PATHOLOGY TEAM

MED 444

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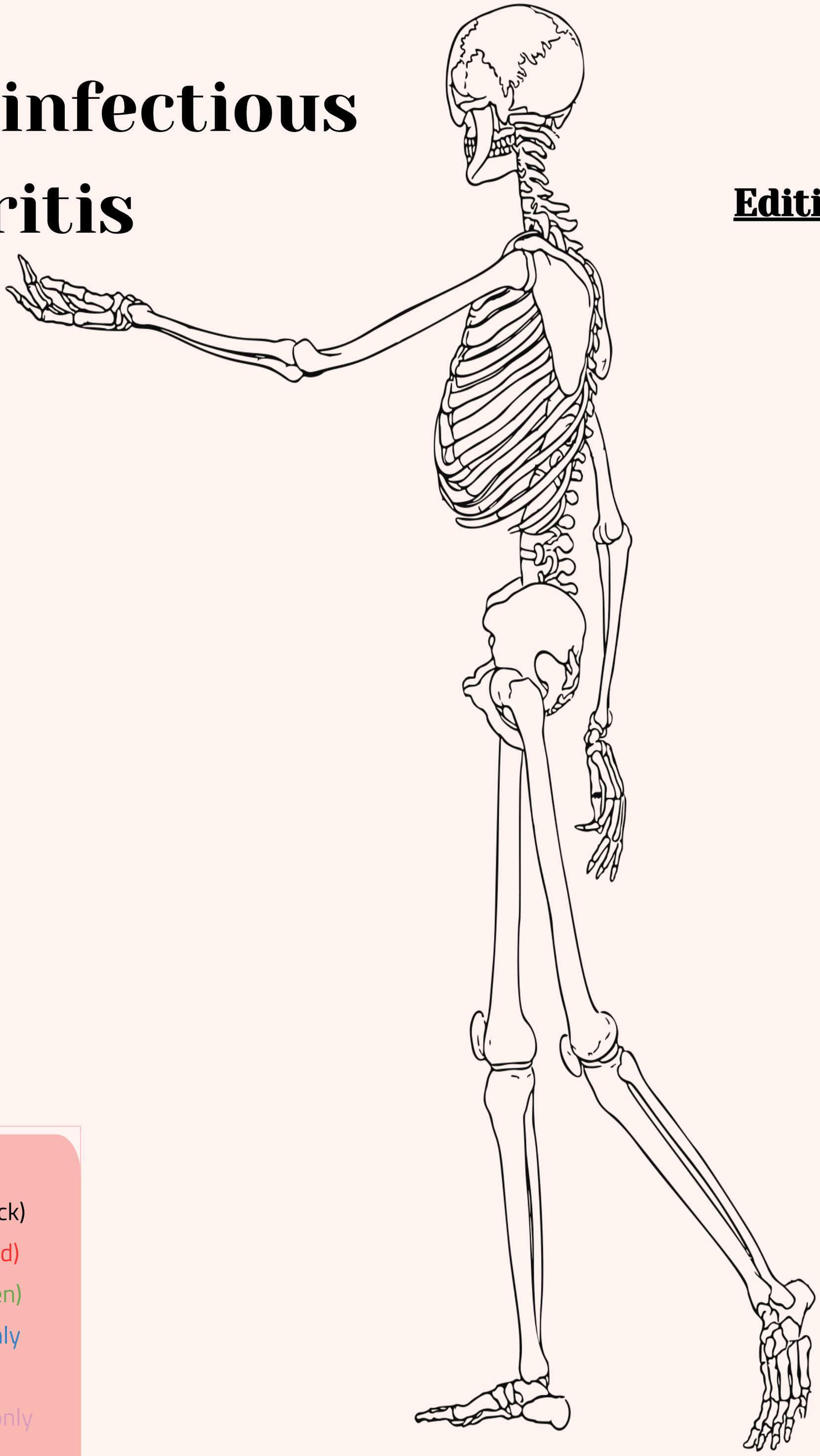
Non-infectious arthritis



Editing File

Color index:

Main text (black)

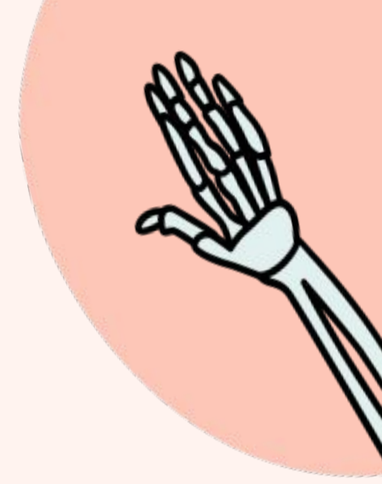
Important (Red)

Dr.Notes (green)

Male slides only (blue)

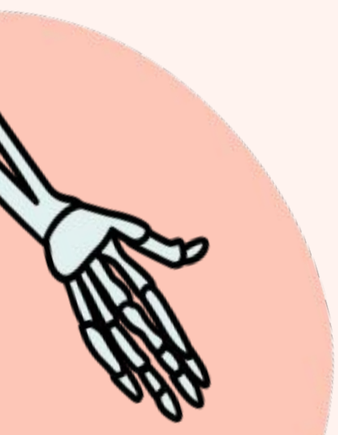
Female slides only (pink)

Extra info(gray)



Objectives

- To know the primary articular defect in osteoarthritis, (degenerative joint disease), its pathogenesis, morphology, major joints affected and the clinical course.
- Understand the etiology, pathogenesis, morphology major joints affected, serology and the clinical course of rheumatoid arthritis.
- Be aware of Gout and gouty arthritis, its pathogenesis, morphology of acute and chronic lesions and its clinical course.



Joint types

- Synovial (diarthroses)
- Non-Synovial (solid joints or synarthrosis)

Inflammatory disease of joints (arthritis and synovitis)

Causes

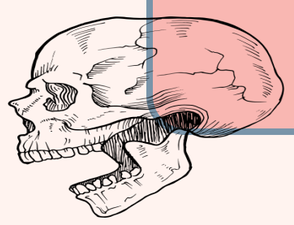
- **degeneration** → **osteoarthritis**
- **Autoimmunity** → **rheumatoid arthritis**
  - SLE
  - rheumatic fever
- **crystal deposition** → **gout and other crystalline arthropathies**
- **infection**
  - septic arthritis
  - tuberculous arthritis

In female slides

Clinical manifestations of joint diseases

1. Joint Pain (Arthralgia)
2. Joint Swelling
3. Joint Crepitus
   - A clinical sign characterized by a peculiar crackling, crinkly, or grating feeling or sound in the joints. It indicates cartilage wear in the joint space
4. Abnormal Joint Mobility

Osteoarthritis



[Helpful Video]

Highly recommended

Definition

- a non-neoplastic disorder of progressive degeneration of cartilage results in structural and functional failure of synovial joints .

- most common disease of joints, and important degenerative disease, with both destructive and reparative components.

- Intrinsic disorder of cartilage in which chondrocytes respond to biochemical and mechanical stresses resulting in the breakdown of the matrix and failure of its repair.

Incidence

- Usually age 50+ years (pressacent in 80% at age 65 years).

Aetiology

the main factors in the development of osteoarthritis are:

1. aging
2. abnormal load on joints
3. crystal deposition
4. inflammation of joints

Pathogenesis

In general, osteoarthritis affects joints that are constantly exposed to wear and tear. It is an important component of occupational joint disease.

e.g. osteoarthritis of:

a. the fingers in typists.
b. the knee in professional footballers

Results from degeneration of the articular cartilage and its disordered repair.

Normal articular cartilage performs two functions:

A. Provides friction-free movement within the joint with the help of the synovial fluid.

B. In weight-bearing joints, it spreads the load across the joint surface.

These functions require the cartilage to be elastic (i.e. to regain normal architecture after compression) and to have high tensile strength.

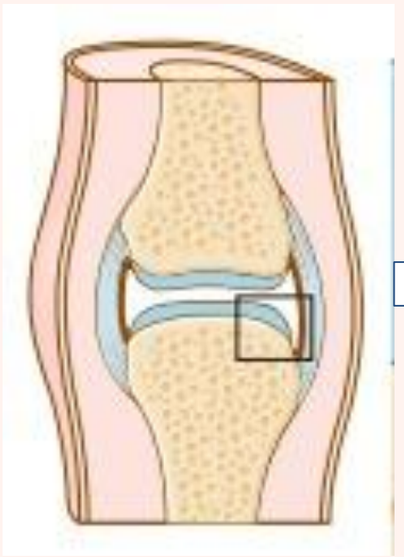
These attributes are provided by proteoglycans and type II collagen, both produced by chondrocytes.

chondrocyte function is affected by a variety of influences: mechanical stresses ,aging and Genetic factors.

Imbalance in the expression, activity, and signaling of cytokines and growth factors that results in degradation and loss of matrix.

The type II collagen network also is diminished as a result of decreased local synthesis and increased breakdown.

Pathogenesis Cont.



The changes to chondrocytes can be divided into three phases:

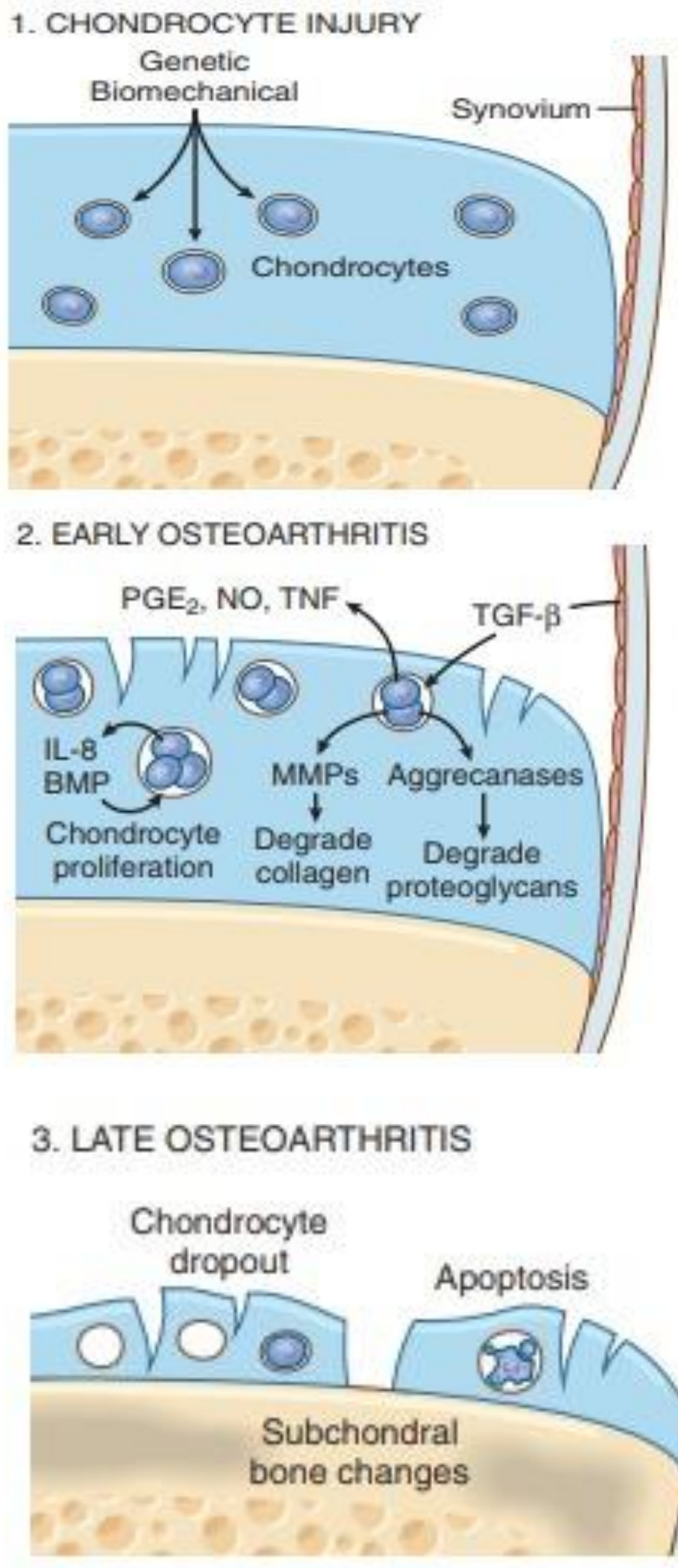
1-Chondrocyte injury, related to genetic and biochemical factors.

2-Early OA:

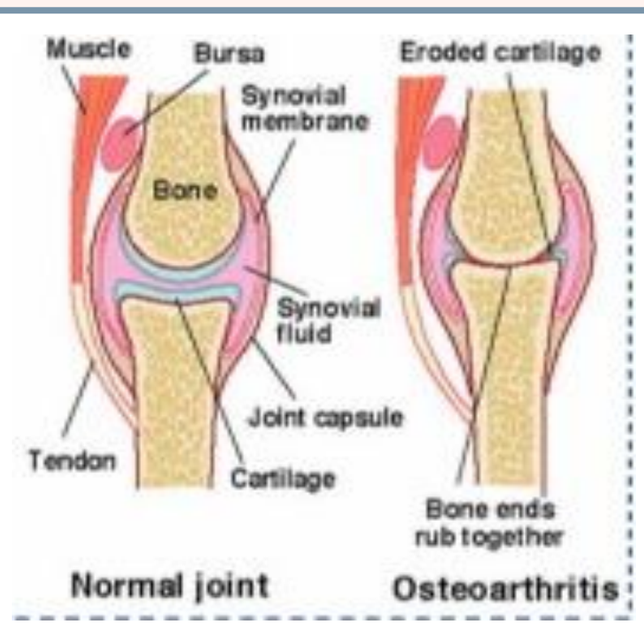
- **Chondrocytes proliferate and secrete inflammatory mediators, collagens, proteoglycans, and proteases.**
- **Result in remodeling of the cartilaginous matrix and initiate secondary inflammatory changes in the synovium and subchondral bone.**

3-Late OA:

- **Repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive subchondral bone changes.**



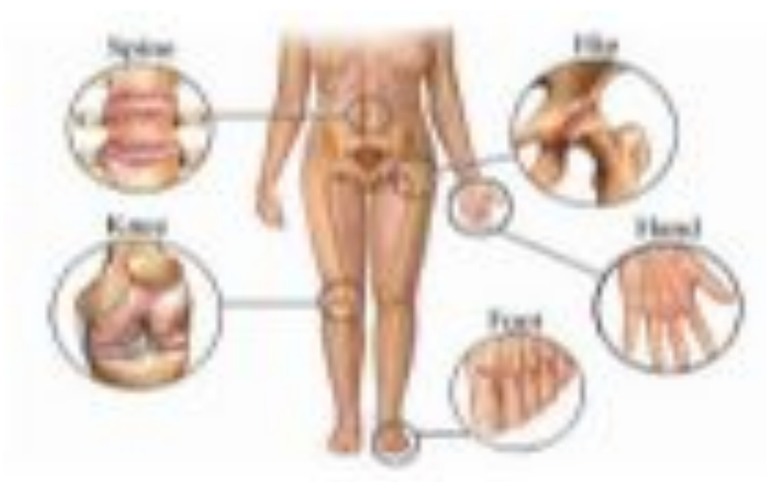
- **The early change: destruction of articular cartilage, which splits (fibrillation), becomes eroded, and leads to narrowing of the joint space on X ray.**
- **There is inflammation and thickening of the joint capsule and synovium.**



Common sites

- usually one joint or same joint bilaterally
- Gender has some influence:
  - Women : knees and hands
  - Men : hips

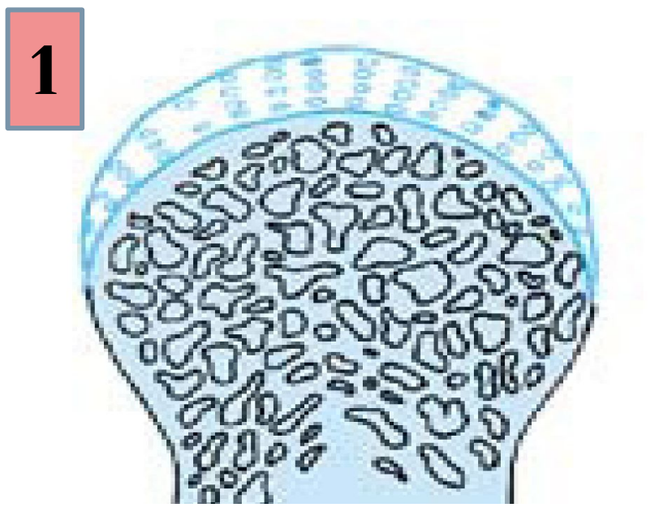
Pathological changes involves:
cartilage , bone , synovium , joint capsule
with secondary effects on muscle(atrophy).

