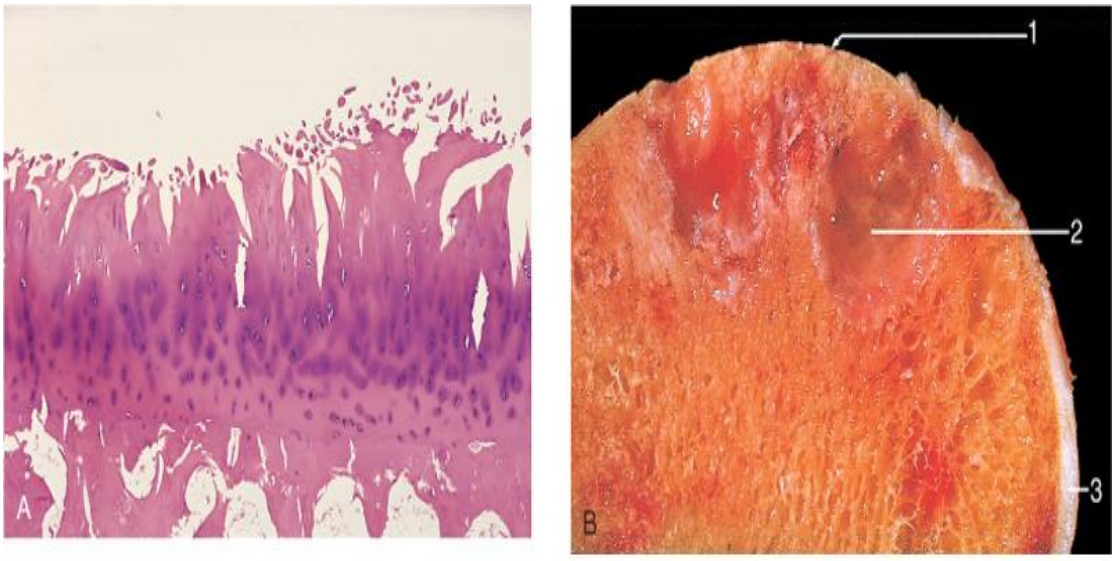
Gross:

Early changes are even degeneration of hyaline cartilage of articular surface, with fibrillation of cartilaginous matrix and possible cartilage fragmentation.

Later thinning of cartilage and overgrowth of apposing joint surface; articular surface is often soft and granular with altered shape, sloughing of cartilage.

