Osteoarthritis

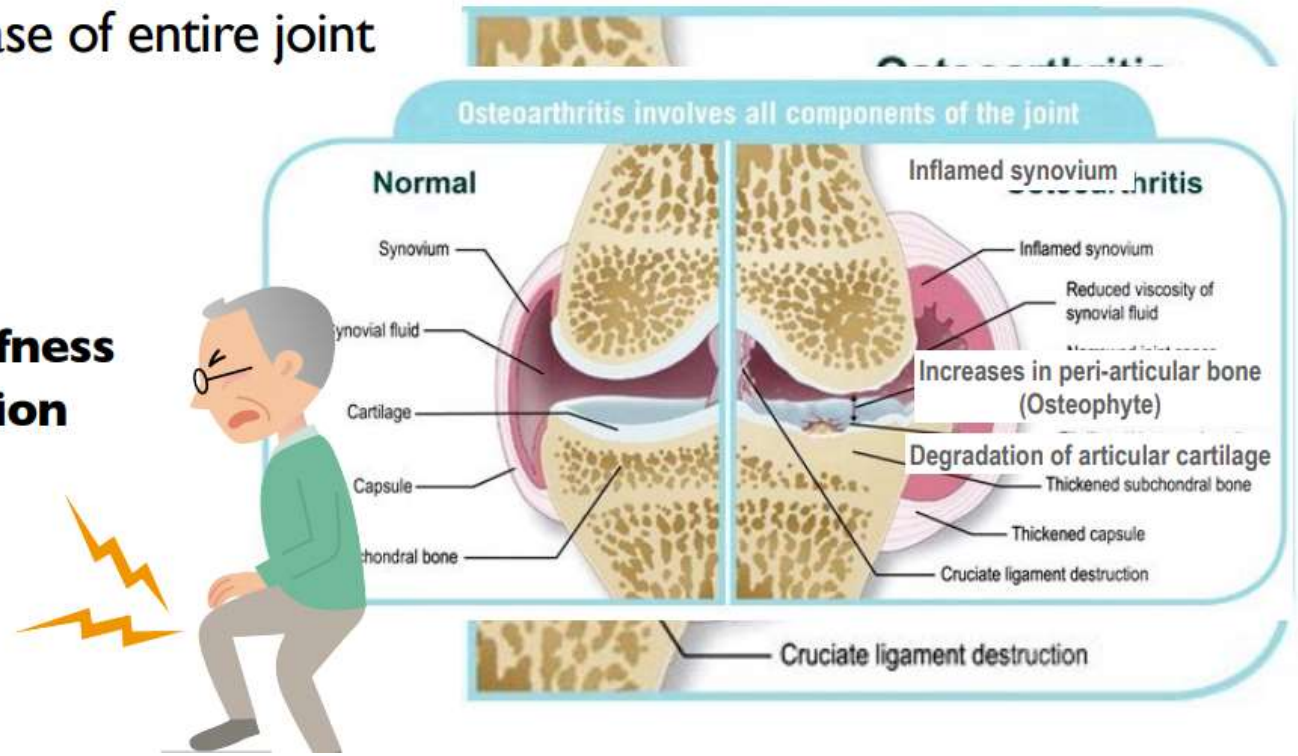
Isbandiyah

FKUMM

What is **Osteoarthritis (OA)**?

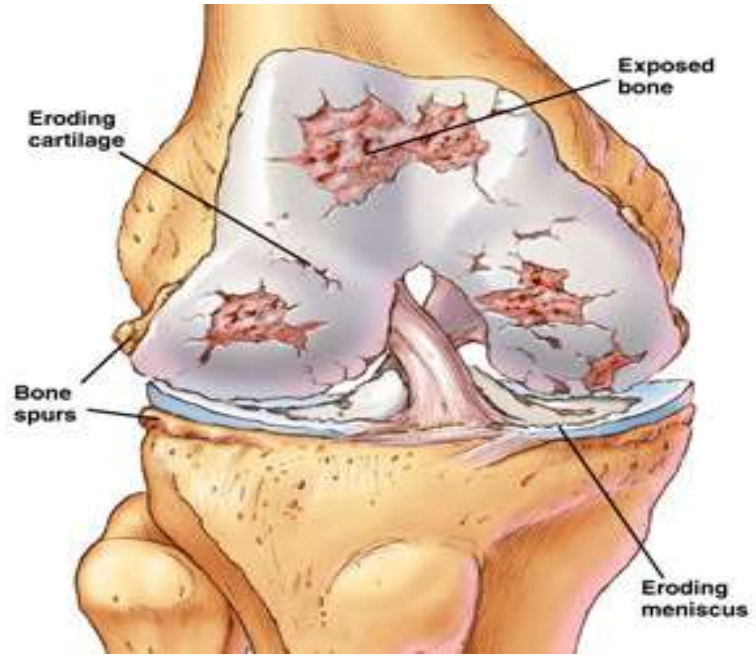
is a local disease of entire joint

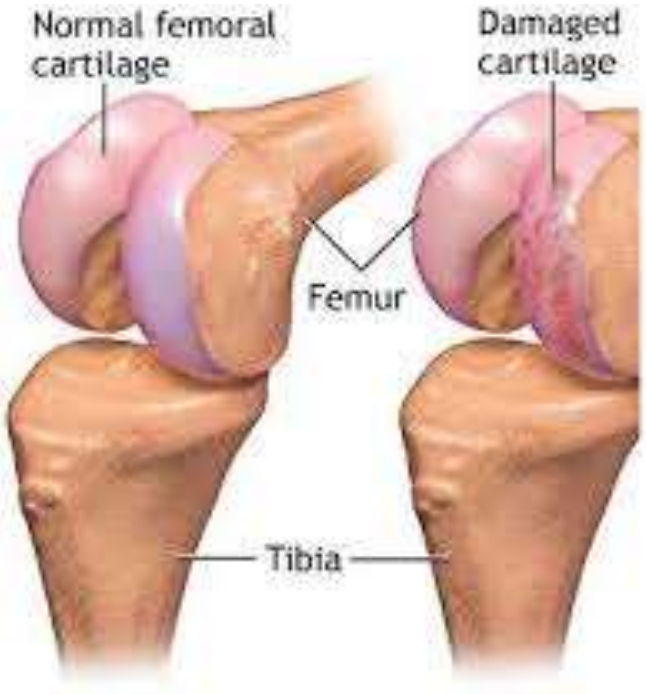
Manifest as **joint pain**, **stiffness** and **loss of joint function**



Definition

- OA can be described as the degradation and loss of articular cartilage accompained by subchondral bone remodelling, osteophytes formation and synovitis





Osteoarthritis

- The most common form of **arthritis**
- 302 million people worldwide
- Prevalence likely to increase as population age

10% 20%

>60 y.o symptomatic **OA**

- Most common cause of **disability** in the elderly
- **Incompletely understood** Clinical Course and Pathogenesis