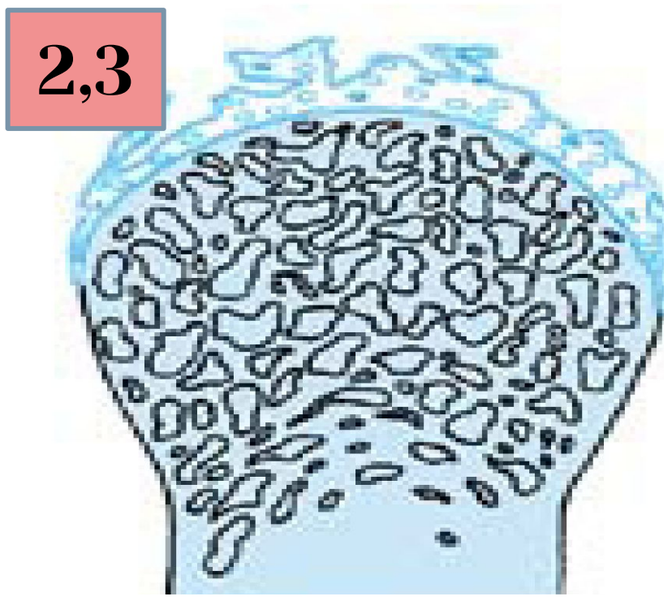


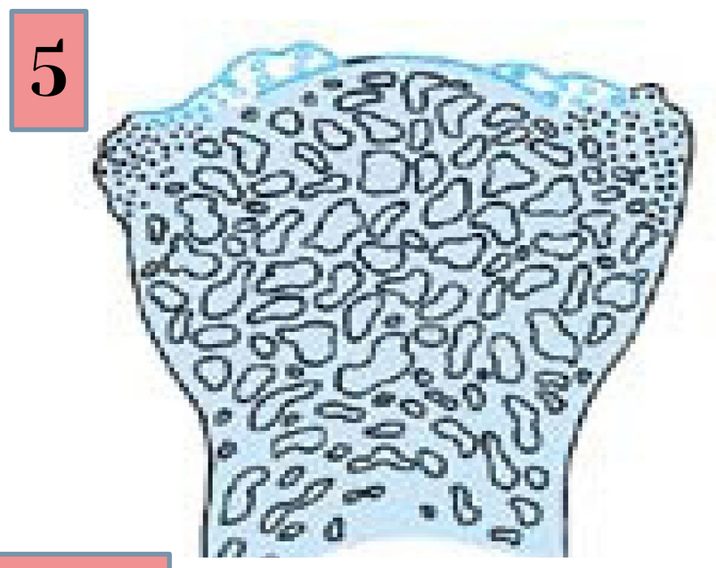
Morphologic changes/pathogenesis

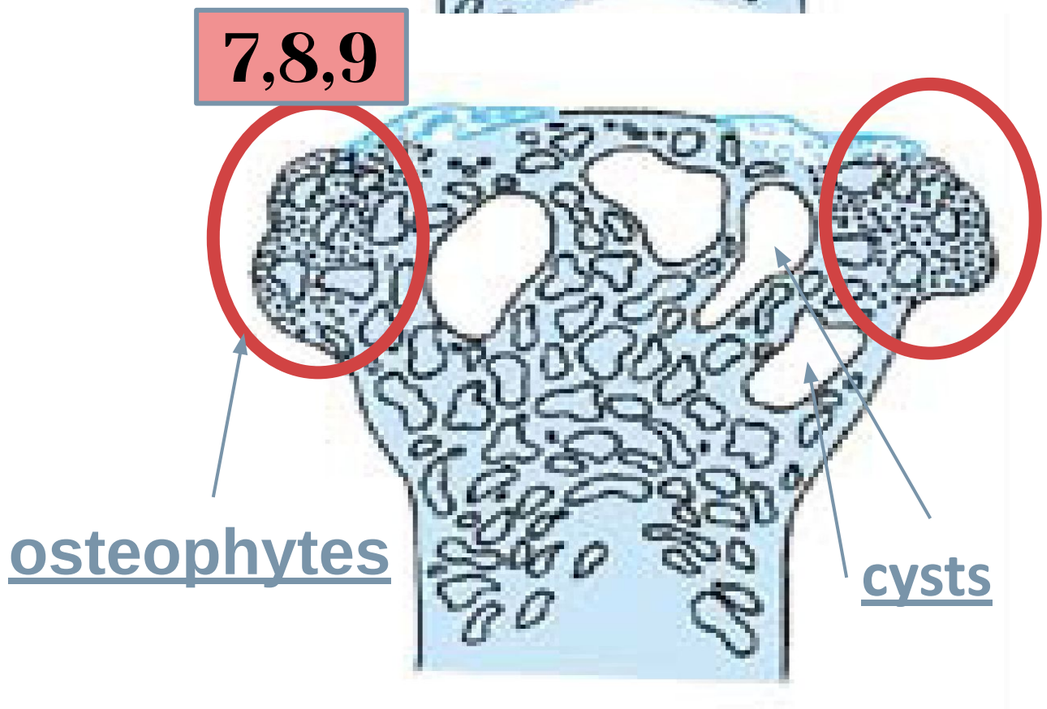
point :2,5,6,7,8 from male slides
point:1,3,4,9 from female slides

1. Normal articular cartilage.
2. Fibrillation and cracking of the matrix occur as the superficial layers of the cartilage are degraded.
3. fragmentation of articular surface and thinning of cartilage.
4. Calcification of cartilage margins.
5. Eventually, full-thickness portions of the cartilage are lost, and the subchondral bone plate is exposed and is smoothened by friction, giving it the appearance of polished ivory, constant friction of bone surfaces with polished bone (bone eburnation).
6. Small fractures can dislodge pieces of cartilage and subchondral bone into the joint, forming loose bodies.
7. The fracture gaps allow synovial fluid to be forced into the subchondral regions to form fibrous walled cysts, of underlying bone (small cyst).
8. formation of lips of new bone Mushroom-shaped <u>osteophytes</u> (bony outgrowths) develop at the margins of the articular surface.
9. extensive loss of cartilage cystic degeneration of underlying bone.









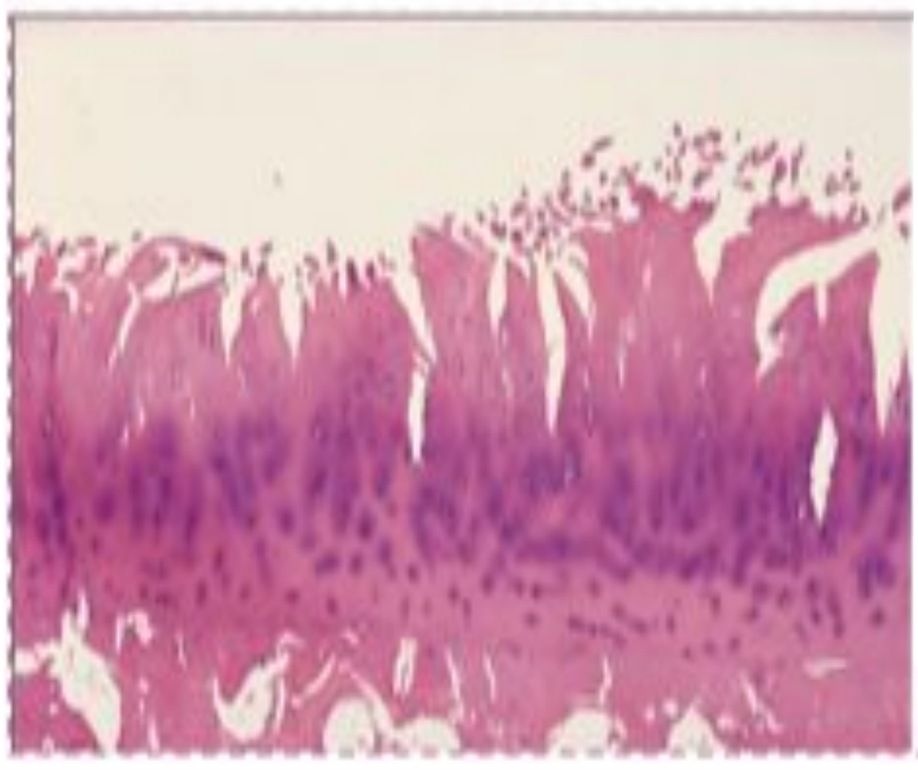
Types

Primary (idiopathic):

- **appears insidiously with age and without apparent initiating cause**
- **usually affecting only a few joints (oligoarticular)**

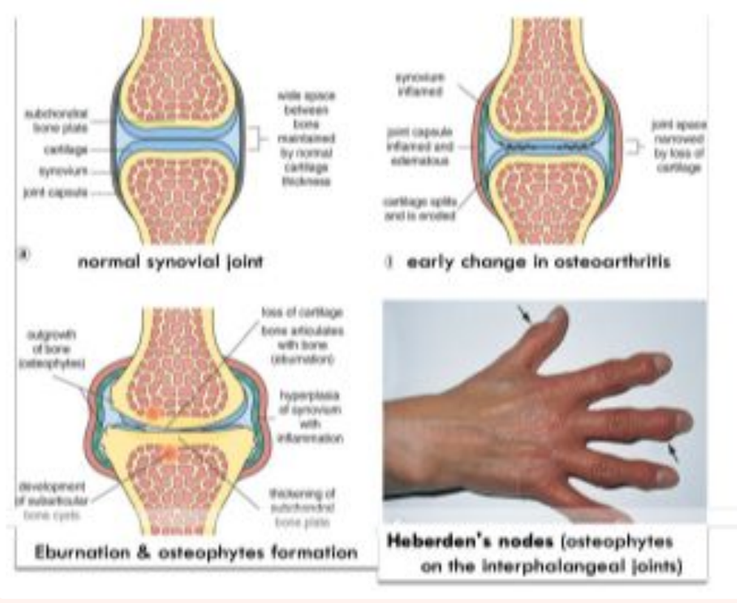
Secondary: in younger age group

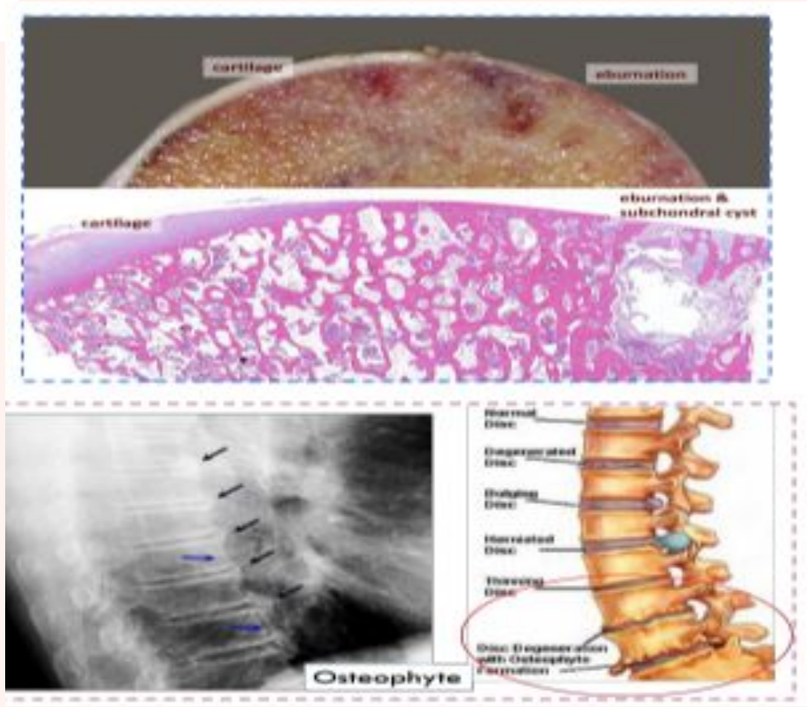
- **Some predisposing condition, such as previous traumatic injury, developmental deformity (anomaly), or underlying systemic disease such as diabetes, ochronosis, hemochromatosis, or marked obesity, SCD.**
- **Secondary osteoarthritis usually affect young**
- **Often involves one or several predisposed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



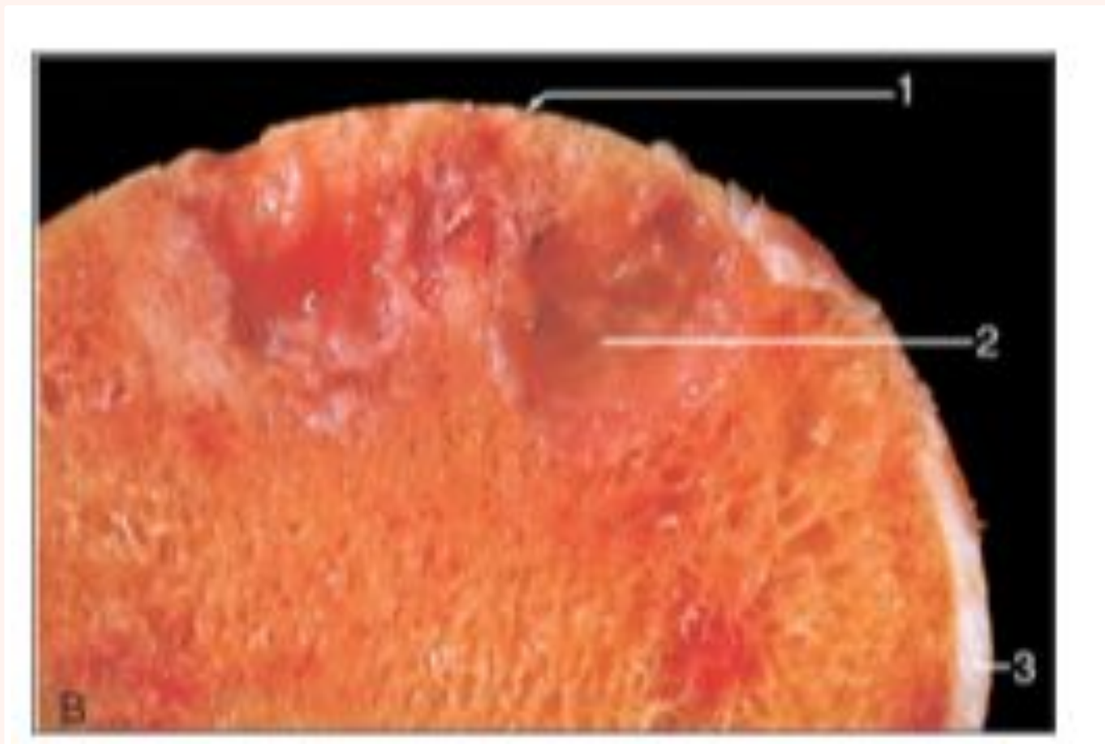
Osteoarthritis: Histologic demonstration of the characteristic fibrillation of the articular cartilage.