cysts: **(synovial fluid forced into fractures via ball valve-like mechanism)**

osteophytes: **(bony outgrowths at margins of articular surface)**

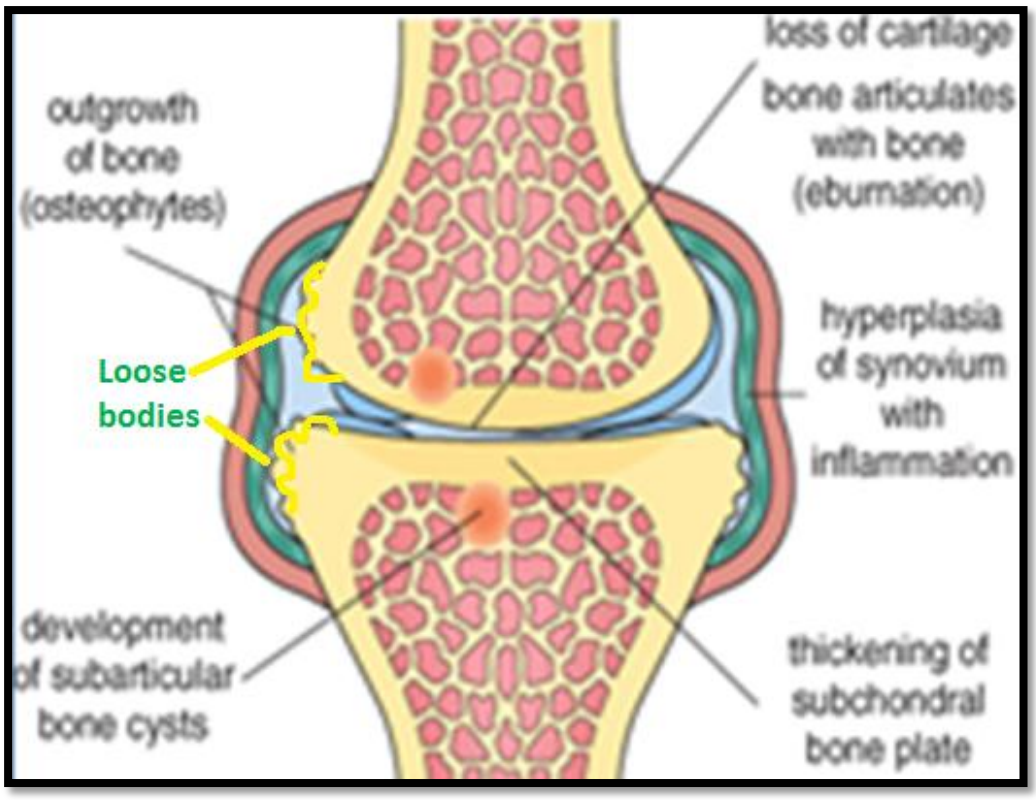


Note :

1- The articular surface became smoother due to friction.

2- Cystic formation occurs in the subchondral layer, filled with synovial fluid.

3- Normal articular cartilage.



Loose bodies: may form if portion of articular cartilage breaks off; normally loose body is nourished by synovium and continues to grow.

Note :
They are the broken pieces of hyaline cartilage found in the synovial cavity, and as they nourish by synovial fluid they increase in size and reduce joint movement.

Pathogenesis

The early changes:

- **Destruction of articular cartilage, which becomes eroded, splits (fibrillation), and leads to narrowing of the joint space on radiography.**
- **There is inflammation and thickening of the joint capsule and synovium.**

Micro:

- **Ghost chondrocytes (no nuclei) or necrotic chondrocytes.**
- **Marked irregularity, thinning.**
- **Fragmentation and fibrillation of thinned cartilage.**
- **Subchondral cysts with mucoid fluid surrounded by sclerotic bone.**
- **Mushroom-shaped osteophytes (bony outgrowths) develop at the margins of the articular surface.**

Usually no significant inflammatory component.

Symptoms:

Pain worse with use of joint, crepitus, limited range of motion, nerve root compression; Heberden nodes in fingers of women only (osteophytes at DIP joints).

Note :
Crepitus =crackling joint sound.
DIP =distal interphalangeal joint.
As osteophytes projrct outward, they press on nerves, then nerve root compression occurs.
Fever never associate with osteoarthritis.

Rheumatoid arthritis

- Chronic systemic inflammatory disorder affecting synovial lining of joints, bursae and tendon sheaths; also skin, blood vessels, heart, lungs, muscles.
- Produces nonsuppurative proliferative synovitis, may progress to destruction of articular cartilage and joint ankylosis.
- 1% of adults, 75% are women, peaks at ages 10-29 years; also menopausal women.

Sites:

Small bones of hand affected first (MCP, PIP joints of hands and feet), then wrist, elbow, knee.

Note :
Nonsuppurative=pertaining to inflammation without the production of pus.
Ankylosis=stiffness of a joint due to abnormal adhesion and rigidity of the bones of the joint, which may be the result of injury or disease.

Pathophysiology:

- Triggered by exposure of immunogenetically susceptible host to arthitogenic microbial antigen.
- Autoimmune reaction then occurs with T helper activation and release of inflammatory mediators and cytokines that destroys joints.
- Circulating immune complexes deposit in cartilage, activate complement, cause cartilage damage.
- Parvovirus B19 may be important in pathogenesis.

Note :
Autoreactive T-cells → release cytokines → deposition on joints → destroy joints.

Genetics:

HLA-DR4, DR1 (65%).

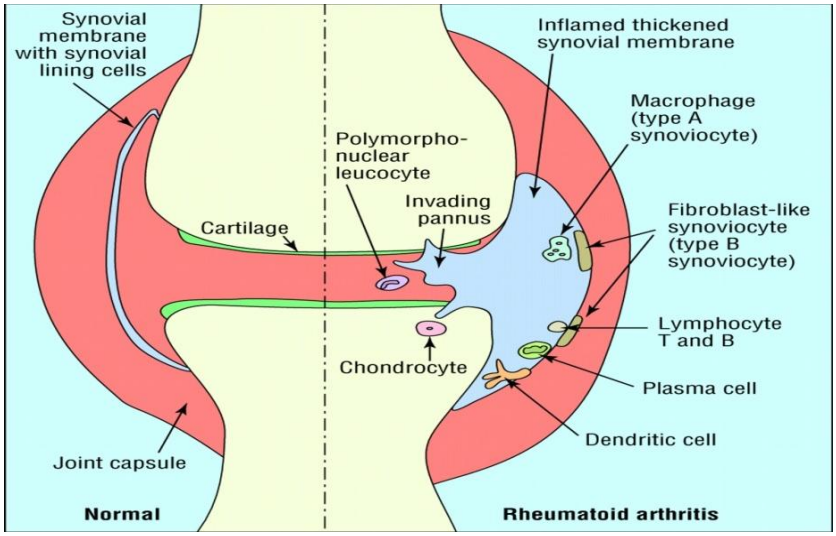
Laboratory:

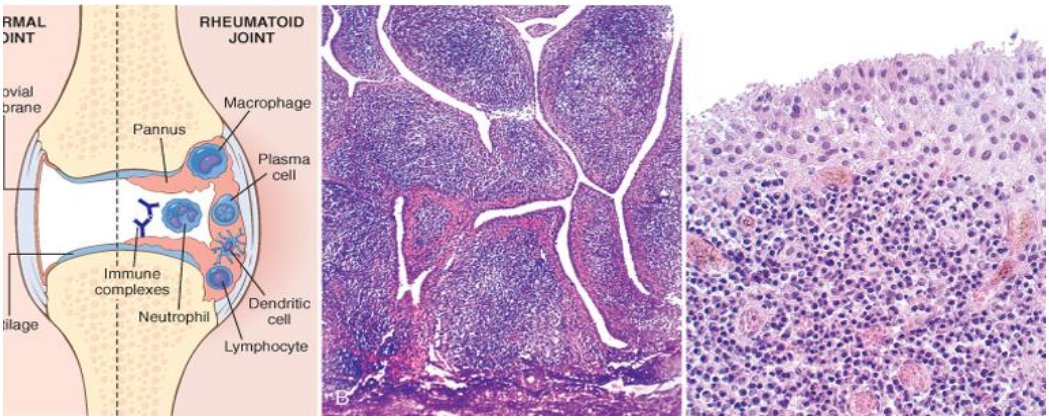
- 80% have IgM autoantibodies to Fc portion of IgG (rheumatoid factor), which is not sensitive or specific.
- Synovial fluid has increased neutrophils (particularly in acute stage), protein, low mucin.
- Other antibodies include antikeratin antibody (specific, not sensitive), antiperinuclear factor, anti-rheumatoid arthritis associated nuclear antigen (RANA).

Note :
HLA=human leukocyte antigen.
IgM attacks IgG in rheumatoid arthritis.
Rheumatoid factor=serum IgM autoantibodies indecates rheumatoid arthritis .

Gross:

joints have edematous, thick, hyperplastic synovium(increased cells count of synovial membrane), covered by delicate and bulbous fronds.





Note the increased number of lymphocytes and giant cell formation

Just for understanding :

Osteoarthritis is the most common type of arthritis. On the other hand, rheumatoid arthritisis recognized as the most crippling or disabling type of arthritis. Osteoarthritis (also referred to as degenerative joint disease) is caused by the breakdown of joint cartilage. Cartilage loss can cause bone to rub on bone in a joint - a condition that is very painful. Usually osteoarthritis begins in a single joint.

Diagnosis:

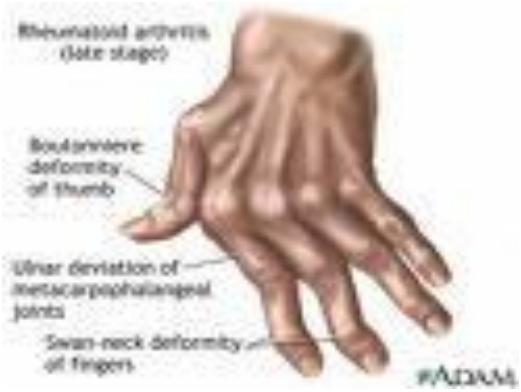
- morning stiffness, arthritis in 3+ joint areas
- arthritis in hand joints,
- symmetric arthritis,
- rheumatoid nodules, rheumatoid factor, typical radiographic changes

Note:
Symmetric arthritis=affect the same joint on both sides of the body.

X-ray:

Joint effusions, juxta-articular osteopenia (erosions in the bone near the articular surface) , erosions.