Pathogenesis

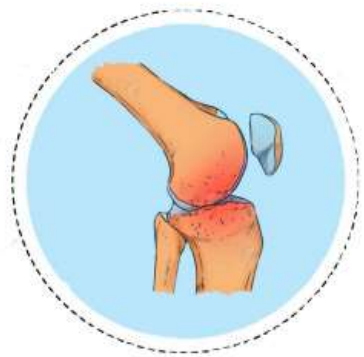
Osteoarthritis

Biomechanical factors

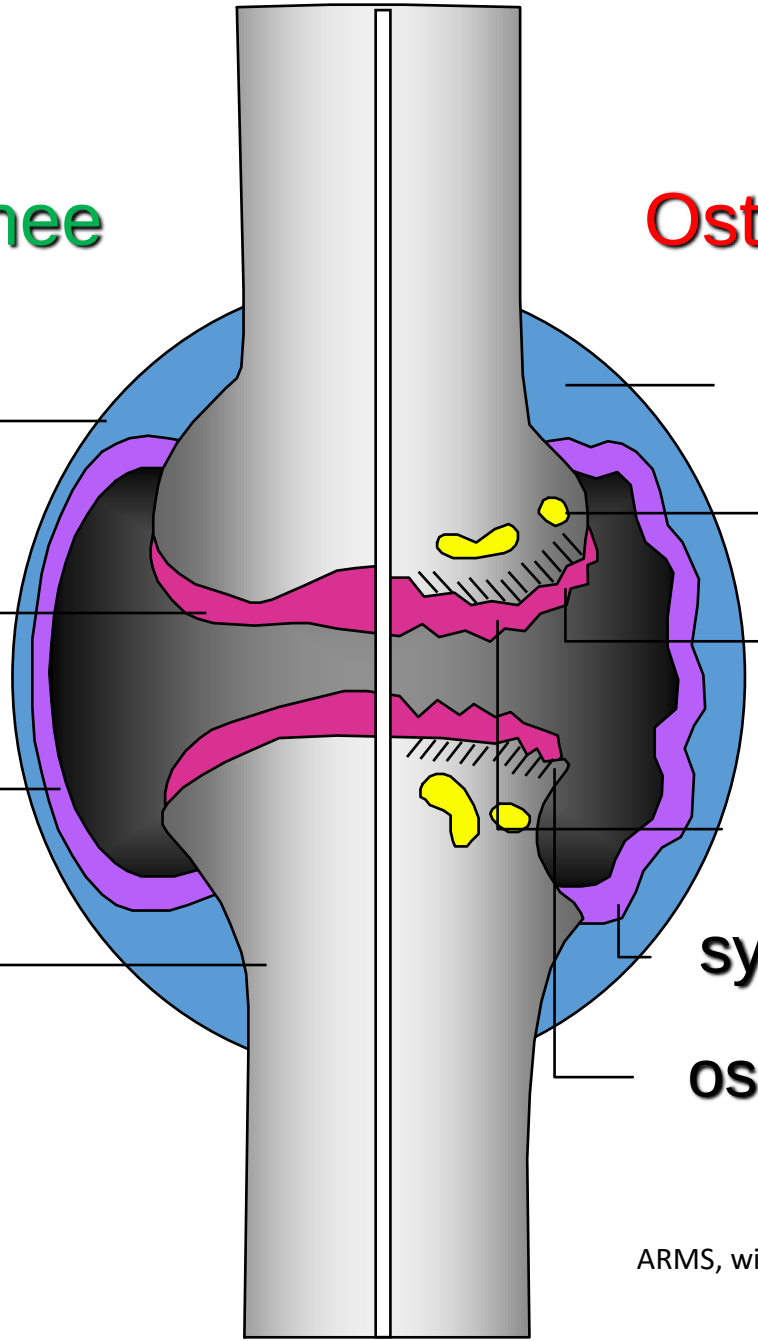
Much more complex than

"wear and tear"