Cracking and fibrillation of cartilage.





1. Eburnated articular surface exposing subchondral bone
2. Subchondral cyst
3. Residual articular cartilage



Clinical features/course

- An insidious disease predominantly affecting patients beginning in their 50s and 60s.
- Characteristic symptoms include deep, aching **pain exacerbated by use, morning stiffness and limited range of movement,** crepitus (grating or popping sensation in the joint).
- Swelling of affected joints
- **Osteophyte impingement on spinal foramina can cause nerve root compression with radicular pain,** muscle spasms, muscle atrophy, **and neurologic deficits.**
- Heberden nodes in fingers of women only (osteophytes at DIP joints).
- **Loose bodies: may form if portion of articular cartilage breaks off.**
- Usually only one or a few joints are involved
    - Common joints: hips, knees, lower lumbar and cervical vertebrae.
    - Proximal and distal interphalangeal joints of the fingers, first carpometacarpal joints, and first tarsometatarsal joints of the feet.

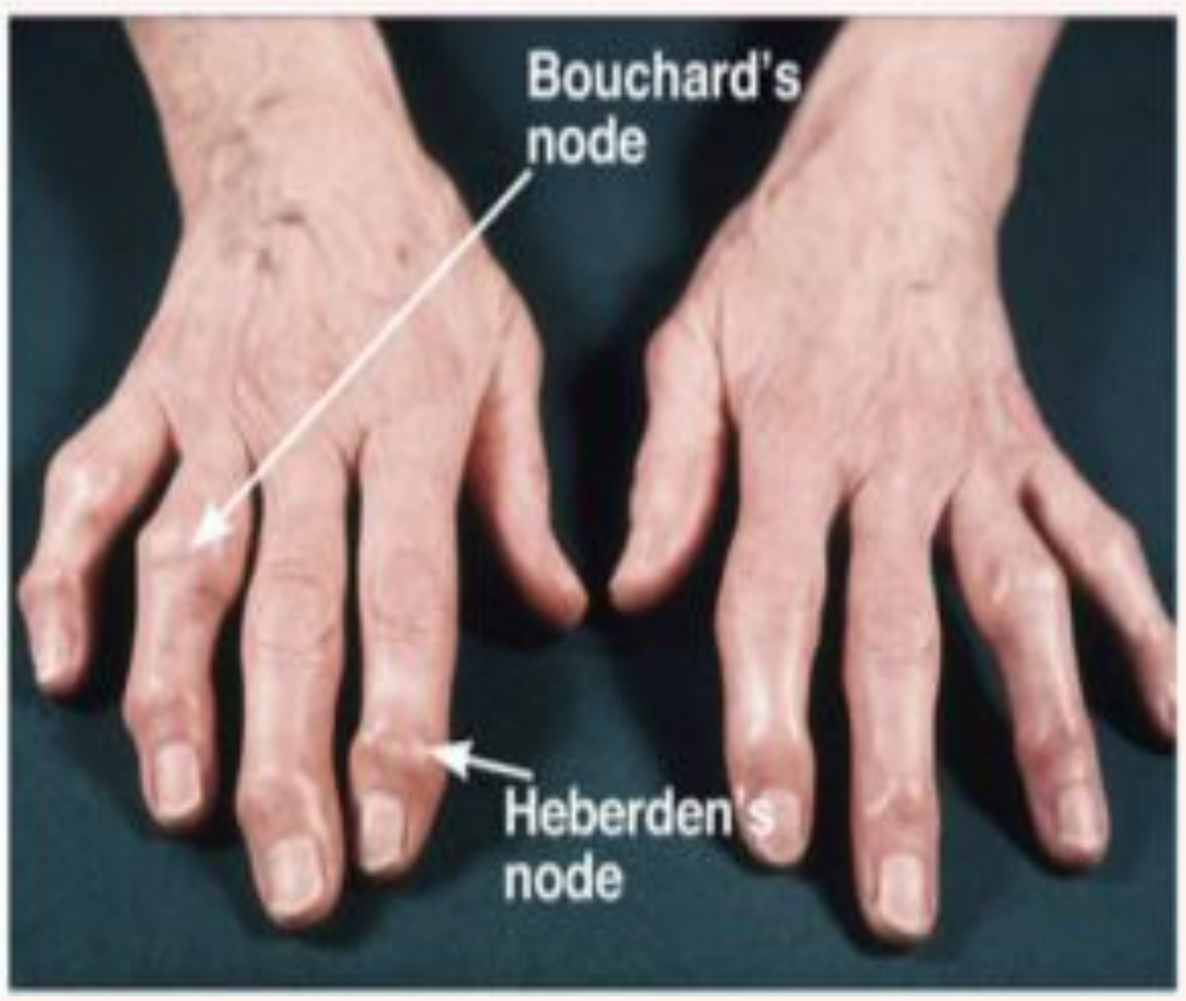
Prognosis

- **Osteoarthrosis is a slowly progressive,** chronic joint disability.
- With time, significant joint deformity can occur.
- Eventually, elderly sufferers may become confined to wheelchairs.
- Recent advancements in the technique of joint replacement with prostheses have improved the outlook of these patients.
- Treatment usually is based on symptoms, with joint replacement in severe cases
    - NSAIDs, (could cause throat pain)intra-articular injection, arthroplasty

Nodes

Heberden nodes

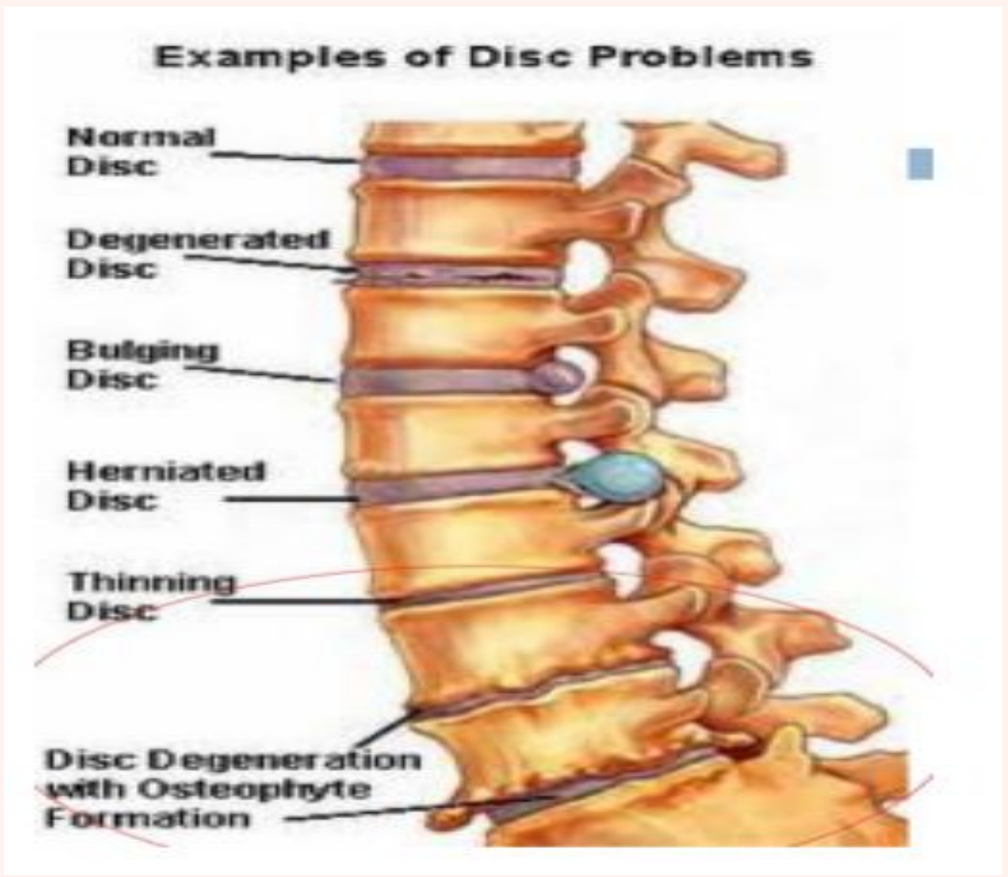
prominent osteophytes at the distal interphalangeal joint, are common in women

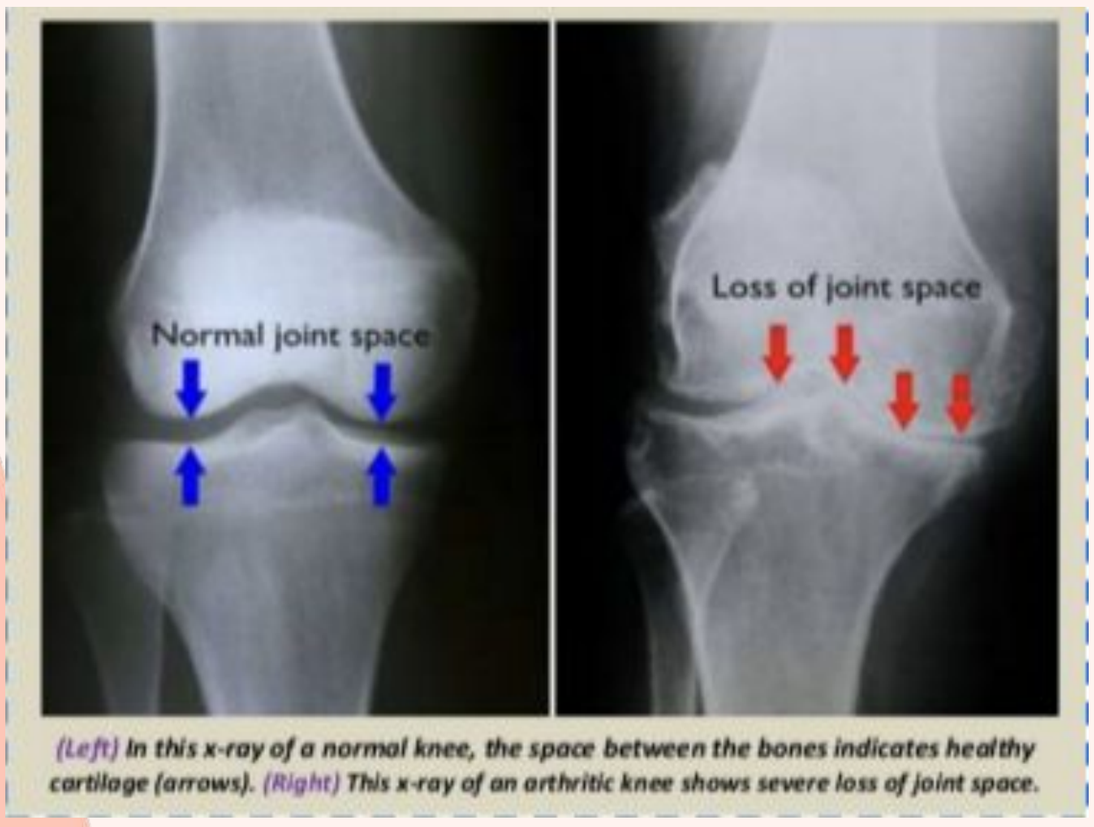


Bouchard node

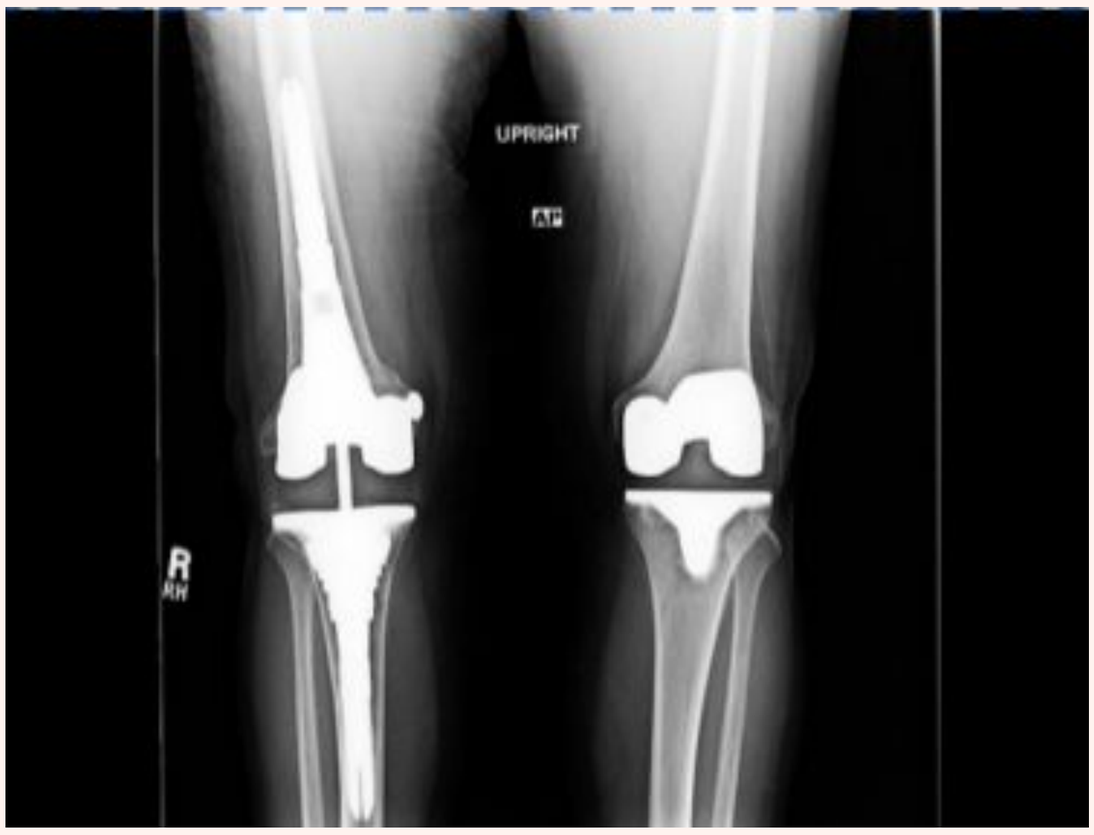
a similar swelling affecting the proximal interphalangeal joint

Bouchard=Proximal





(Left) In this x-ray of a normal knee, the space between the bones indicates healthy cartilage (arrows). (Right) This x-ray of an arthritic knee shows severe loss of joint space.



Summary

definition: a non-neoplastic disorder of progressive erosion (**degeneration**) of articular cartilage

Incidence:
common after 50 year

types:
- Primary
- Secondary

Pathogenesis: erosion of articular cartilage.

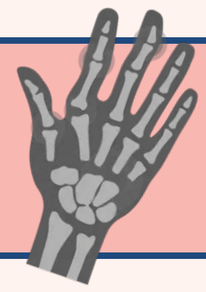
Clinical features:
pain and limitation of function.