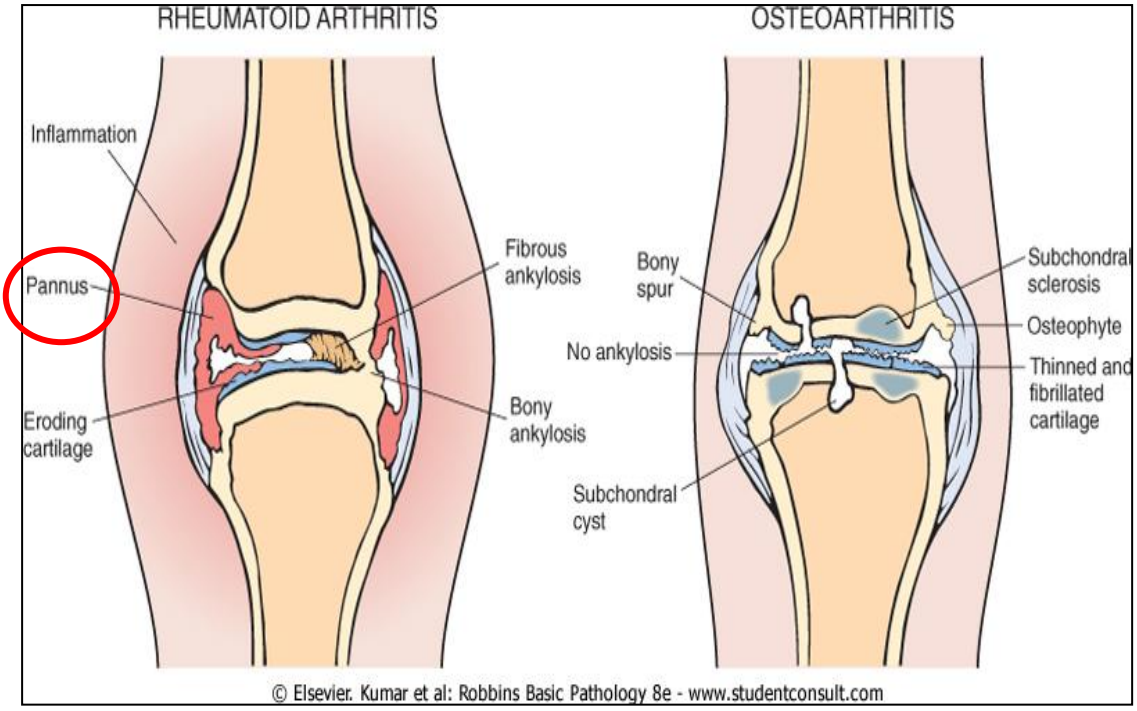
Narrowing of joint space; destruction of tendons, ligaments and joint capsules produce radial deviation of wrist, ulnar deviation of digits, swan neck finger abnormalities.



Clinical course:

- Variable; malaise, fatigue, musculoskeletal pain, then joint involvement.
- Joints are warm, swollen, painful, stiff in morning; 10% have acute onset of severe symptoms, but usually joint involvement occurs over months to years; 50% have spinal involvement.
- Reduces life expectancy by 3-7 years, death due to amyloidosis, vasculitis, GI bleeds from NSAIDS, infections from steroids.

Note:
Malaise=feeling of general discomfort.



Note:
Pennus=membrane of granulation tissue covering the normal surface of the articular cartilages.

GOUT

Gout and gouty arthritis

Transient attacks of acute arthritis initiated by crystallization of urates and neutrophils infiltration, followed by chronic gouty arthritis with tophi in joints and urate nephropathy.

Causes : 2-5% of chronic joint disease

Sites: 50% have initial attack in first metatarsophalangeal joint; also ankles, heels, knees, wrists, fingers, elbows.

Note:
Tophi=irregular shaped crystal of uric acid appear in the affected joint.