Biochemical factors



NORMAL vs. OA JOINT



Normal knee

capsule

cartilage

synovium

bone

Osteoarthritic knee

thickened capsule

cyst formation

sclerosis in subchondral bone

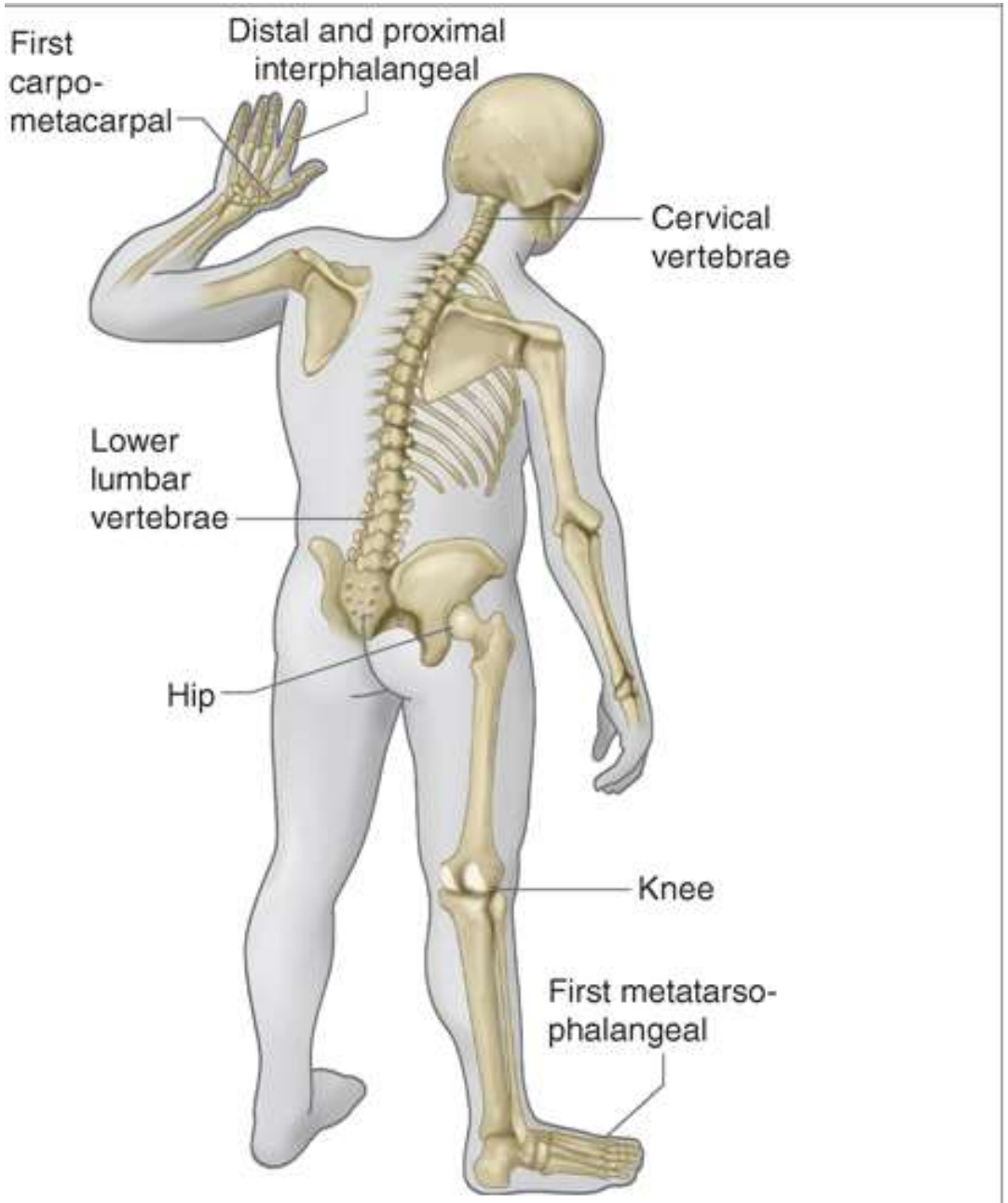
fibrillated cartilage

synovial hypertrophy

osteophyte formation

ARMS, with permission from the author, Sandra Navarra, 2003

Distribution of OA

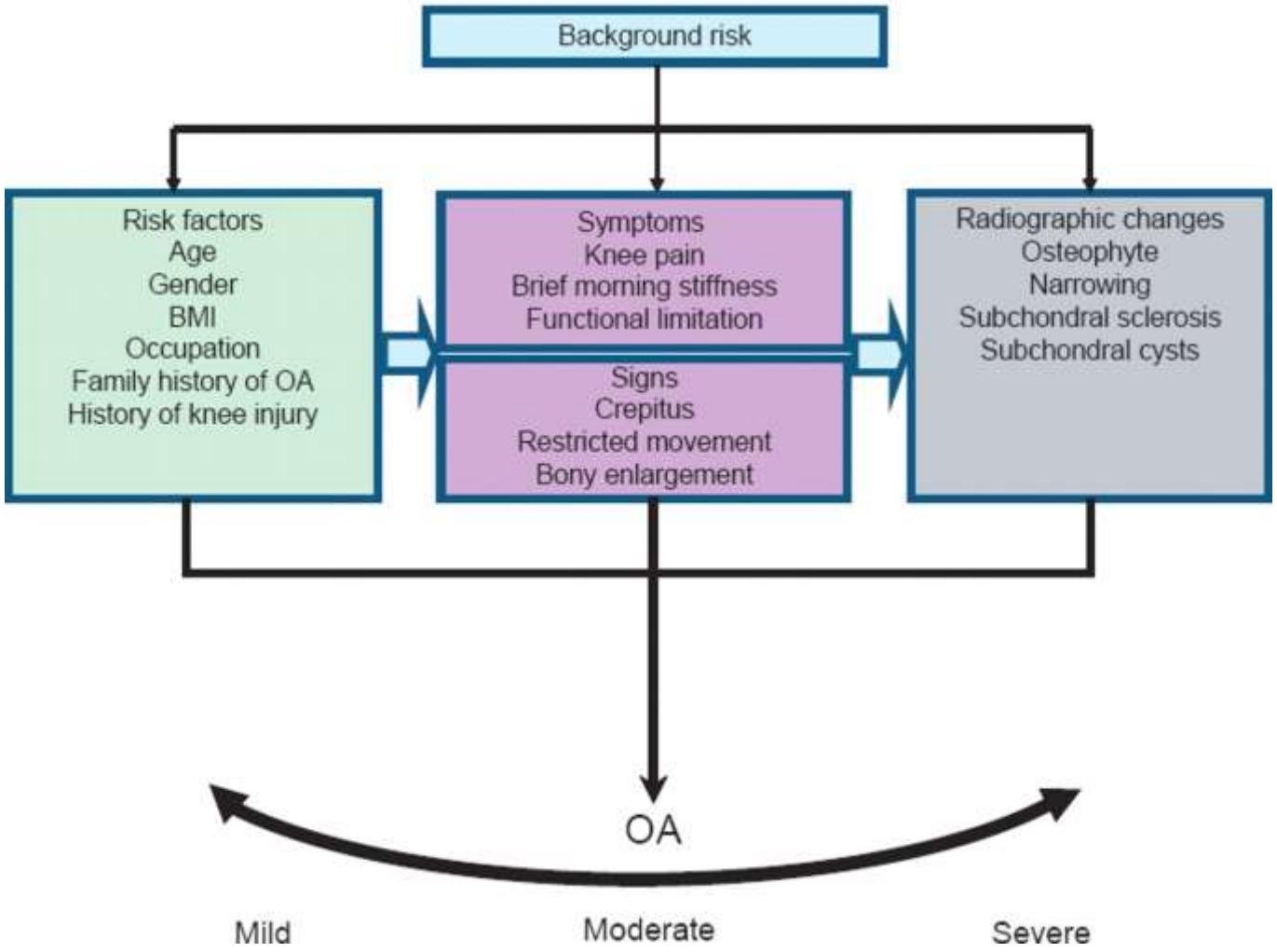


Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

Risk Factors

- Genetics
  - Abnormal components of the joint as an organ
  - Abnormal range of motion
  - Congenital anomalies
- Trauma
- Overuse syndromes
- Post infectious
- Obesity
- Age
- Gender

Major components in the diagnosis of knee osteoarthritis (OA).



Zhang W et al. Ann Rheum Dis 2010;69:483-489

ARD

Symptoms of osteoarthritis

- Joint pain
- Tenderness
- Swelling
- Stiffness
- Locking
- Sometimes an effusion
- Reduced motion
- Decreased movement can lead to pain, regional muscles may atrophy

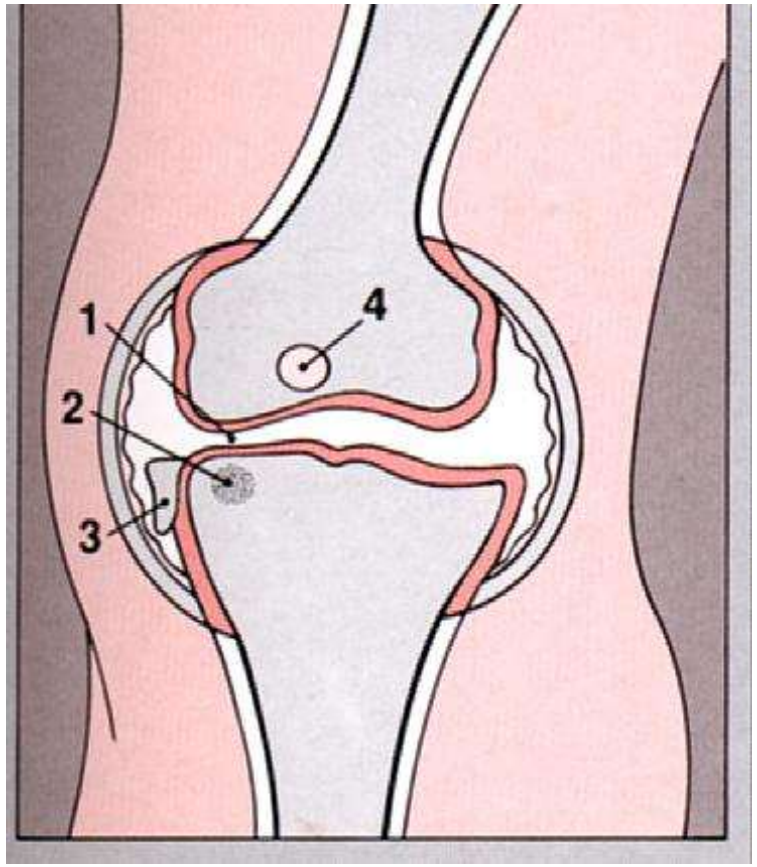
Physical Examination

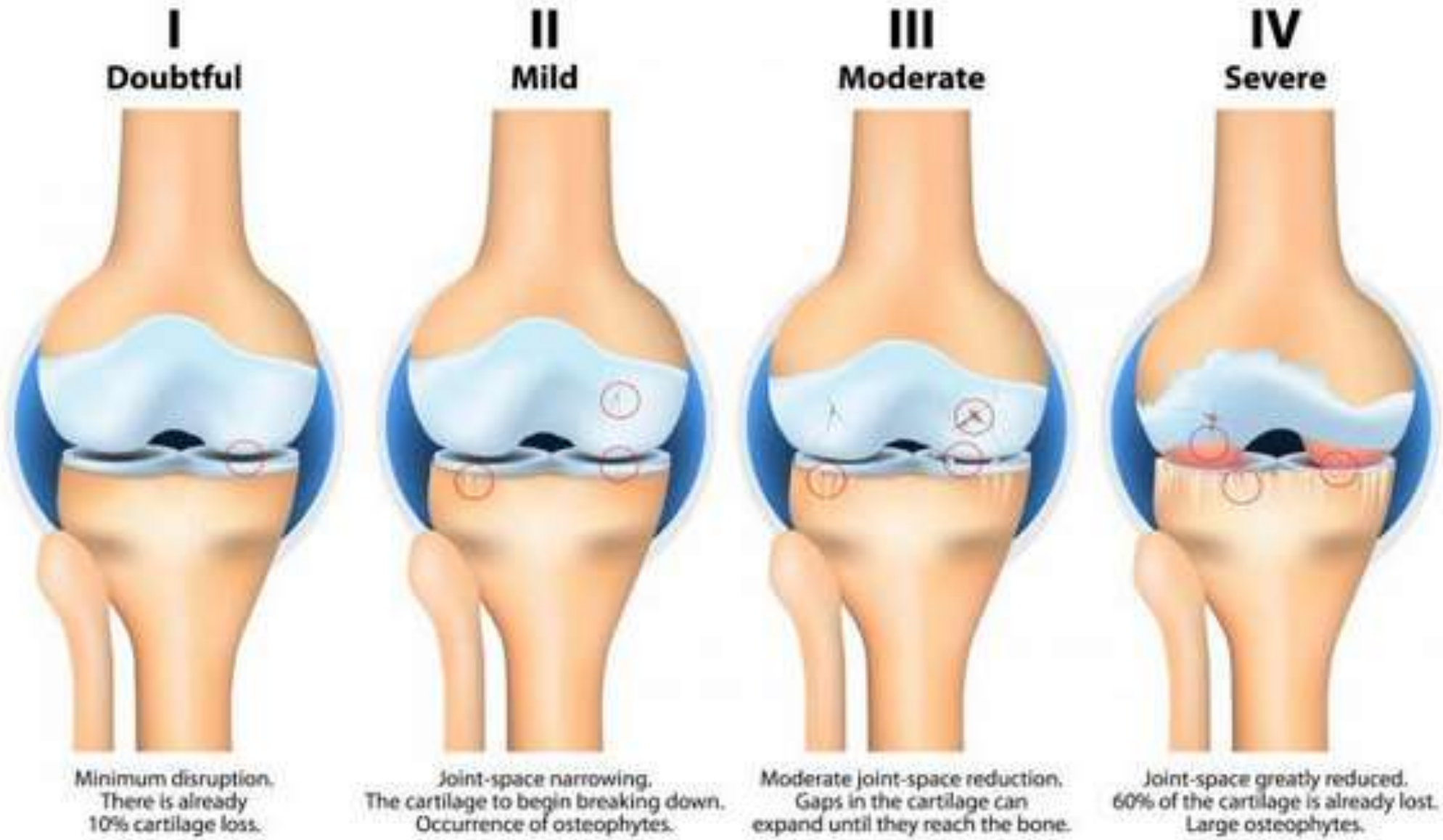
- Asymetry of findings usually of large joints
- Heberdens / bouchard's nodes
  - Classic hand involvement: DIP, PIP,
- joint tenderness
- creaking or grating (crepitus) sounds
- bony swelling
- excess fluid
- reduced movement
- joint instability
- muscle thinning

Radiographic features of the knee in OA

1. Joint space narrowing
2. Boney sclerosis
3. Marginal osteophytes
4. Subchondral cysts

- malalignment




I
Doubtful
II
Mild
III
Moderate
IV
Severe
Minimum disruption.
There is already
10% cartilage loss.
Joint-space narrowing.
The cartilage to begin breaking down.
Occurrence of osteophytes.
Moderate joint-space reduction.
Gaps in the cartilage can
expand until they reach the bone.
Joint-space greatly reduced.
60% of the cartilage is already lost.
Large osteophytes.