Deep Focus Question

Which of the following is most characteristic of osteoarthritis?
A. Positive serology
B. Symmetric distribution
C. Normal X-rays in late disease
D. Wide distribution of joints
Answer: D

Special Thx to 443

Rheumatoid arthritis



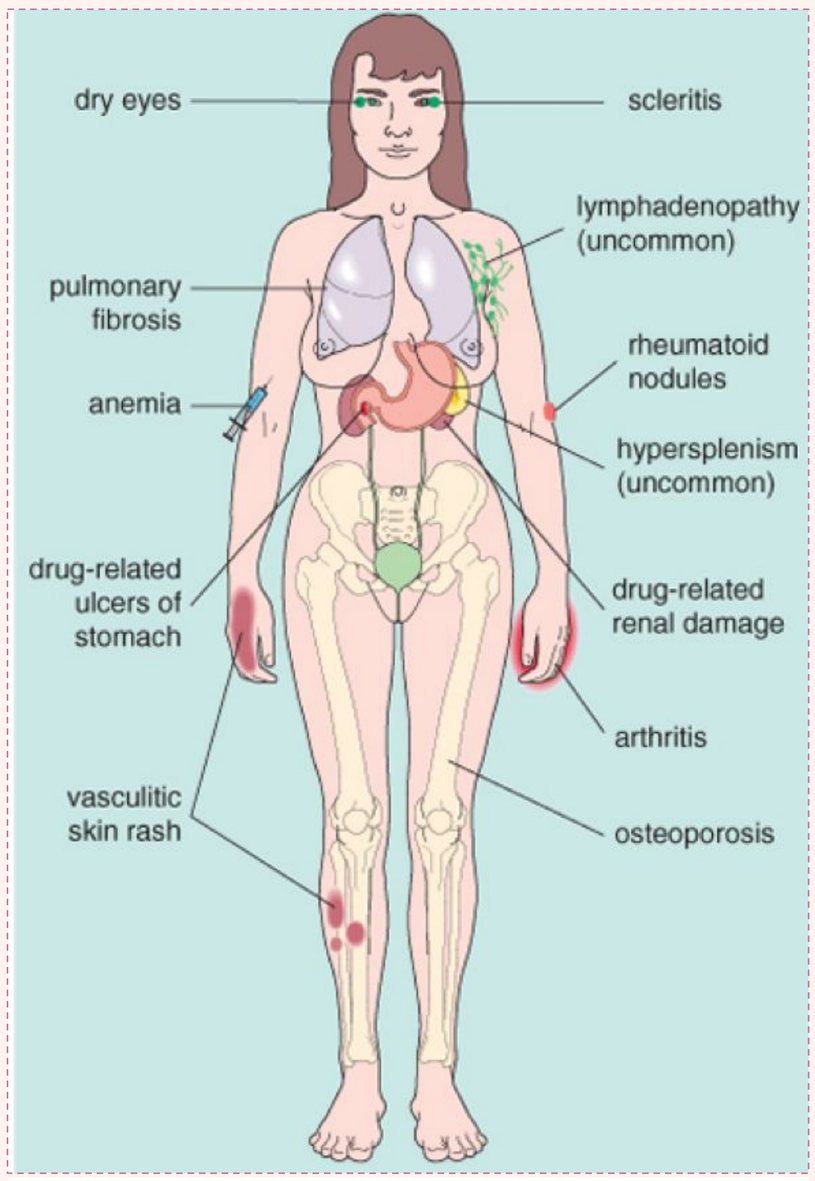
Helpful Video

DEFINITION

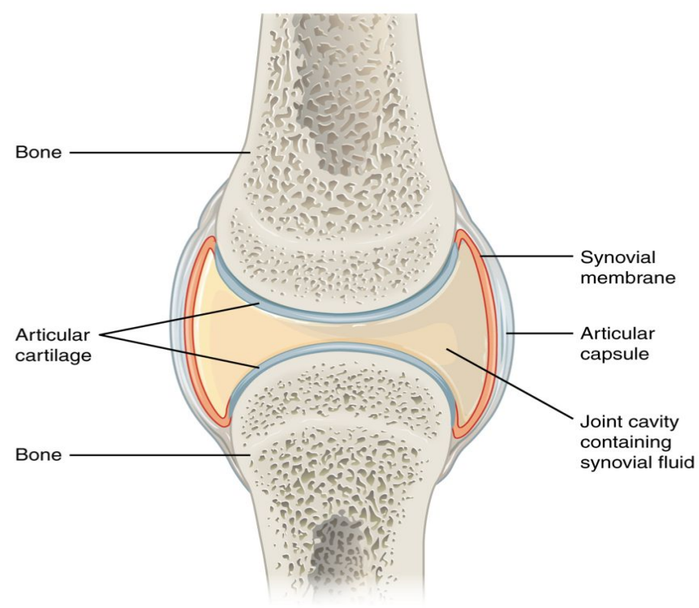
- Systemic, chronic inflammatory autoimmune disease affecting synovial lining of joints, bursa and tendon sheaths and many tissues(skin, heart, blood vessel, lung,muscles) but principally attacking the joints
- Cause (produces) a nonsuppurative proliferative synovitis that frequently progresses to destroy articular cartilage and joint ankylosis and underlying bone with resulting disabling arthritis and ankylosis (adhesions)

Incidents

1. Prevalence of approximately 1% of adults
2. Peak incidence is in the second to fourth decades of life
3. Three to five times more common in women than in men 75% are women, peaks at ages 10-29 years; also menopausal women



Extra-Articular Manifestations



Pathogenesis/Aetiology

1

The changes are caused mainly by cytokine-mediated inflammation, with CD4+ T helper cells being the principal source of the cytokines
autoimmune reaction then occurs with T helper activation
and release of inflammatory mediators, TNF and
cytokines, that destroys joints

2

Initiate the autoimmune response in RA by reacting with an arthritogenic agent, perhaps microbial or a self-antigen

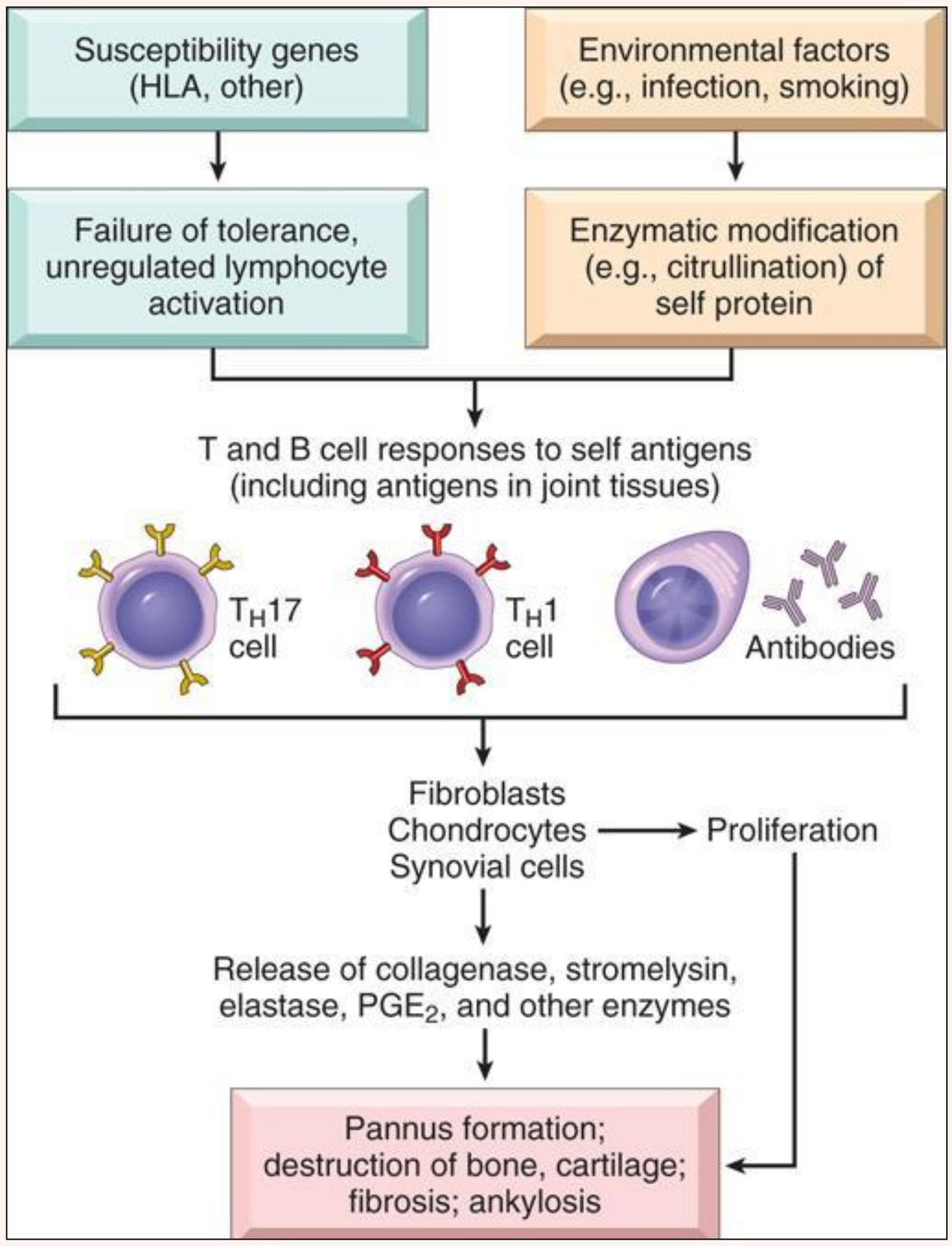
triggered by exposure of immunogenetically susceptible host to arthitogenic microbial antigen

3

Many patients produce antibodies against cyclic citrullinated peptides (CCPs)
- Antibodies to citrullinated fibrinogen, type II collagen, α-enolase, and vimentin are the
most important
- Form immune complexes that deposit in the joints

4

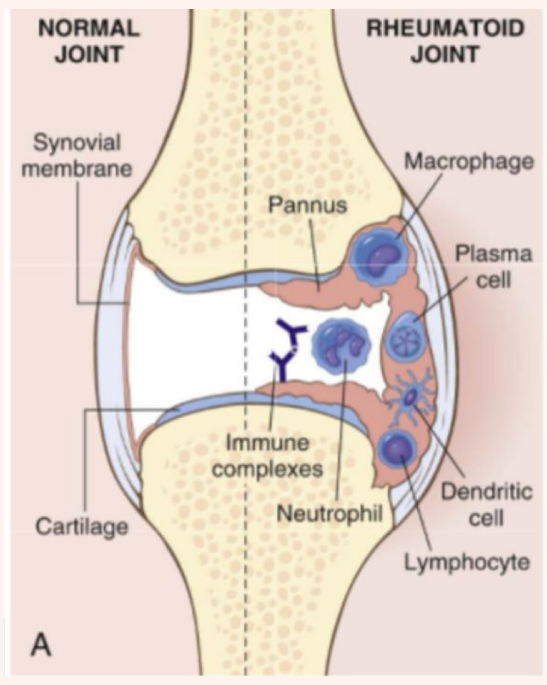
circulating immune complexes deposit in cartilage,
activate complement, cause cartilage damage
Parvovirus B19 may be important in pathogenesis

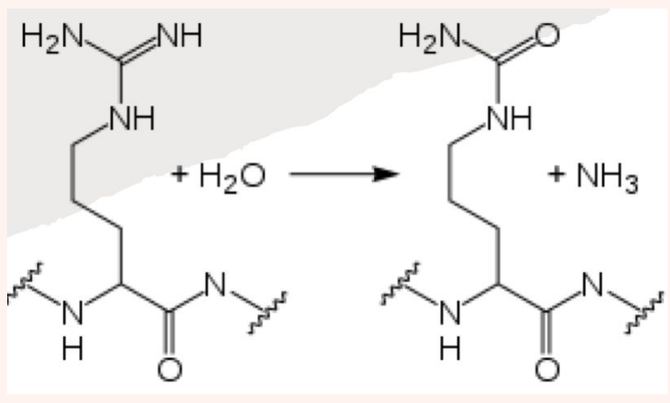


are derived from proteins in which arginine residues are converted to citrulline residues posttranslationally

Antibodies against cyclic citrullinated peptides (CCP protein antibodies) is the most specific for a diagnosis of rheumatoid arthritis

- These antibodies are a diagnostic marker for the disease and may be involved in tissue injury





Rheumatoid factor (autoantibodies)

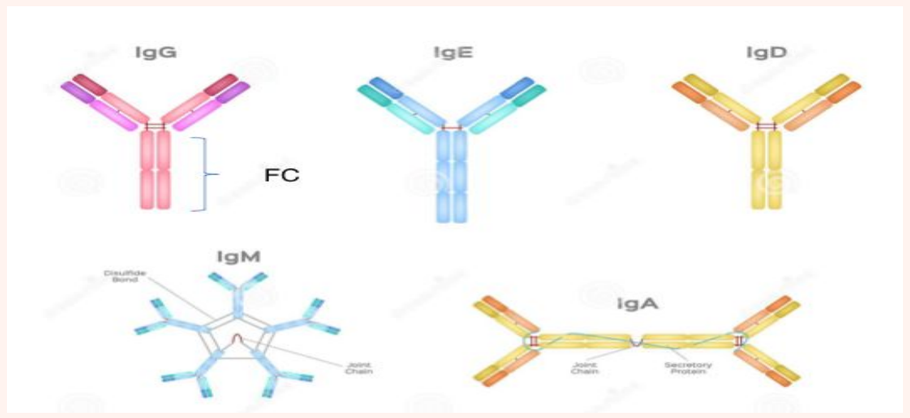
Males slides only

Can be found in patients without the disease

About 80% of patients have serum immunoglobulin M (IgM) or IgA autoantibodies that bind to the Fc portions of their own IgG

They may form immune complexes with self- IgG that deposit in joints and other tissues, leading to inflammation and tissue damage

They are not uniformly present in all patients with RA and also can be found in patients without the disease



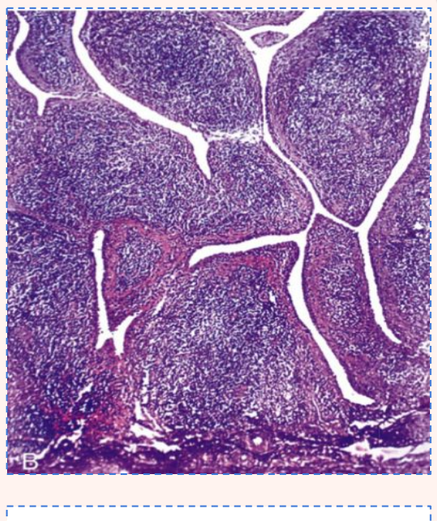
Morphology

- Typically manifests as a symmetric arthritis principally affecting the small joints of the hand and feet
- The characteristic histologic features include

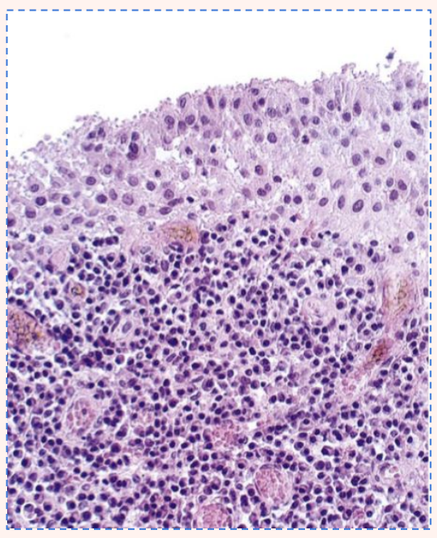
1. Synovial cell hyperplasia and proliferation
2. Dense perivascular inflammatory cell infiltrates (frequently forming lymphoid follicles) in the synovium composed of CD4+ T cells, plasma cells, and macrophages
3. Increased vascularity due to angiogenesis
4. Increased osteoclast activity in the underlying bone - bone erosion.
5. Osteoclastic activity in underlying bone, allowing the synovium to penetrate into the bone and cause periarticular erosions and subchondral cysts
6. Fibrin exudate on the synovial and joint surfaces
7. Neutrophils and aggregates of organizing fibrin on the synovial surface

Pannus

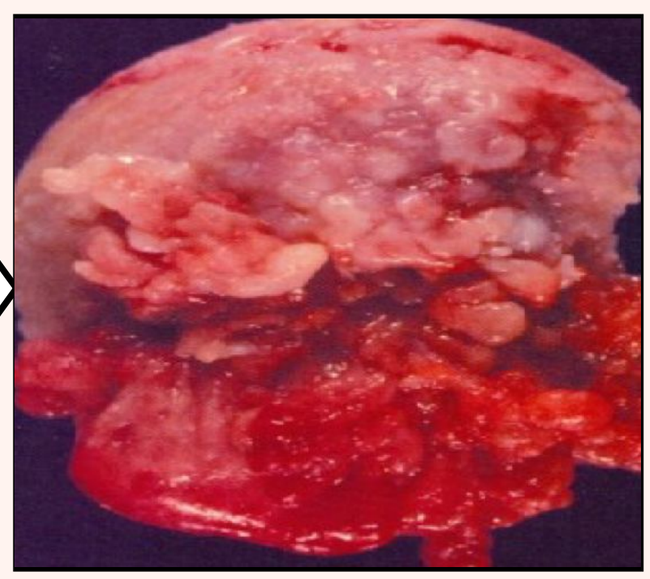
a mass of edematous synovium,formed by proliferating synovial-lining cells admixed with inflammatory cells, granulation tissue, and fibroblast fibrous connective tissue that grows over the articular cartilage and causes its erosion



- Eventually the pannus fills the joint space, In time, after the cartilage has been destroyed ,the pannus bridges the opposing bones to form a fibrous ankylosis, which eventually ossifies and results in fusion of the bones, called bony ankylosis (subsequent fibrosis and calcification may cause permanent ankylosis.)