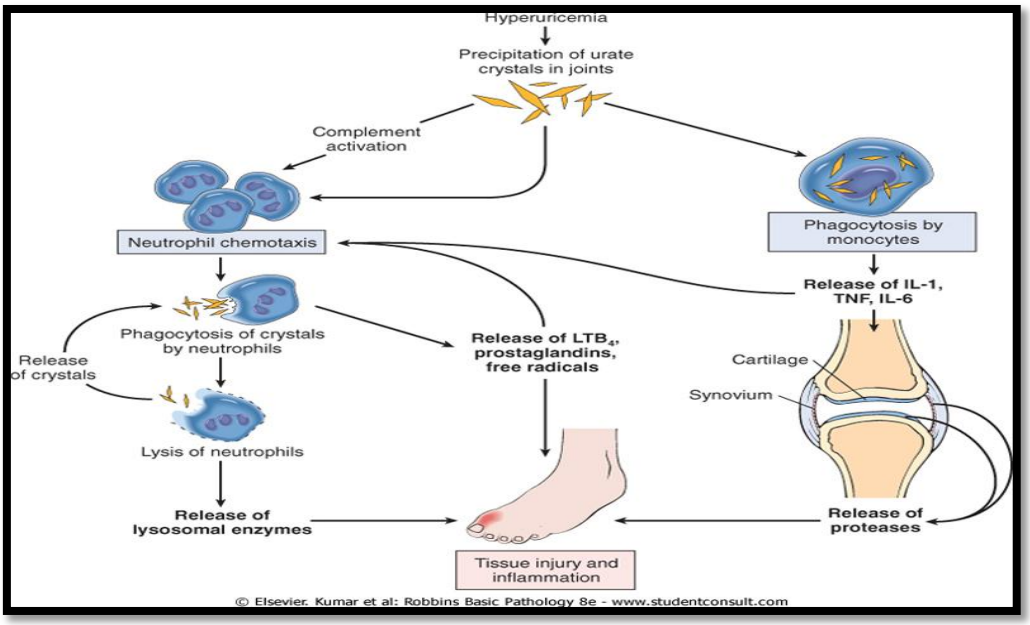
Types of Gout

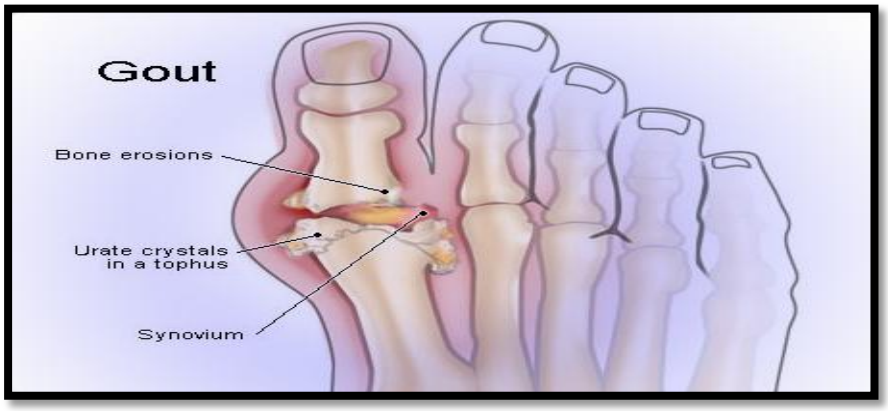
1. ***Primary gout (90%):*** **idiopathic (85%) with overproduction of uric acid or known enzyme defects (partial hypoxanthine-guanine phosphoribosyl transferase deficiency [HGPRT]).**
2. ***Secondary gout (10%):*** **increased nucleic acid turnover due to leukemia/lymphoma, chronic renal disease, HGPRT deficiency.**

- **Deposition of monosodium urate crystals in joints and viscera and uric acid kidney stone formation.**
- **Need serum urate > 7 mg/dl for deposition.**
- **Risk factors for gout with hyperuricemia are age > 30 years, familial history of gout, alcohol use, obesity, thiazide administration, lead.**

Gout arthritis:

- **Synovial fluid is poorer solvent for sodium urate than plasma, uric acid accumulation occurs, so it occurs with hyperuricemia.**
- **Crystals develop in synovial lining cells, stimulate formation of antibodies, complement, generates c3a, c5a, attracts more neutrophils.**
- **Releases free radicals, releases lysosomal enzymes which eventually causes acute arthritis that last days to weeks without treatment.**
- **Ankle joint is the most affected.**





- **Repeated attacks of acute arthritis cause chronic arthritis and formation of tophi in synovial membranes and periarticular tissue, which eventually damages joints.**
- **Aspirate: grossly white-gray and granular; strongly birefringent needle-shaped crystals under polarized light; foreign body giant cells.**
- **With chronic disease, urate deposits may be present in soft tissue, ligaments, skin.**
- **Gouty deposits may be surrounded by fibrous tissue and be rimmed by histiocytes and giant cells.**

Pseudogout

- also known as chondrocalcinosis.
- calcium pyrophosphate crystal deposition disease.
- most probably they involve enzymes that produce or degrade pyrophosphate, resulting in its accumulation and eventual crystallization with calcium.
- age 50 or older.
- Site: the knees, followed by the wrists, elbows, shoulders, and ankles, are most commonly affected.
- no known treatment prevents or retards crystal formation.