Clinical classification criteria for **Knee Osteoarthritis**

AMERICAN COLLEGE of RHEUMATOLOGY
Empowering Rheumatology Professionals

- Knee pain + at least 3 of 6:
  - Age > 50 years
  - Stiffness < 30 minutes
  - Crepitus
  - Bony tenderness
  - Bony enlargement
  - No palpable warmth

eular
EUROPEAN LEAGUE AGAINST RHEUMATISM

- Age > 40 years
- Movement-related joint pain
- Morning stiffness < 30 min
- Functional limitations
- One or more examination findings:
  - Crepitus
  - Restricted movement
  - Bony enlargement

NICE
National Institute for Health and Care Excellence

- Age > 45 years
- Activity-related joint pain
- No morning knee-stiffness or stiffness < 30 min.

Clinical classification criteria for **Knee Osteoarthritis**

AMERICAN COLLEGE of RHEUMATOLOGY
Empowering Rheumatology Professionals

Clinical

- Knee pain + at least 3 of 6:
  - Age > 50 years
  - Stiffness < 30 minutes
  - Crepitus
  - Bony tenderness
  - Bony enlargement
  - No palpable warmth

Clinical and Laboratory

- Knee pain + at least 5 of 9:
  - Age > 50 years
  - Stiffness < 30 minutes
  - Crepitus
  - Bony tenderness
  - Bony enlargement
  - No palpable warmth
  - ESR < 40 mm/hour
  - RF < 1:40
  - Synovial fluid OA

Clinical and Radiographic

- Knee pain + at least 1 of 3:
  - Age > 50 years
  - Stiffness < 30 minutes
  - Crepitus

+

Osteophyte

Clinical classification criteria for **Hand Osteoarthritis**

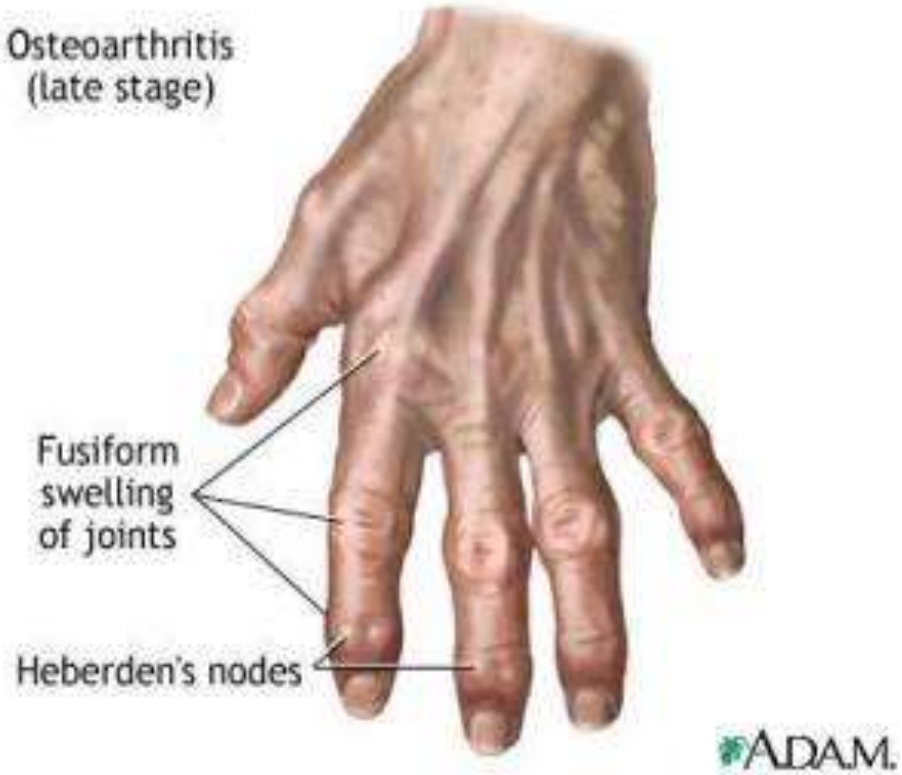
1990

Hand pain, aching, or stiffness and 3 or 4 of the following features:

- Hard tissue enlargement of 2 more of 10 selected joints;
  - 2nd and 3rd DIP joints both hands
  - 2nd and 3rd PIP joints both hands
  - 1st CMC joints both hands
- Hard tissue enlargement of 2 more DIP joints
- Fewer than 3 swollen MCP joints
- Deformity of at least 1 of 10 selected joints

Altman R, et al. *Arthritis Rheum.* 1990;33(11):1601-1610.


Rheumatoid arthritis
(late stage)
Boutonniere
deformity
of thumb
Ulnar deviation of
metacarpophalangeal
joints
Swan-neck deformity
of fingers
A.D.A.M.


Osteoarthritis
(late stage)
Fusiform
swelling
of joints
Heberden's nodes
A.D.A.M.

Clinical classification criteria for **Hip Osteoarthritis**

1991

Clinical

Hip pain AND hip internal rotation 15° AND ESR <45 mm/h (If ESR is not available, use hip flexion ≤ 115°)

OR

Hip internal rotation ≥ 15° AND pain on hip internal rotation AND morning stiffness <60 minutes AND Age >50 years

Clinical and radiographic

Hand and at least 2 of the following 3 features:

- ESR <20 mm/hour
- Radiographic femoral or acetabular osteophytes
- Radiographic joint space narrowing (superior, axial, and/or medial)

Laboratory Tests

Blood test

- No blood test for osteoarthritis as such
- Suggested to rule out other types of arthritis e.g. rheumatoid arthritis

Joint Fluid Analysis/Joint aspiration

- A needle is used to draw fluid out of the affected joint after the administration of anesthesia
- Examination and testing of the fluid to determine presence of inflammation , crystals or joint deterioration

TREATMENT OF OA

- Symptomatic treatment
- Structure modifying treatment
- Surgical treatment

Pharmacologic recommendations for the management of knee OA

We conditionally recommend that patients with knee OA should use one of the following:

- Acetaminophen
- Oral NSAIDs/ Cox2 inhibitor
- Topical NSAIDs
- Tramadol
- Intraarticular corticosteroid injections

We conditionally recommend that patients with knee OA **should not** use the following:

- Chondroitin sulfate
- Glucosamine
- Topical capsaicin

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SPECIAL ARTICLE

American College of Rheumatology 2012 Recommendations for the Use of Nonpharmacologic and Pharmacologic Therapies in Osteoarthritis of the Hand, Hip, and Knee

MARC C. HOCHBERG,[1] ROY D. ALTMAN,[2] KARINE TOUPIN APRIL,[3] MARIA BENKHALTI,[3] GORDON GUYATT,[4] JESSIE McGOWAN,[3] TANVEER TOWHEED,[5] VIVIAN WELCH,[3] GEORGE WELLS,[3] AND PETER TUGWELL[3]

Acetaminophen
Max Dose 4000 mg/day

Topical NSAID
or
Oral NSAID
or
Corticosteroid IA

- Age > 75 yo
- CKD stg IV-V

Don't use oral NSAID

GI Ulcer:
Cox 2 inh
or
NSAID + PPI

History of GI bleeding:

Cox 2 inh + PPI

Hyaluronate IA
Or
Tramadol
Or
Duloxetine

Total joint arthroplasty

Opioid

SYMPTOMATIC TREATMENT OF OA

- **Decrease of joint loading**
  - **Weight control**
  - Splinting
  - Walking sticks
- **Exercises**
  - Swimming
  - Walking
  - Strengthening
- **Patient education**

STRUCTURE MODIFYING TREATMENT

- Hyaluronic acid injection (HA)
- Glycose amino glycans (GAG)

INDICATIONS OF SURGICAL INTERVENTION

- Severe joint **pain**,
  resistant to conservative treatment methods
- Limitation of **daily living activities**
- **Deformity**, angular deviations, instability