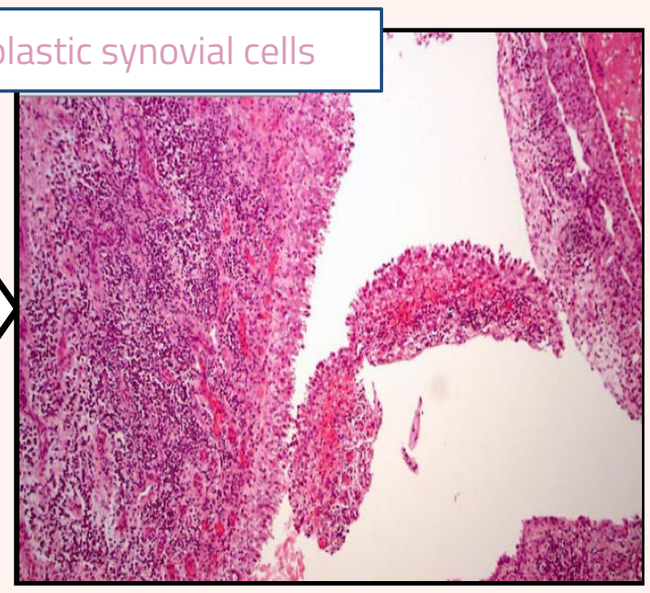


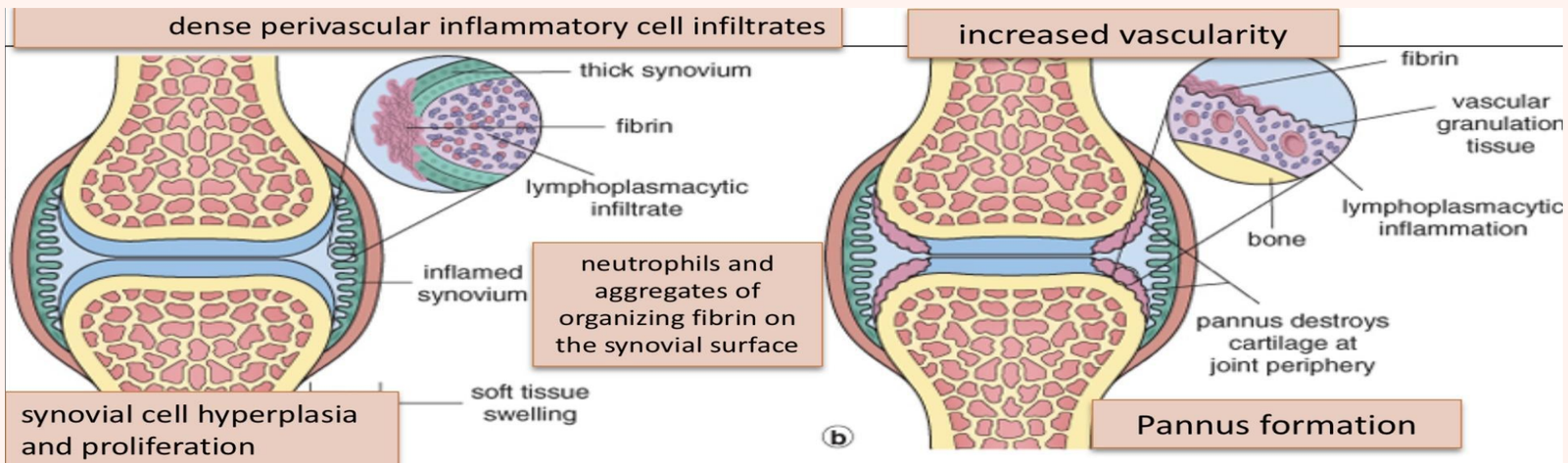
Congested hyperplastic synovium Eroded bone and cartilage

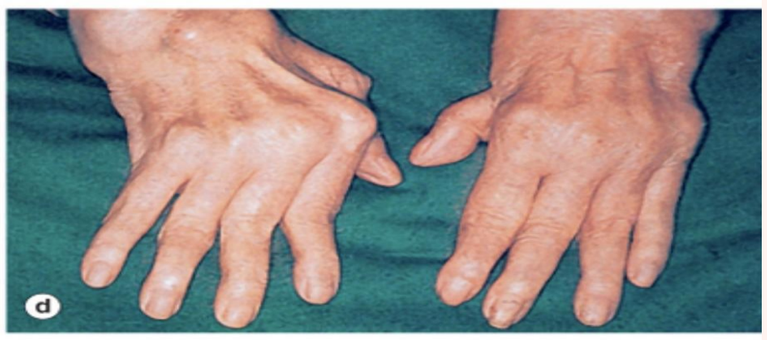


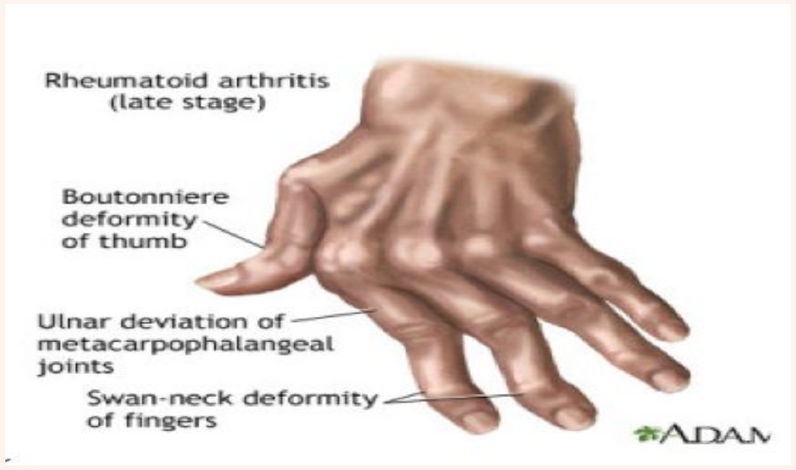
Dens chronic inflammation consisting of lymphocytes and plasma cells

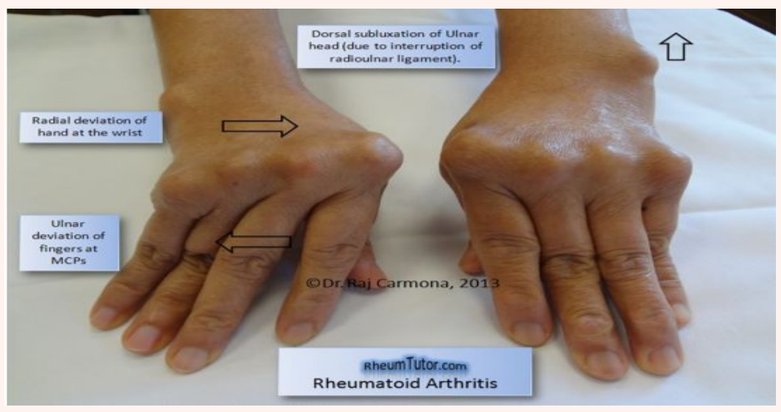
Hyperplastic synovial cells



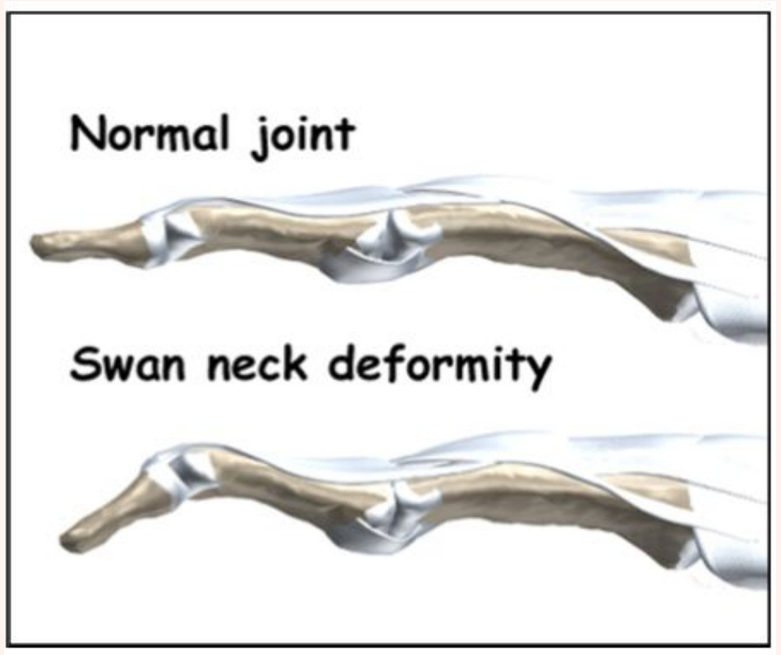








Swan neck:
hyperextension of the proximal interphalangeal joint (PIP) and flexion of the distal interphalangeal joint (DIP) Boutonniere: flexion of the PIP and hyperextension of the DIP.



Laboratory findings

Not sensitive or specific

Anti-CCP (cyclic citrullinated peptides)protein antibodies most specific for a diagnosis of rheumatoid arthritis

Rheumatoid factor: 80% have IgM autoantibodies to Fc portion of IgG

Other antibodies include antikeratin antibody (specific, not sensitive), antiperinuclear factor, anti-rheumatoid arthritis associated nuclear antigen (RANA), ESR and C-reactive protein

- **Synovial fluid** has increased neutrophils (particularly in acute stage) & protein
- **Genetics:** HLA-DR4, DR1 (65%)

Clinical Features

In females slides

morning stiffness, arthritis in 3+ joint areas → arthritis in hand joints → symmetric arthritis Affecting small joints

Clinical Features

Males slides

1. Symmetric arthritis, principally affecting the small joints of the hands and feet, ankles, knees, wrists, elbows, and shoulders
2. Most often, the proximal interphalangeal and metacarpophalangeal joints are affected, but distal interphalangeal joints are spared
3. Weakness , low grade fever
4. Axial involvement, when it occurs, is limited to the upper cervical spine; similarly, hip joint involvement is extremely uncommon
5. - Weakness , low grade fever , -Aching and stiffness of the joints, particularly in the morning ,As the disease advances, -the joints become enlarged, motion is limited

Clinical features : Characteristic deformities develop

Males slides

- Radial deviation at the wrists
- Ulnar deviation at the fingers
- Flexion and hyperextension deformities of the fingers (swan neck and Boutonniere deformities)

Clinical course

In females slides

1. variable; malaise, fatigue, musculoskeletal pain and joint involvement
2. 10% have acute onset of severe symptoms, but usually joint involvement occurs over months to years
3. 50% have spinal involvement
4. joints are warm, swollen, painful, stiff in morning
5. Dx: clinical data, rheumatoid nodules, rheumatoid factor, Antibodies against cyclic citrullinated peptides and typical radiographic changes

Rheumatoid nodules

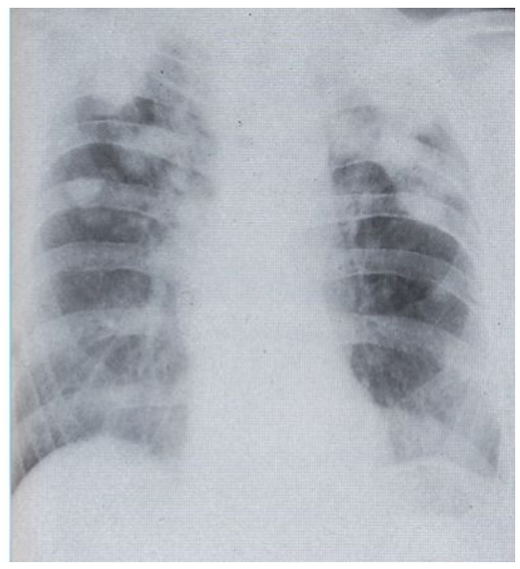
Males slides

- (1/4 of RA patients)
- Subcutaneous tissue including the forearm, elbows, occiput, and lumbosacral area
- Rarely, RA can involve the lungs (rheumatoid nodules, interstitial lung disease)
- Rheumatoid nodules are firm, nontender, oval or rounded masses as large as 2 cm in diameter
- Histologically resemble necrotizing granulomas
- Central focus of fibrinoid necrosis surrounded by a palisade of macrophages
- Which in turn is rimmed by granulation tissue and lymphocytes

Example : Female slide only

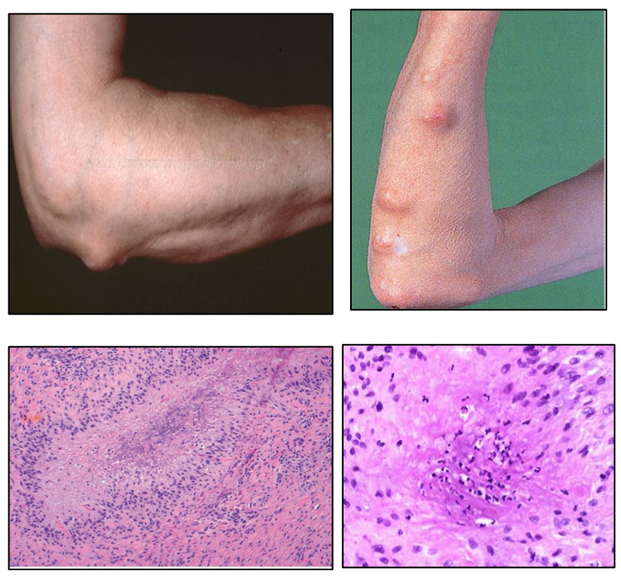
Multiple rheumatoid nodules in the lung

These nodules are more common in men. They may cavitate; they may require further investigation to exclude the possibility of malignancy



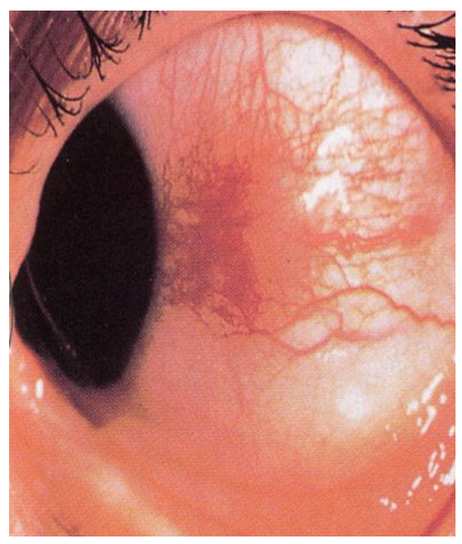
Subcutaneous rheumatoid nodule

The upper forearm and elbow are the most common sites for skin nodules in rheumatoid arthritis. These nodules result from vasculitis,and they may ulcerate or become necrotic,as has occurred at the elbow of this patient.