GOUTY ARTHRITIS

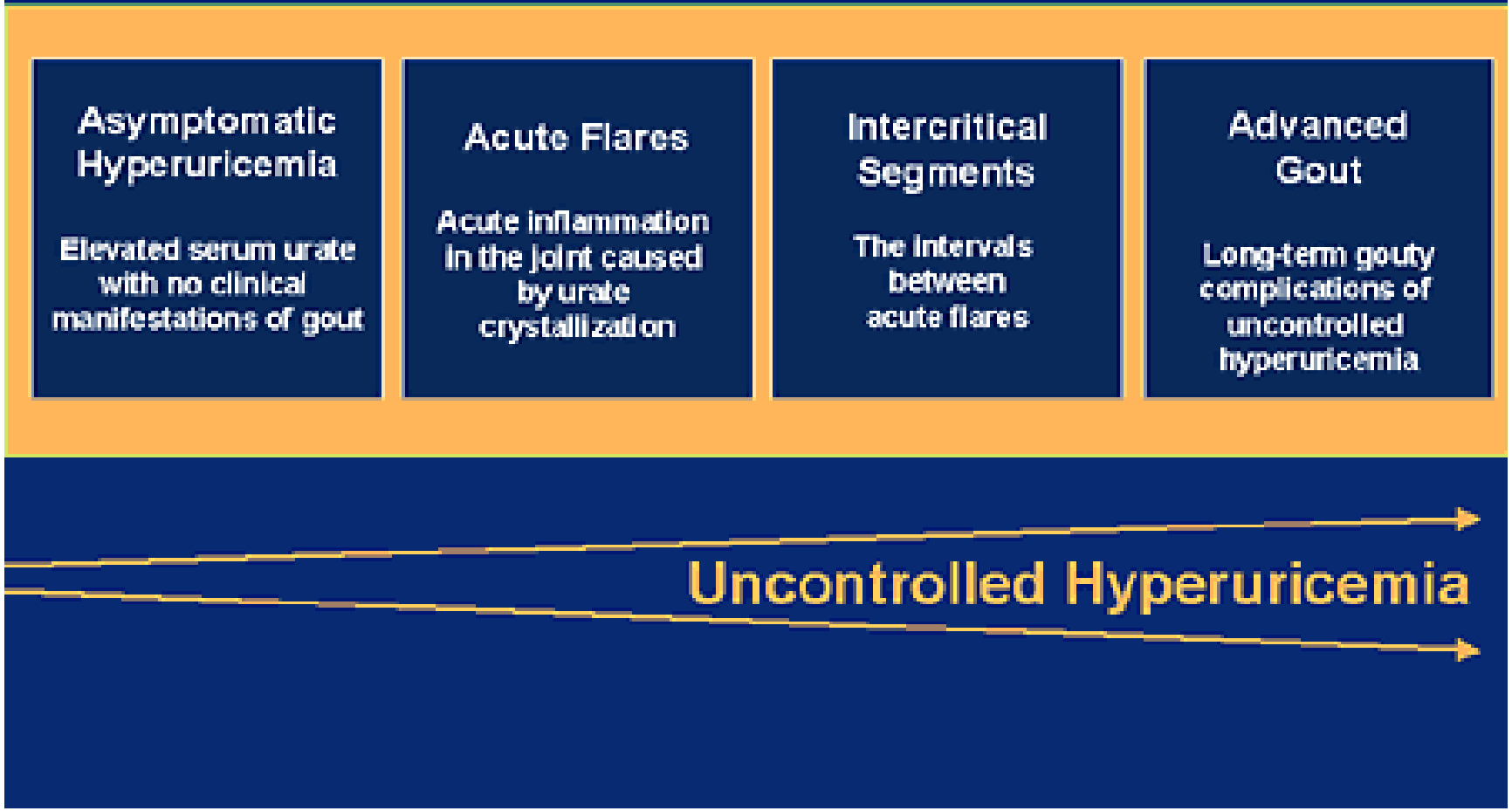
Isbandiyah dr, SpPD

SPECTRUM OF GOUT

- Hyperuricemia
- Acute gouty arthritis
- Tophaceous deposition of urate crystals
- Urolithiasis
- Interstitial deposition of urate crystals in renal parenchyma
- Uric acid nephropathy

Gout

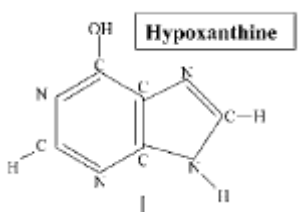
One Chronic Disease, Best Described by 4 Stages

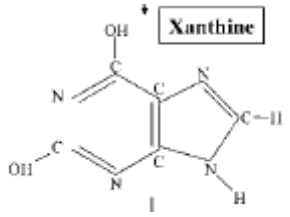


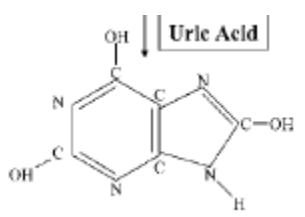
Pathophysiology

- Caused by the deposition of monosodium urate crystals in tissues
- Uric acid is a metabolic by-product of purine catabolism
- Purines→hypoxanthine→xanthine→uric acid
- Reaction catalyzed by xanthine oxidase, found in the liver
- When the balance of dietary intake, synthesis and rate of excretion are disrupted, hyperuricemia results
  - Overproduction (10%)
  - Underexcretion (90%)
- Results in arthritis, soft tissue masses, nephrolithiasis and urate nephropathy

Pathophysiology





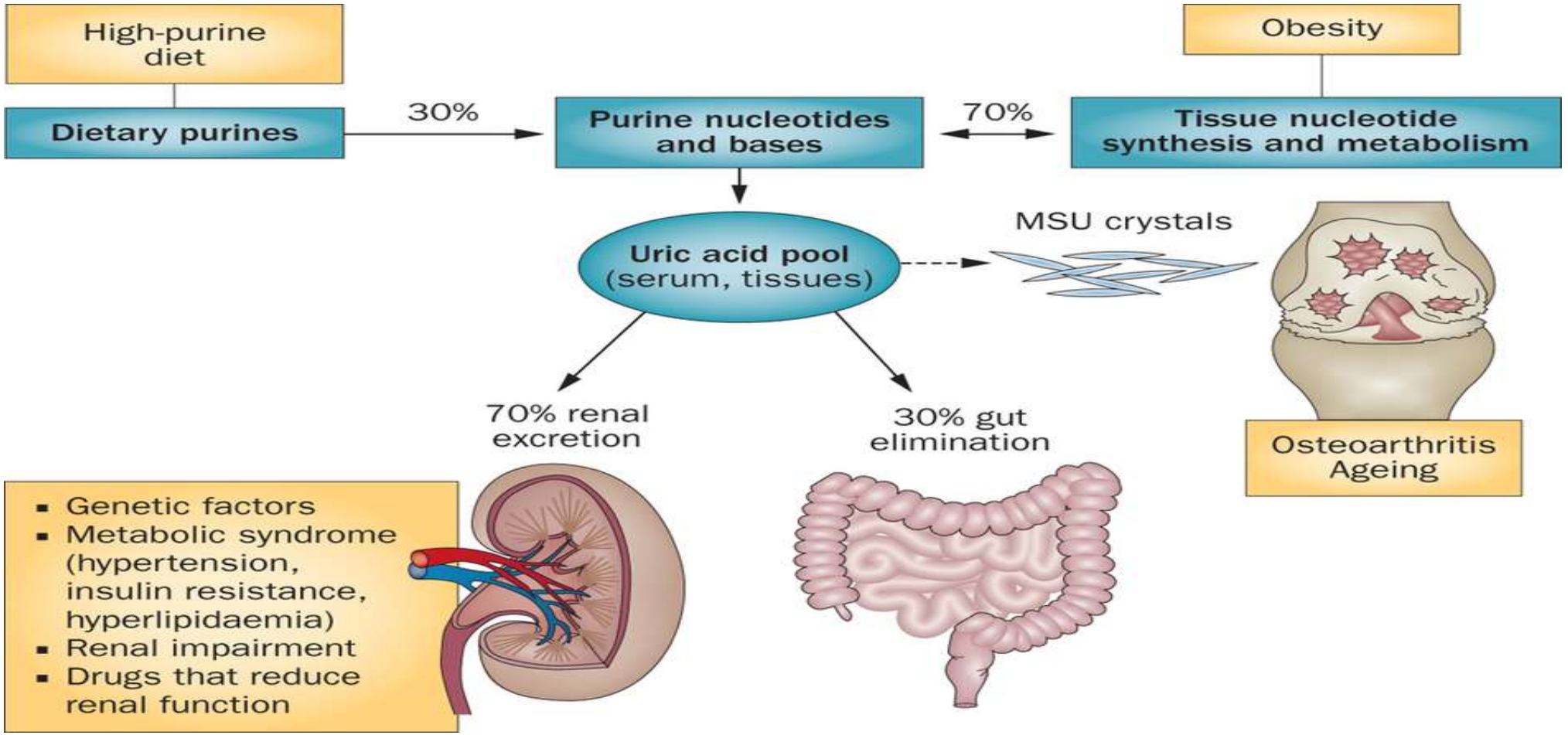


XO=xanthine oxidase

Allopurinol and febuxostat inhibit xanthine oxidase and block uric acid formation

Markel A. *IMAJ*, 2005.