Rheumatoid episcleritis

May present acute as a localized area of inflammation.It is common ocular complication of rheumatoid arthritis but does not carry the same poor prognosis as scleritis



Prognosis

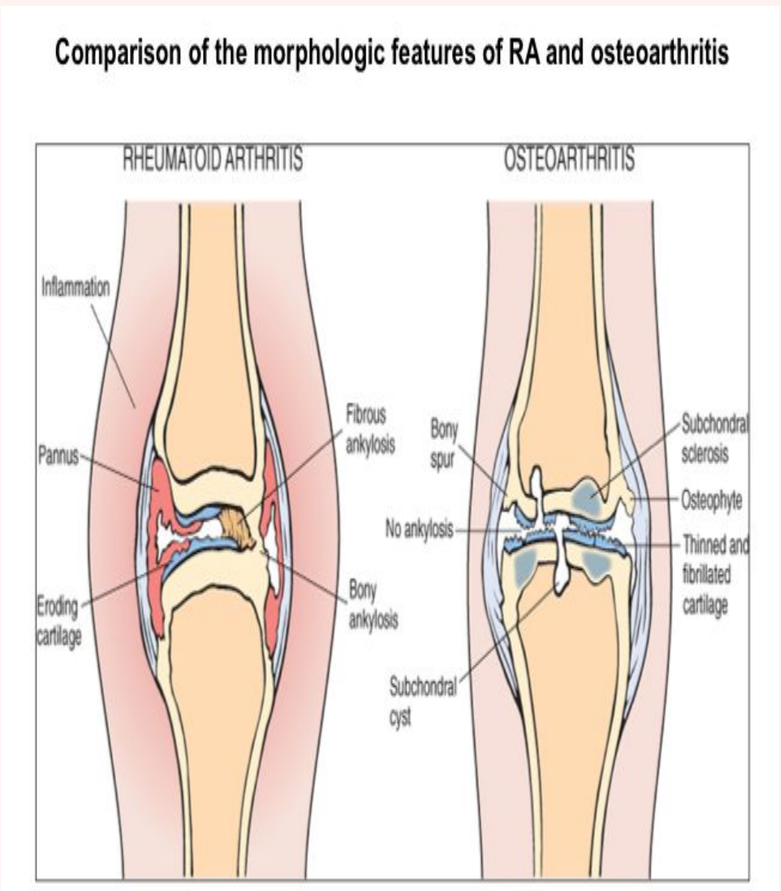
- The clinical course of RA is highly variable
- In a minority of patients, the disease may stabilize or even regress
- In most patients it pursues a chronic, remitting-relapsing course
- Progressive joint destruction leading to disability after 10 to 15 years
- The outcome has been dramatically improved by recent advances in therapy
- Reactive amyloidosis, which develops in 5% to 10% of these patients, particularly those with long-standing severe disease
- Reduces life expectancy by 3-7 years
- Death due to amyloidosis, vasculitis, GI bleeds from NSAIDs, infections from steroids.

Treatment

- **Corticosteroid**
- **Immunosuppressant like methotrexate, TNF antagonist**
- **Long term treatment with TNF antagonists carries with it increased risk of infections with organisms such as M. tuberculosis**

Summary

- The disease is caused by an autoimmune response against an unknown self antigen(s)
- RA is a chronic inflammatory disease that affects mainly the joints, especially small joints, but can affect multiple tissues
- The cytokine TNF plays a central role, and antagonists against TNF are of great benefit
- This leads to T-cell reactions in the joint with production cytokines that activate phagocytes that damage tissues and stimulate proliferation of synovial cells (synovitis)



Comparison of Osteoarthrosis & Rheumatoid Arthritis

	Osteoarthrosis	Rheumatoid Arthritis
Basic process	Degenerative	Immunologic, inflammatory
Site of initial lesion	Articular cartilage	Synovium
Age	50 plus	Any, but peaks at age 20–40 years
Sex	Male or female	Female > male
Joints involved	Especially knees, hips, spine; asymmetric involvement	Hands, later large joints; multiple symmetric involvement
Fingers	Herberden's nodes	Ulnar deviation, spindle swelling
Nodules	No	Rheumatoid nodules
Systemic features	None	Uveitis, pericarditis, etc.
Constitutional symptoms	None	Fever, malaise in some
Laboratory findings	None	Rheumatoid factor; ↑erythrocyte sedimentation rate; anemia, leukocytosis, hyperglobulinemia
Joint fluid	Clear, normally viscous; no inflammatory cells	Clear; low viscosity, high protein; neutrophils, some lymphocytes; immunoglobulins, complement, rheumatoid factor

Gout(podagra)

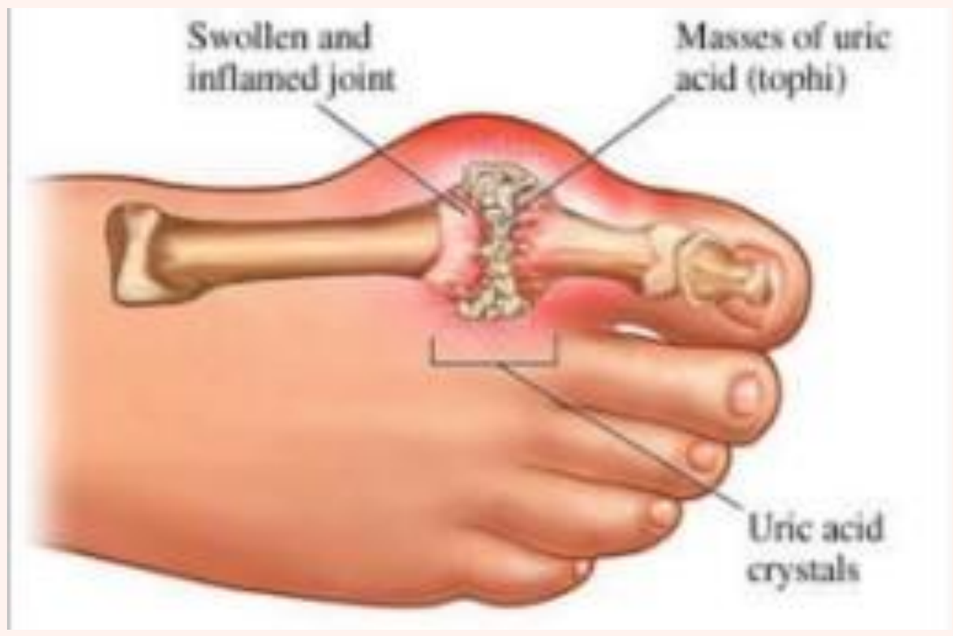
[Helpful Video]

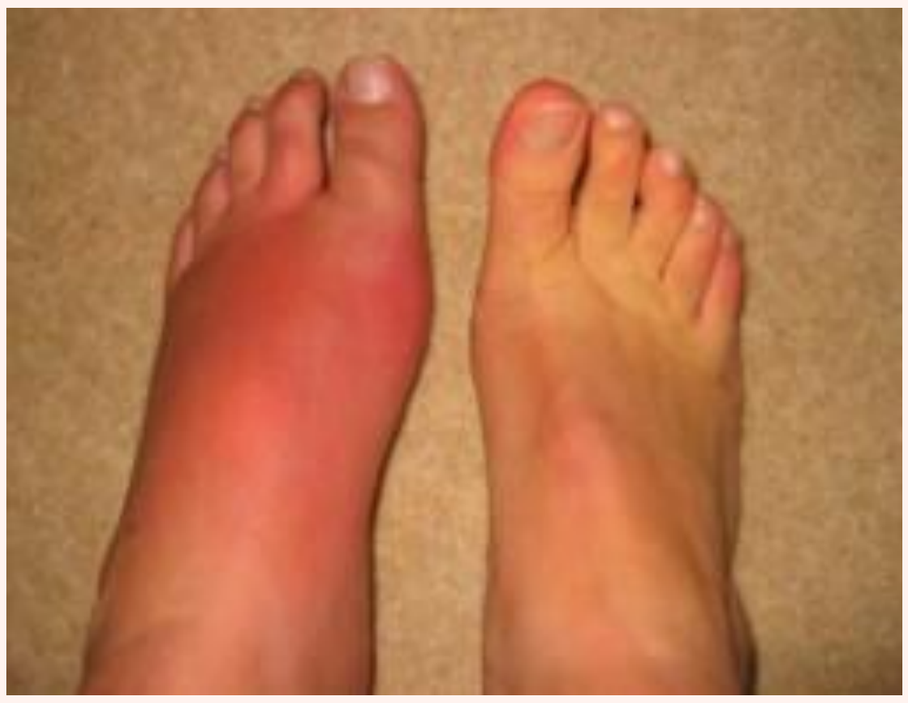
Gout is an inflammatory disease.

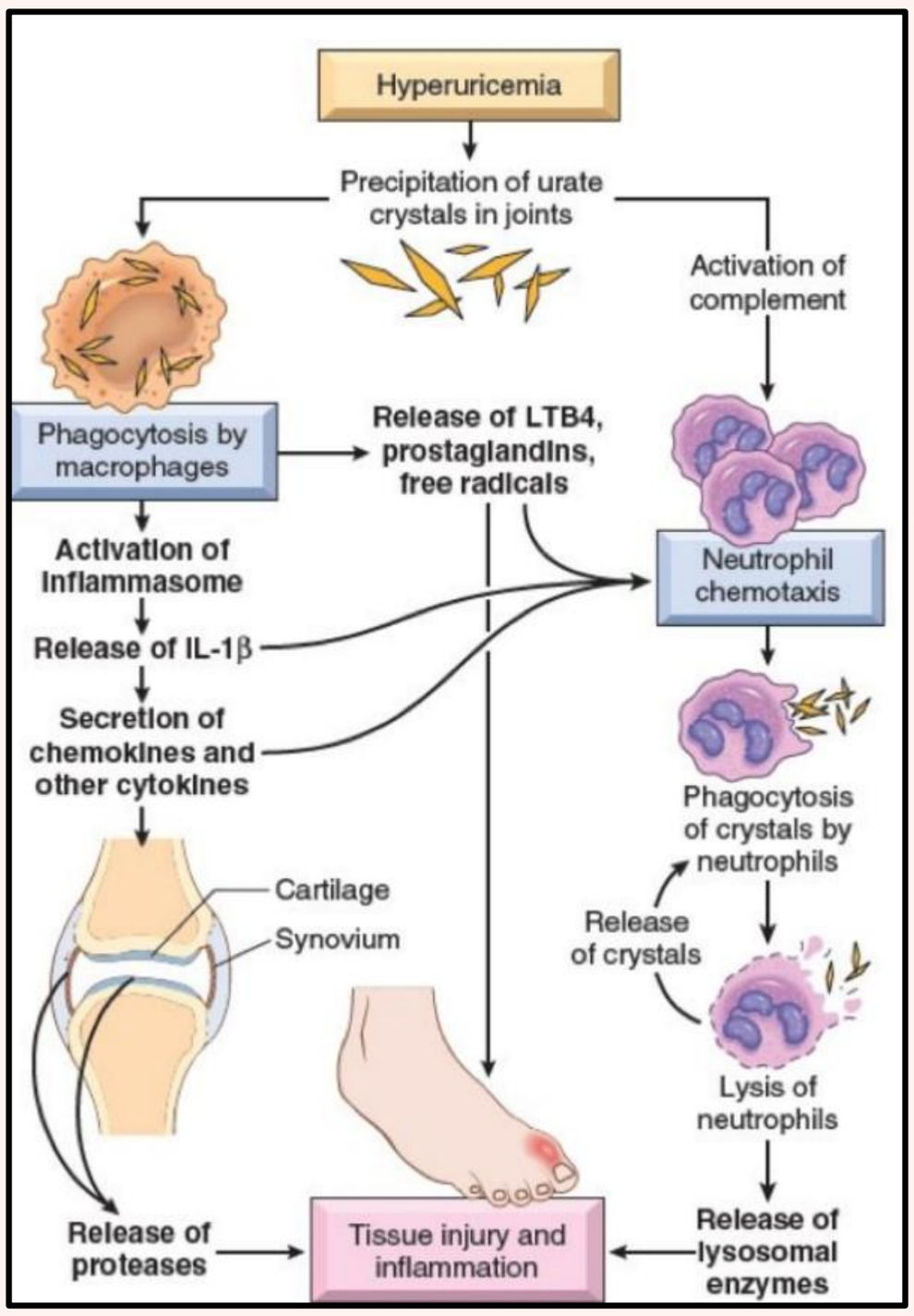
The most commonly affected site
is: first metatarsophalangeal joint.

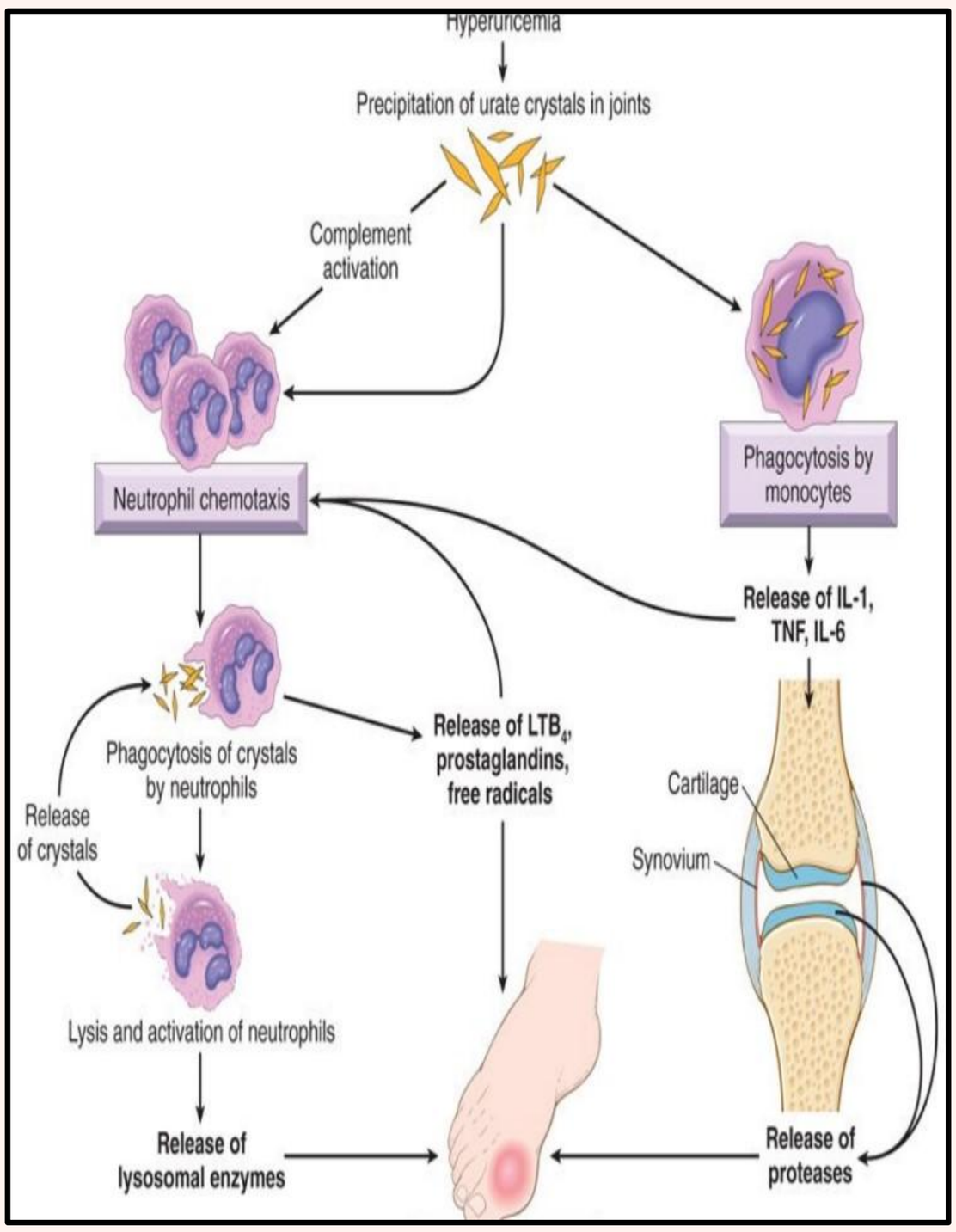
It is swollen, red, and very painful

sodium urate crystals have precipitated into the joint,producing an acute inflammatory response.









الصورتين نفس الفكرة مع اختلافات بسيطة

In male slides

Gout(podagra)

1. Transient attacks of acute arthritis initiated by urate crystals deposited within and around joints
2. Gout affects about 1% of the population, and shows a predilection for males
3. It is caused by excessive amounts of uric acid
   - Overproduction
   - Under excretion
4. Hyperuricemia (plasma urate level above 6.8 mg/dL) is necessary, but not sufficient, for the development of gout
5. Monosodium urate crystals precipitate from supersaturated body fluids and induce an acute inflammatory reaction
6. Recurrent episodes of acute arthritis, sometimes accompanied by the formation of large crystalline aggregates called tophi, and eventual permanent joint deformity

Risk Factors

In male slides

Age (more than 30)	Genetic	Obesity	Excess alcohol intake	Consumption of purine-rich foods	Renal failure	Drugs(e.g.,thiazides)that reduce excretion of urate

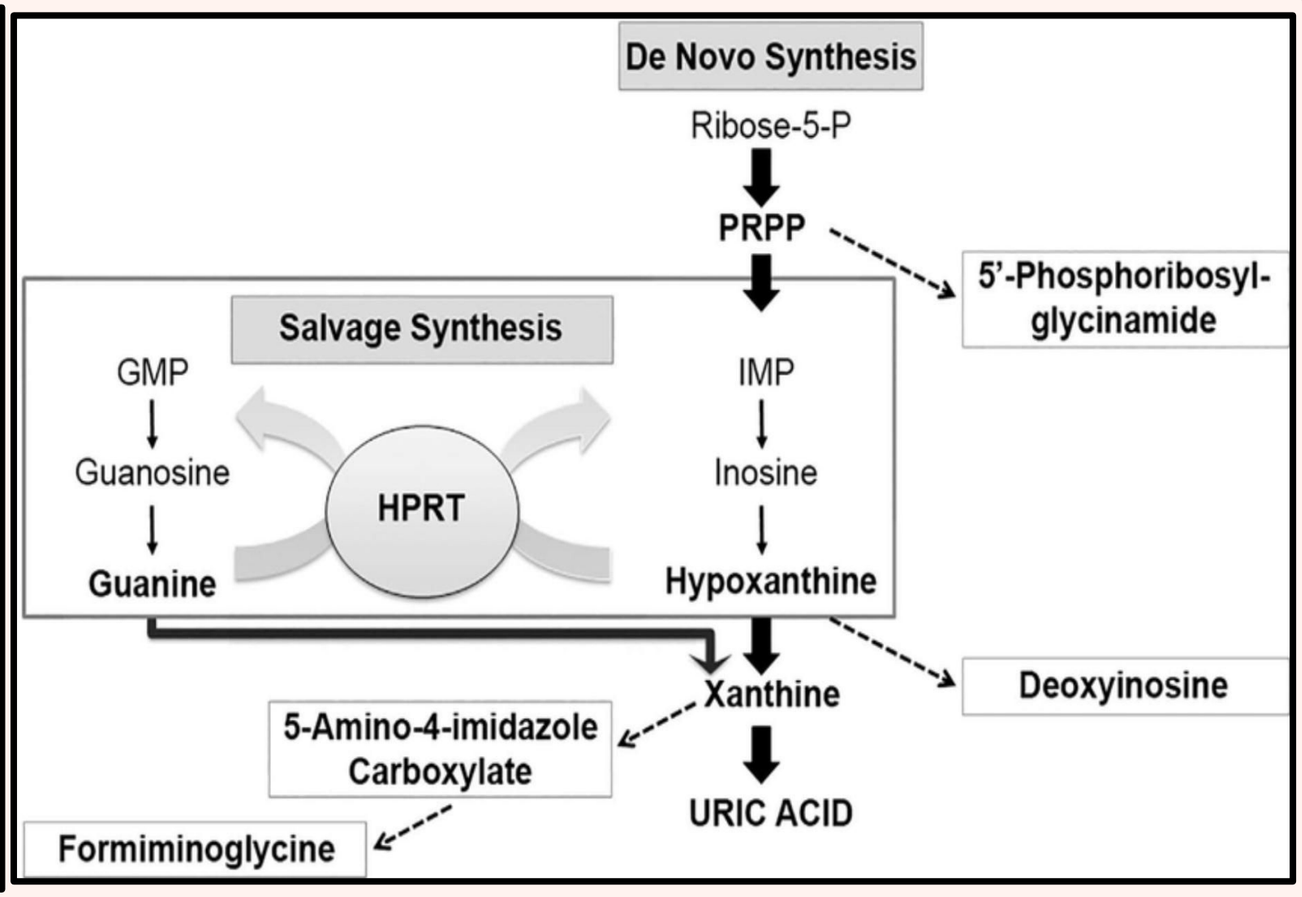
داء النقرس كان يسمى سابقً ا داء الأغنياء
لأنه يأتي من كثرة تناول اللحم

In male slides

Gout(podagra)

Clinical Category	Uric Acid Production	Uric Acid Excretion
Primary Gout (90%)		
Unknown enzyme defects (85%-90%)	↑ (majority) ↑↑ (minority) Normal	Normal ↑ ↓
Known enzyme defects (e.g., partial HGPRT deficiency)	↑	Normal
Secondary Gout (10%)		
Increased nucleic acid turnover (e.g., leukemia)	↑↑	↑
Chronic renal disease	Normal	↓
Congenital (e.g., Lesch-Nyhan syndrome HGPRT deficiency)	↑↑	↑

HGPRT, Hypoxanthine guanine phosphoribosyl transferase.



Morphology

In male slides

Morphology

Acute arthritis

- Dense inflammation in synovium and synovial fluid
- Crystals in cytoplasm of neutrophils and synovium
- Long, slender, needle, negative birefringence
- Synovium is edematous and congested with few lymphocytes, plasma cells and macrophages

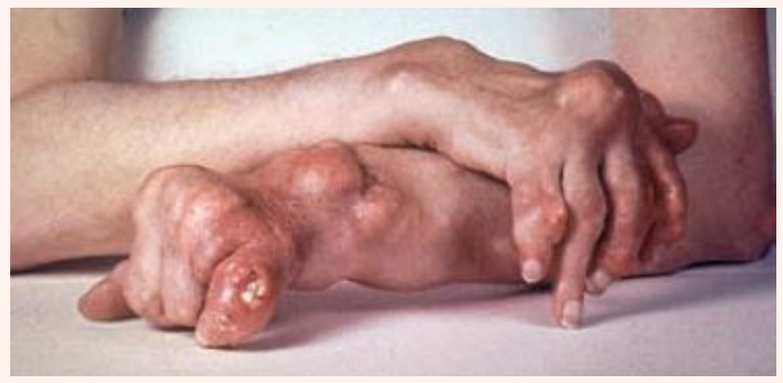
Chronic tophaceous arthritis

- Repetitive precipitation of crystals
- Chalky deposits in synovium
- Synovium: hyperplastic, fibrotic and thickened by inflammatory cells that form PANNUS destroy the cartilage

In female slides

Gout(podagra)

Clinical presentation



This is an example of chronic gout with gouty tophi. Note that there are numerous asymmetrical,periarticular swellings

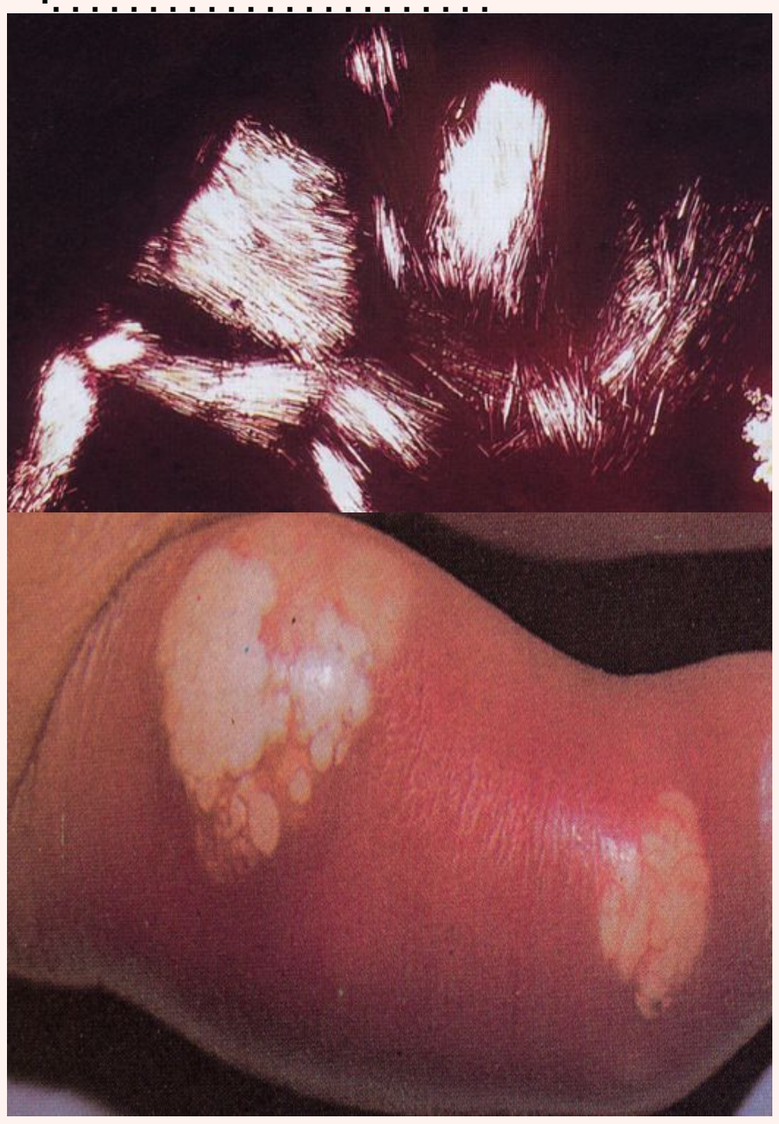
These represent inflammatory reaction to sodium urate crystals

Tophi appear only after repeated attacks of gout in patients whose hyperuricemia has not been treated

Acute on chronic gout in the little finger

The tophi helped to confirm the diagnosis. On aspiration, they were found to contain monosodium urate monohydrate crystals

polarized light

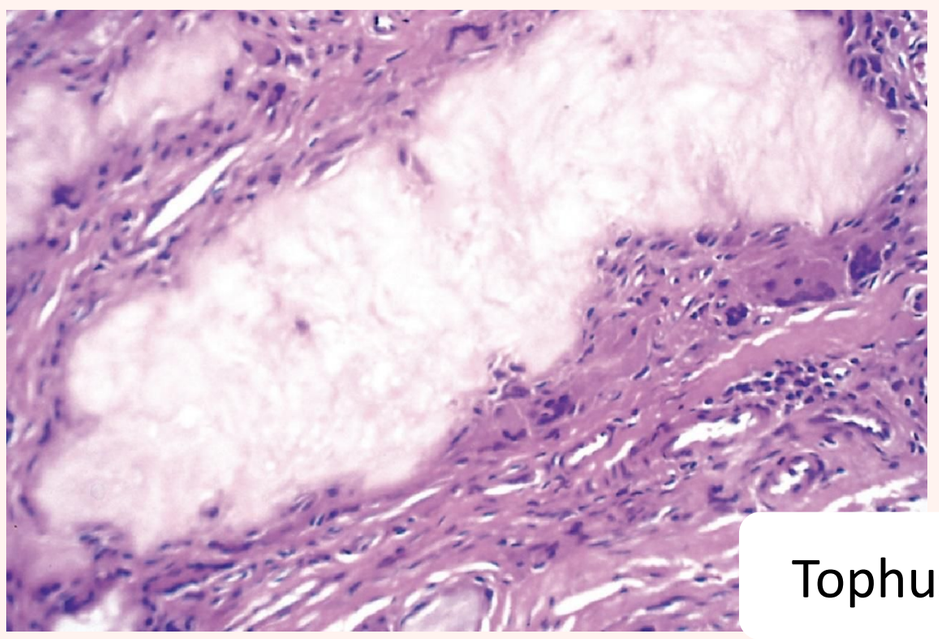


Gout(podagra)

Long, slender, needle-shaped monosodium urate crystals

TOPHI

- Large aggregations of urate crystals surrounded by an macrophages,
- lymphocytes intense foreign body giant cell reaction. In routinely processed sections, the crystals are removed during processing.
- Cartilage, ligaments, tendons, and bursae are pathognomonic of gout



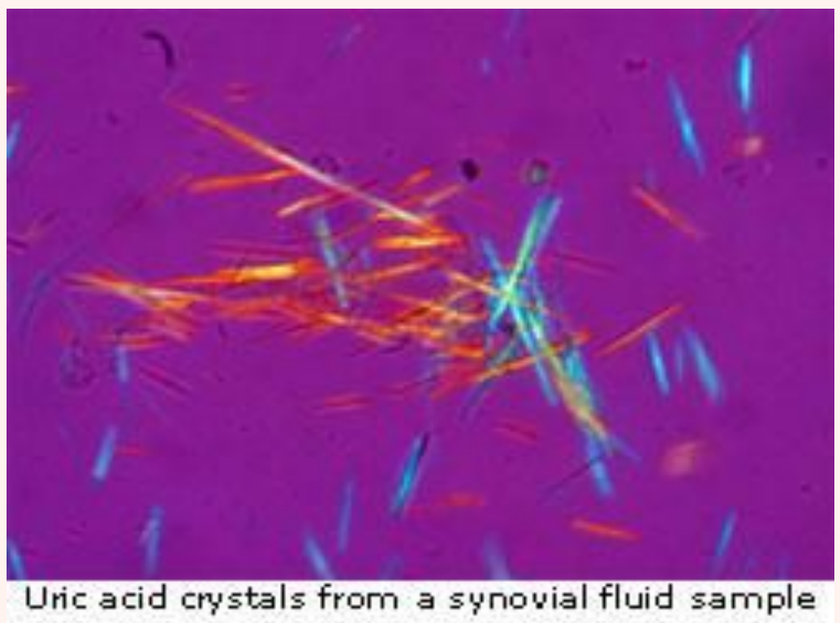
Tophus

Gouty nephropathy

- Urate crystals or tophi in the renal medullary interstitium or tubules
- Uric acid nephrolithiasis and pyelonephritis