Pathophysiology



Rees, F. *et al.* (2014) Optimizing current treatment of gout
Nat. Rev. Rheumatol. doi:10.1038/nrrheum.2014.32

Risk Factors

- High Purine Diet (Red Meat, Fatty Poultry, High Fat Dairy, Seafood)
- Alcohol Consumption
- Trauma
- Surgery
- Starvation
- Dehydration
- Obesity
- Drugs (Allopurinol, uricosuric agents, thiazides, loop diuretics, low dose aspirin)
- Renal Impairment
- Genetic Mutations (SLC22A9, SLC22A12, ABCG2)

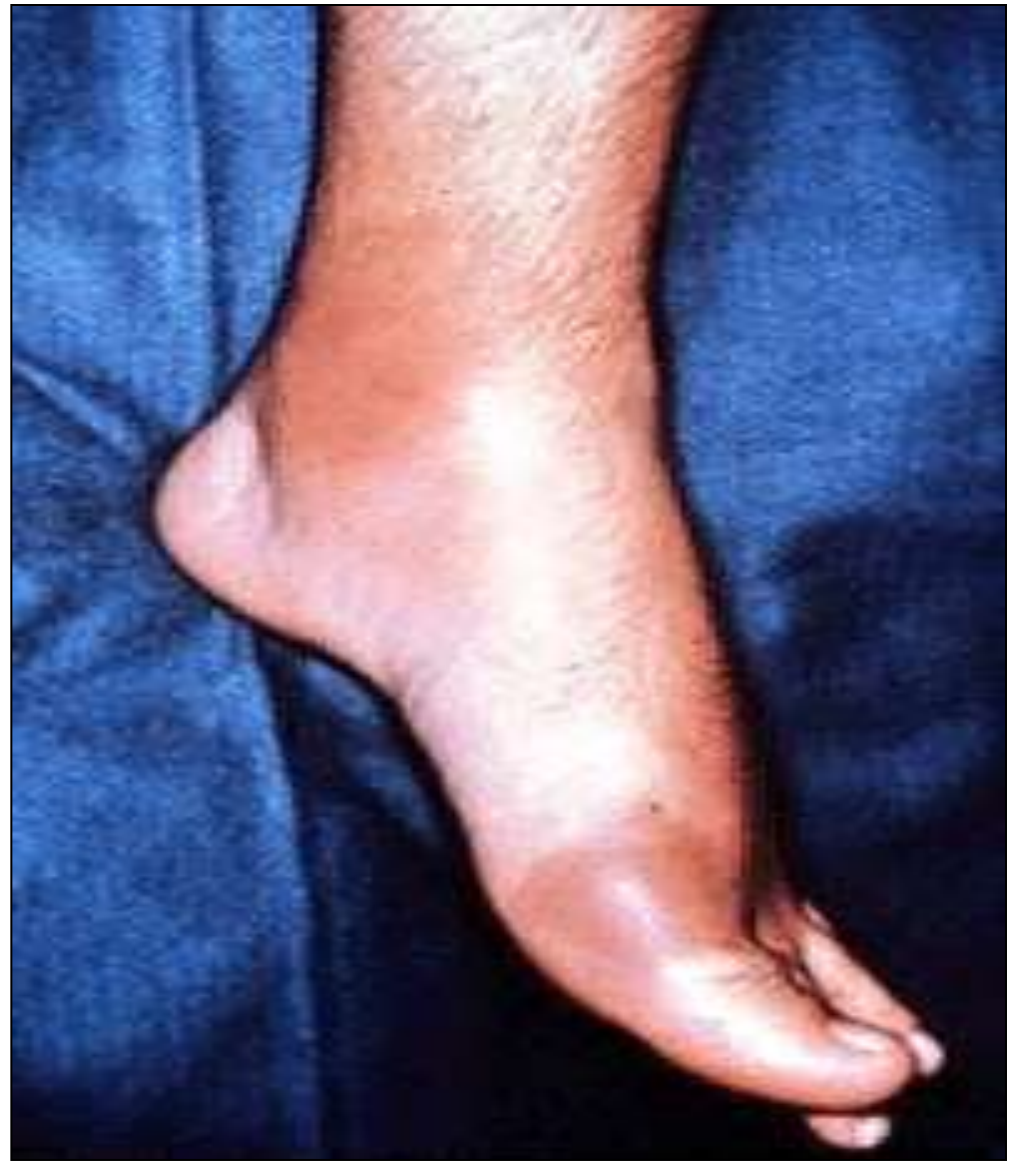
Table 1 – 1977 ACR criteria for the classification of acute gouty arthritis

A. Presence of MSU crystals in joint fluid, and/or
B. Presence of a tophus proven to contain MSU crystals, and/or
C. Presence of 6 of the following 12 clinical, laboratory, and radiographic phenomena:
   a. More than 1 attack of acute arthritis
   b. Development of maximal inflammation within 1 day
   c. Attack of monarticular arthritis
   d. Observation of joint erythema
   e. Pain or swelling in the first MTP joint
   f. Unilateral attack involving the first MTP joint (podagra)
   g. Unilateral attack involving tarsal joint
   h. Suspected tophus
   i. Hyperuricemia
   j. Asymmetrical swelling within a joint on x-ray films
   k. Subcortical cyst without erosions on x-ray films
   l. Negative culture of joint fluid for microorganisms during attack of joint inflammation

ACR, American College of Rheumatology; MSU, monosodium urate; MTP, metatarsophalangeal.

Acute Gout

- Often presents as involvement of a single joint or multiple joints in the lower extremities: first metatarsophalangeal (podagra; 50% of people with gout), midtarsal, ankle and knee joints
- Characterized by pain, erythema, swelling and warmth. Can have desquamation of skin.
- Can even cause fever and leukocytosis
- Maximal severity reached within 12-24 hours
- Even without treatment, attacks subside within days to several weeks



Chronic Gout

- Characterized by chronic arthritis and tophi, resulting in chronic inflammatory and destructive changes

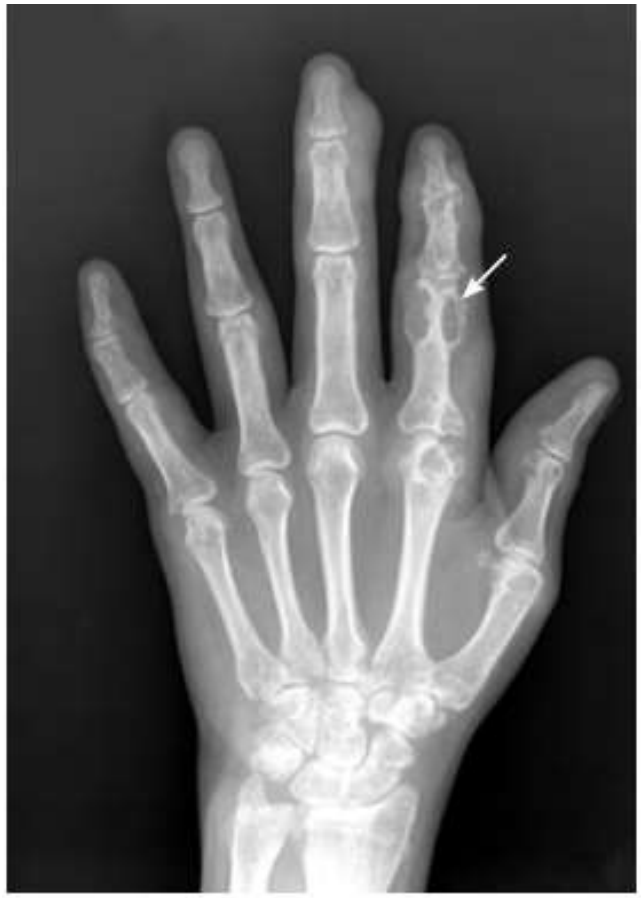
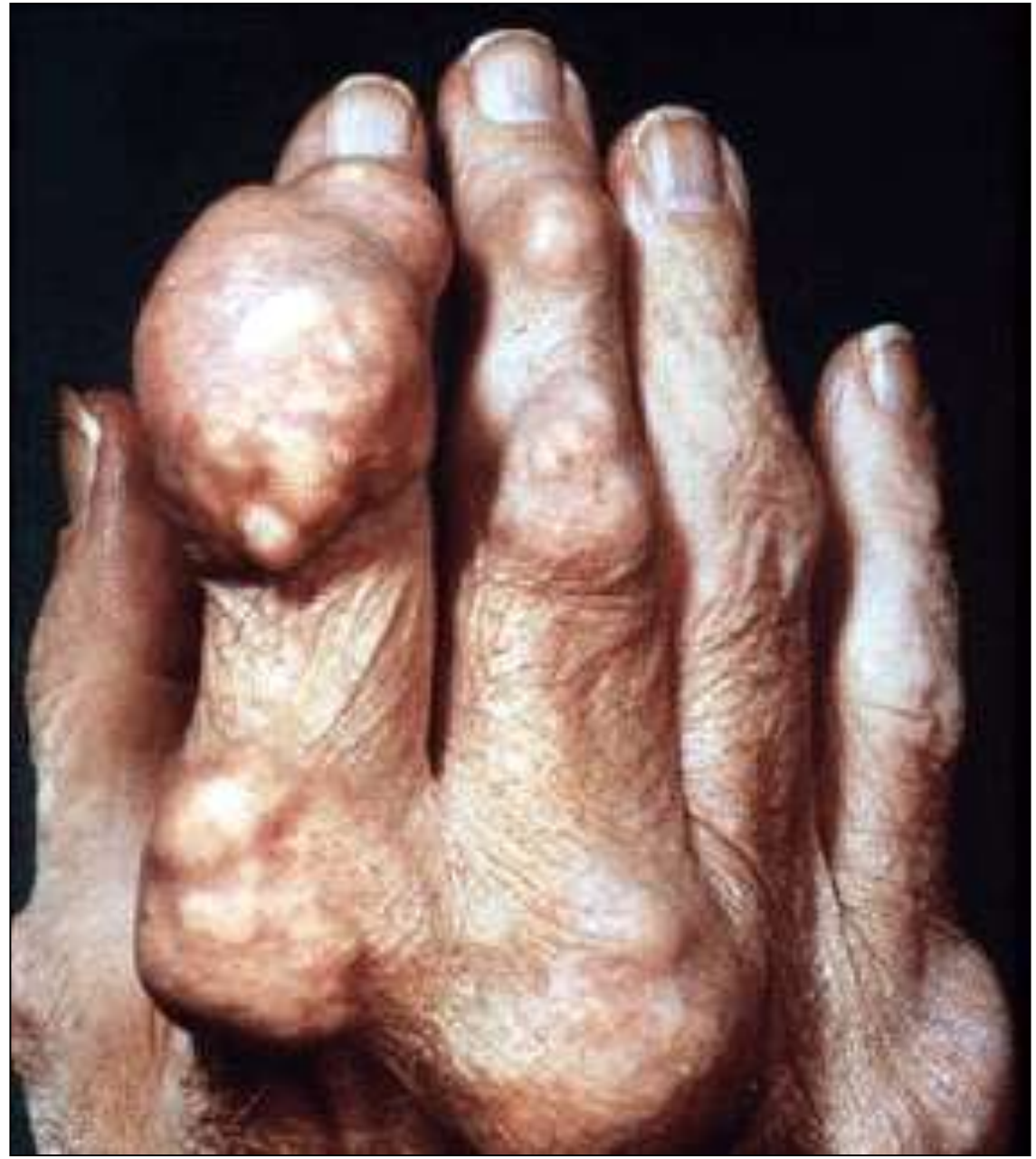