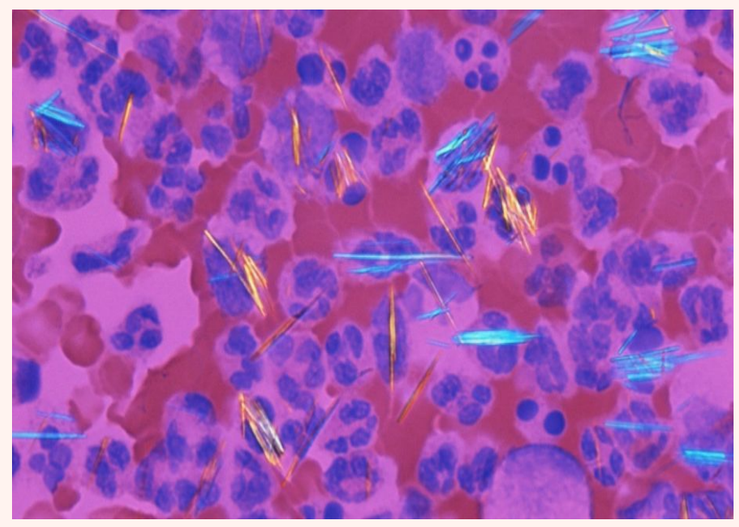
Approximately 20% of patients die of renal the collecting tubes failure. Renal lesions are many:

- precipitation of urates in the medulla forms tophi uric acid stones
- acute renal failure due to precipitation of urates in the collecting tubes



Uric acid crystals from a synovial fluid sample

long ,slender, needle-shaped monosodium urate crystals



In male slides

Gout(podagra)

Clinical Course

Four clinical stages are recognized:

stages

1	2	3	4
Asymptomatic hyperuricemia	Acute arthritis	Asymptomatic intercritical period	chronic tophaceous gout
Appears around puberty in men and after menopause in women	Sudden, excruciating joint pain, hyperemia, warmth	Resolution of acute	12 years after initial attack
	50% metatarsophalangeal joint	Symptom free	Radiology: show characteristic juxta-articular bone erosion caused by osteoclastic bone resorption
	Last for hours to weeks	Frequent attacks and multiple joints (in the absence of appropriate treatment)	Loss of joint space

In male slides

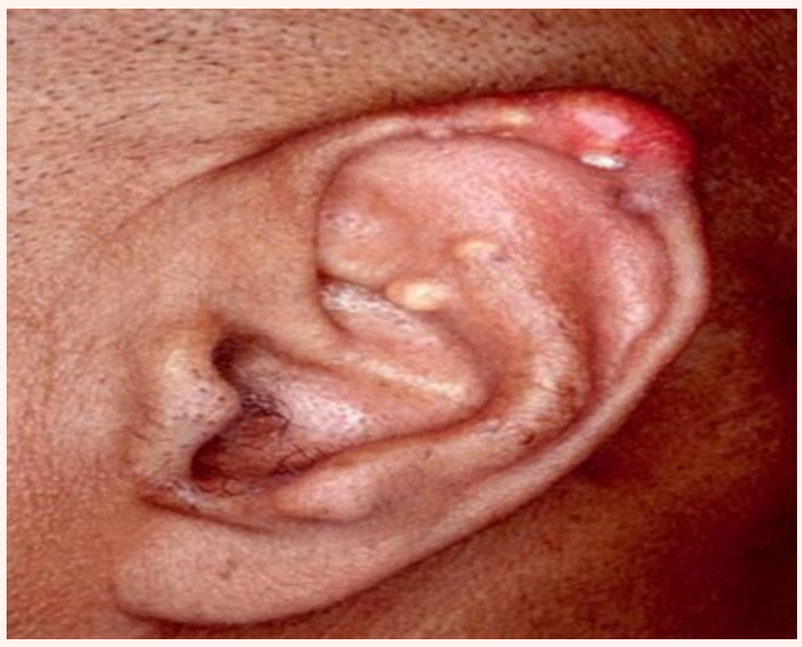
Gout(podagra)

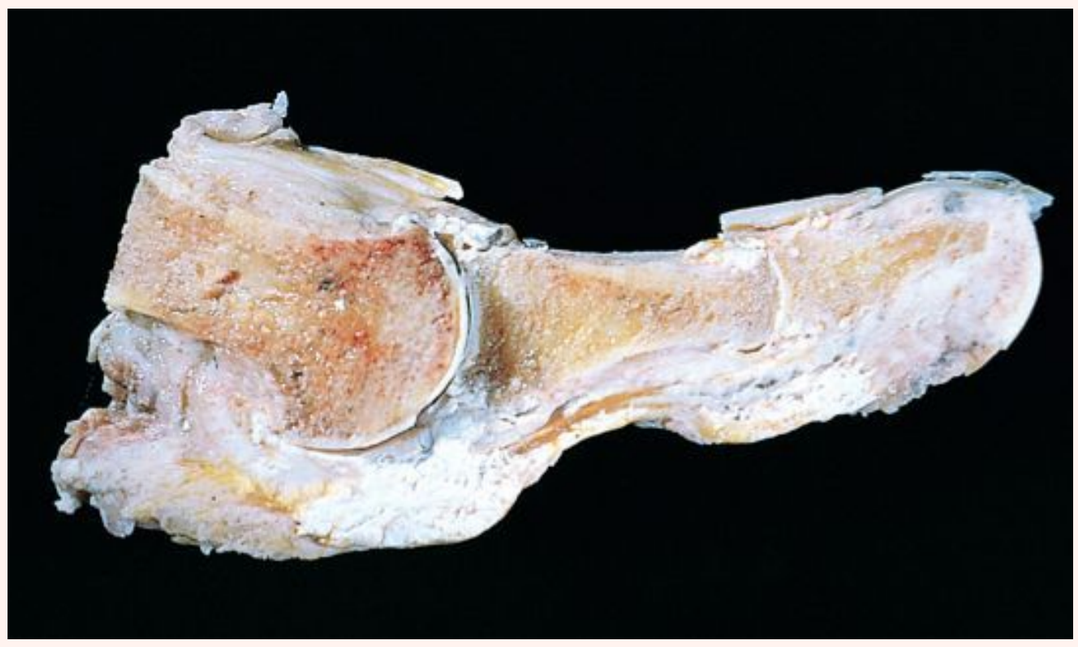
Treatment

Lifestyle modification

Reduce symptoms (NSAIDs)

Lower urate level (medications)





In female slides

White tophi involving the joint and soft tissues

Disease	Findings
Pyogenic arthritis	Purulent fluid exudate; large numbers of neutrophils; culture positive for bacteria
Tuberculous arthritis	Fluid exudate (high protein and specific gravity); neutrophils and mononuclear cells; culture positive for *Mycobacterium tuberculosis*
Rheumatoid arthritis	Clear fluid, high protein content; inflammatory cells: neutrophils and mononuclear cells; increased immunoglobulins and complement; rheumatoid factor present in many cases
Osteoarthrosis	Clear fluid, high protein content; no inflammatory cells
Gout	Urate crystals
Chondrocalcinosis	Calcium pyrophosphate crystals

In male slides

Calcium Pyrophosphate Crystal Deposition Disease (Pseudogout)

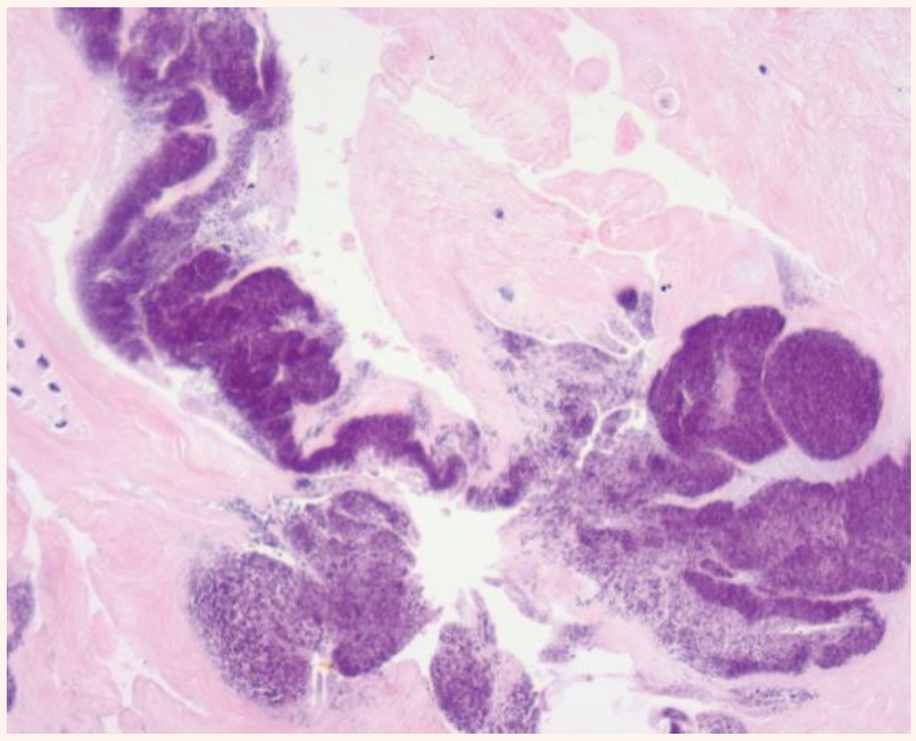
- Also known as pseudo-gout and chondrocalcinosis
- This condition is due to the deposition of CPP in the synovium (pseudogout) and articular cartilage (chondrocalcinosis)
- It can occur in three main settings:

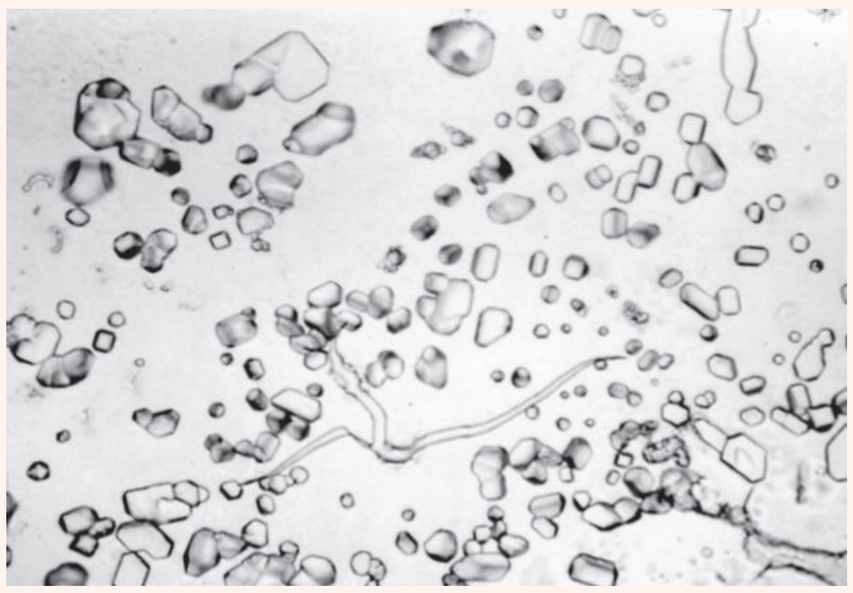
1. Sporadic (more common in the elderly)
2. Hereditary
3. Secondary to other conditions, such as previous joint damage, hyperparathyroidism, hypothyroidism, haemochromatosis and diabetes

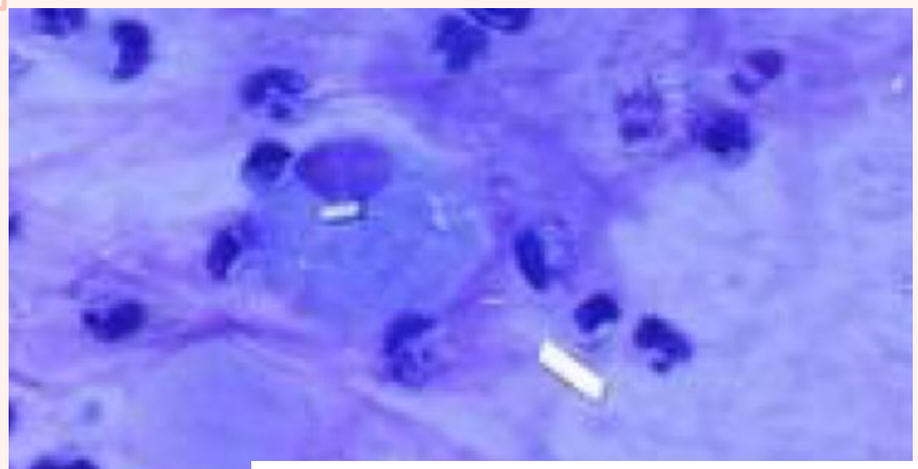
- The crystals first develop in the articular cartilage (chondrocalcinosis), which is usually asymptomatic.
- From here, the crystals may shed into the joint cavity resulting in an acute arthritis, which mimics gout and is therefore called pseudogout.

Pseudogout can be differentiated from gout in three ways:

- The knee is most commonly involved, wrist, elbow, shoulder, ankle
- X-rays show the characteristic line of calcification of the articular cartilage
- The crystals look different under polarizing microscopy, they are rhomboid in shape, positively birefringent



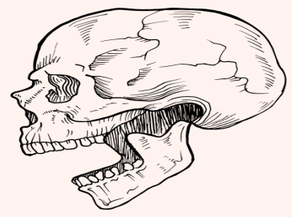




Chondrocalcinosis

MISSION

Keywords



Word (disease)	The word that leads to the disease (symptoms, features or a word)
Rheumatoid arthritis	Associated with HLA-DR4 , Hallmark is synovitis leading to formation of pannus (inflamed granulation tissue) , Anti-CCP is specific marker for this disease
Rheumatoid arthritis	swan neck shaped fingers
Osteoarthritis	Results from degeneration of the articular cartilage and its disordered repair.
Osteoarthritis	formation of lips of new bone Mushroom-shaped osteophytes The fracture gaps allow synovial fluid to be forced into the subchondral regions to form fibrous walled cysts
Osteoarthritis	constant friction of bone surfaces with polished bone (bone eburnation).
Osteoarthritis	Bouchard node=Proximal IPJ Heberden nodes=Distal IPJ

MCQs

Q1: A 50 years old woman was complaining of joint pain that she felt in different intervals in the last couple of years mainly in hands and feet, after examination it was revealed that she had swan neck shaped fingers with ulnar deviation What serologic test would confirm the diagnosis ?						
A. IgM		**B. Anti CCp**		**C. IgG**		**D. IgA**
Q2: As a medical student, you have been asked by your supervisor to perform a clinical examination on a 59-year old lady who is a known case of rheumatoid arthritis. Which one of the following can be detected in her hands and wrists?						
A. Bouchard's nodes		**B. Swan-neck deformity**		**C. Heberden's nodes**		**D. Tophi**
Q3-a non-neoplastic disorder of progressive erosion of articular cartilage?						
Osteoarthritis		**rheumatoid arthritis**		**septic arthritis**		**SLE**
Q4-A 36 year old type 2 diabetic(Obese) Male was complaining of aching pain in his Knee joint with limited joint mobility, Which one of the following is most likely to be the cause?						
Primary Osteoarthritis		**rheumatoid arthritis**		**Secondary Osteoarthritis**		**gout**
Q5:Which ONE of the following is a possible cause for secondary gout?						
A. Increase uric acid production		**B. Increased purine degradation**		**C. Lambert eaton syndrome**		**D. Increased purine excretion**
Q6:What joint is most commonly involved in gout?						
A. 2nd metatarsophal angeal joint		**B. 1st proximal interphalangeal joint**		**C. 1st metatarso-phalang eal joint**		**D. 1st distal interphalangeal joint**

answers; 1:B , 2:B , 3:A , 4:C , 5:A, 6:C

Pathology Team

leaders:

Layal alkhalifah

Abdulaziz Nasser

Members:

Ghida Alkahtani

Abdulaziz Alanazi

Aram Alzahrani

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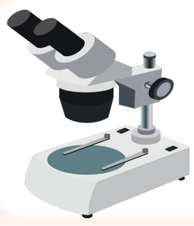

Sahar Alfallaj

Faisal alghamdi

Norah Alnoshan

Faisal Alamoud

Raseel Aldajany

Tariq Alshamrani

Moath al abdussalam