Figure 1. Plain radiograph showing severe tophaceous gout with erosions *(arrow)* around the proximal phalanx.



Renal Complications

- Nephrolithiasis
  - Risk factors: increase uric acid excretion, reduced urine volume, and low urine pH

- Chronic urate nephropathy
  - Urate crystals can deposit in renal medullary interstitium producing inflammatory changes and fibrosis
  - Clinical features are non specific: renal function impairment, bland urinary sediment, mild proteinuria and serum urate concentrations often higher than expected for the degree of renal impairment.
  - Biopsy confirms diagnosis

DIFFERENTIAL DIAGNOSIS FOR MONOARTHRITIS

- Gout
- Pseudogout
- Septic arthritis
- Reactive arthritis
- Trauma
- Beginning polyarthritis

DDX: Pseudogout and Septic Arthritis

Table 2. Differential Diagnosis of Acute Gout

		Synovial fluid findings			
Diagnosis	*Joint distribution*	*WBC count**	*Gram stain/ culture*	*Synovial fluid crystals†*	*Radiography findings*
Gout	Lower extremities: metatarsophalangeal, midtarsal, or knee joints; initial attacks may be less common in upper extremities	2,000 to 50,000 per mm^3 (2×10^9 to 50×10^9 per L)	Negative	Needle shaped, negative birefringence	Acute: asymmetric swelling Chronic: periarticular erosions with overhanging edges
Pseudogout (calcium pyrophosphate deposition disease)	Knee, wrist, or first metatarsophalangeal	2,000 to 50,000 per mm^3	Negative	Rhomboid shaped, weak positive birefringence	Soft tissue swelling, chondrocalcinosis (calcification of cartilage)
Septic arthritis	Knee is most commonly involved (may be any joint distribution)	> 50,000 per mm^3	Positive	No crystals	Joint effusion; radiography results otherwise normal early in the disease

NOTE: *This table applies to immunocompetent patients.*

WBC = white blood cell.

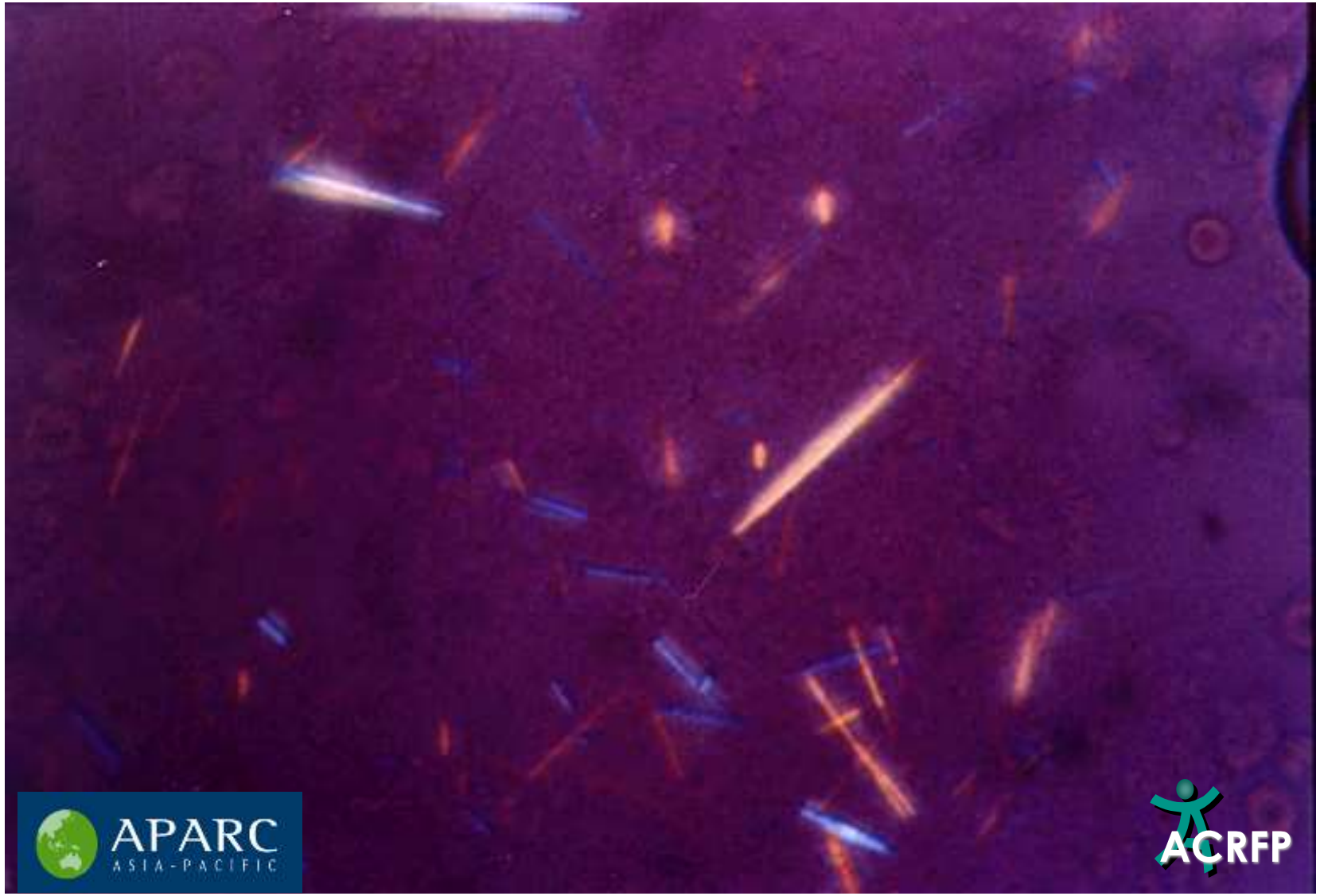
**—The synovial fluid WBC count should not be used alone to exclude infection.*

†—Septic arthritis may coexist with crystalline arthritis.

Diagnosis

- Arthrocentesis should be done in patients in whom the diagnosis has not been previously established .
- Labs: cell count with differential, gram stain, culture, examination for crystals under polarized light microscopy

Monosodium urate crystals



Gout Management Approach

INITIATE
(acute flare)

- Treat acute flare rapidly with anti-inflammatory agent

RESOLVE
(urate-lowering therapy)

- Initiate urate-lowering therapy to achieve sUA <6
- Use concomitant anti-inflammatory prophylaxis for up to 6 mo to prevent mobilization flares

MAINTAIN
(treatment to control sUA)

- Continue urate lowering therapy to control flares and avoid crystal deposition
- Prophylaxis use for at least 3-6 months until sUA normalizes

Treatment

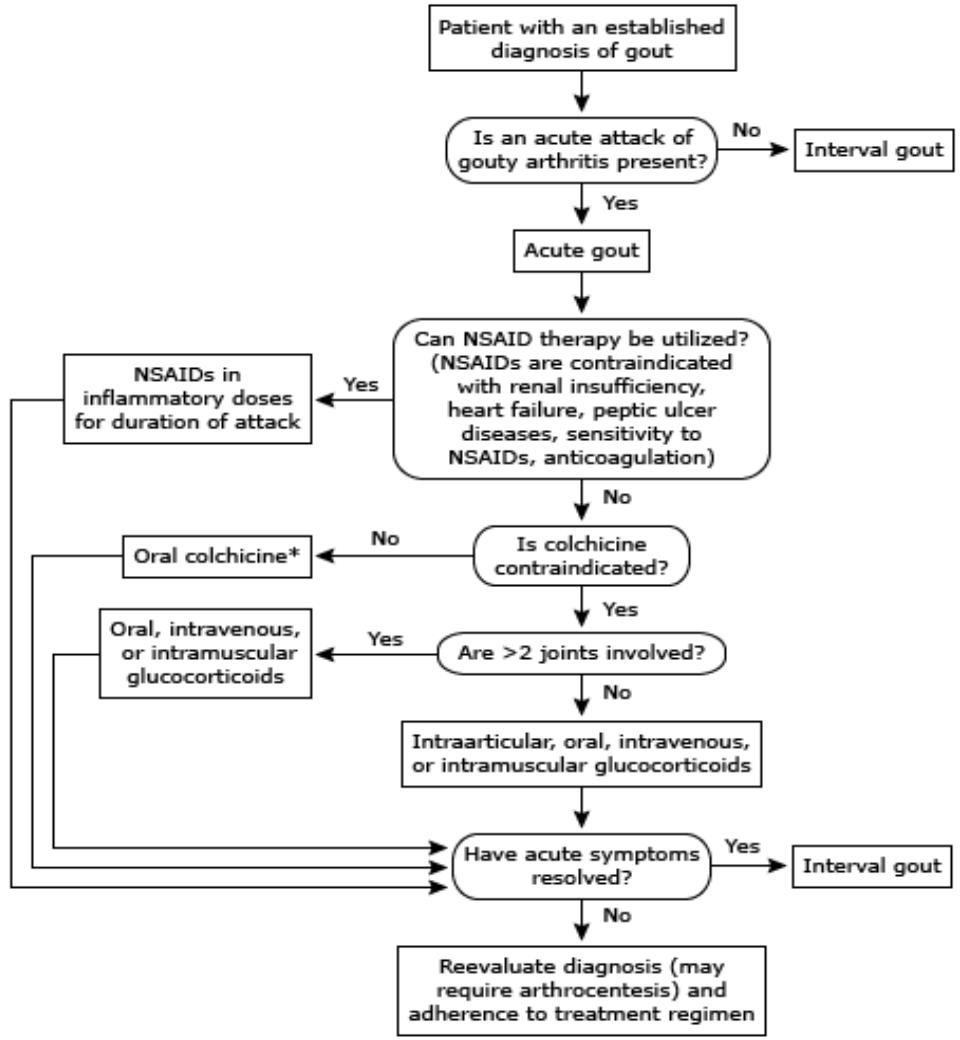
Table 4. Pharmacotherapy for Acute Gout

Therapy/dosing	*Cautions*	*Comments*
NSAIDs Indomethacin (Indocin), 50 mg three times daily for four to 10 days Naproxen (Naprosyn), 500 mg twice daily for four to 10 days Sulindac (Clinoril), 200 mg twice daily for four to 10 days	Use with caution in older patients and in patients with renal insufficiency, heart failure, peptic ulcer disease, or liver disease and in those receiving anticoagulation therapy	Any NSAID is effective
Corticosteroids Prednisone, 20 to 40 mg daily for two or three days, then taper over 10 to 14 days Intra-articular methylprednisolone (Depo-Medrol), one 20- to 40-mg dose Intramuscular methylprednisolone, one 80- to 120-mg dose	Avoid in patients with joint sepsis and use cautiously in patients with diabetes	Intra-articular therapy may be the treatment of choice if only one or two accessible joints are involved
Colchicine, 0.6 mg orally two or three times daily Suggested renal dosing (based on creatinine clearance): > 50 mL per minute (0.83 mL per second): 0.6 mg twice daily 35 to 50 mL per minute (0.58 to 0.83 mL per second): 0.6 mg daily 10 to 34 mL per minute (0.17 to 0.57 mL per second): 0.6 mg every two or three days < 10 mL per minute (0.17 mL per second): avoid	Avoid in patients with severe renal or hepatic impairment because it can lead to bone marrow suppression and neuromyopathy	Avoid intravenous use; best if used within the first 24 hours of the attack; the most common adverse effects are nausea, vomiting, and diarrhea; reduce the dosage in older patients

NOTE: *NSAIDs or corticosteroids are first-line therapies, depending on comorbidities; colchicine is an effective second-line therapy.*

NSAID = nonsteroidal anti-inflammatory drug.

Initial management of acute gout



NSAIDs: nonsteroidal antiinflammatory drugs.
* Adjust dose in patients with renal insufficiency.

Adapted and revised from: Algorithms for the diagnosis and management of musculoskeletal complaints. Am J Med 1997; 103:49S. Original figure modified for this publication. Illustration used with the permission of Elsevier Inc. All rights reserved.

Treatment of acute gouty

- **Colchicine** : reduces pain, swelling, and inflammation; pain subsides within 12 hrs and relief occurs after 48 hrs
- **NSAID or COX-2 inhibitor**
- **Glucocorticoid** (Prednisone 30-60 mg/day for 10-14 days)
- **Allopurinol** or **Probenecid** : decreases the serum uric acid level given after the acute attack resolves

Treatment

Table 5. Pharmacologic Options for Urate-Lowering Therapy in Patients with Chronic Gout

Therapy/dosing	*Cautions*	*Comments*	*Monthly cost (generic)**
Allopurinol (Zyloprim), 50 to 300 mg daily (maximal daily dosage: 800 mg) Suggested initial daily renal dosing (based on creatinine clearance): ≥ 90 mL per minute (1.50 mL per second): 300 mg 60 to 89 mL per minute (1.00 to 1.49 mL per second): 200 mg 30 to 59 mL per minute (0.50 to 0.98 mL per second): 100 mg 10 to 29 mL per minute (0.16 to 0.48 mL per second): 50 to 100 mg < 10 mL per minute (0.16 mL per second): use very cautiously	May precipitate acute gout, hypersensitivity syndrome, or mild rash; avoid using with azathioprine (Imuran); interacts with warfarin (Coumadin)	Do not initiate until four to six weeks after an acute attack; concurrent prophylaxis with colchicine (0.6 mg once or twice daily for six months) may prevent flare-ups; titrate dose until the uric acid level is less than 6 mg per dL (355 µmol per L); continue therapy during acute flare-ups	Thirty 300-mg tablets: $34 (6 to 18)
Probenecid, initially 250 mg twice daily, gradually titrated to 500 mg to 2 g per day	May precipitate acute gout, nephrolithiasis, gastrointestinal upset, or rash; modifies renal handling of other drugs; use cautiously with heparin	Maintain hydration (about 2 L per day); avoid using with low-dose aspirin; ineffective if creatinine clearance is less than 50 mL per minute	Sixty 500-mg tablets†: (59 to 131)
Febuxostat, 80 mg daily	Avoid in patients with hepatic impairment	Investigational medication not yet approved by the U.S. Food and Drug Administration	—

NOTE: *Urate-lowering therapy should not commence until the acute phase of gout has completely resolved because fluctuations in serum uric acid will exacerbate the inflammatory process.*

- Acute gout flares are treated with 1 tablet of colchicine hourly until the patient develops diarrhea or gets better.

Maintenance Doses of Allopurinol for Adults based on CrCl

Stage 1 renal damage with normal GFR (GFR > 90 ml/min)
Stage 2 Mild CKD (GFR = 60-89 ml/min)
Stage 3 Modererate CKD (GFR = 30-59 ml/min)
Stage 4 Severe CKD (GFR = 15-29 ml/min)
Stage 5 End Stage CKD (GFR <15 ml/min)

CrCl (mL/min)	Maintenance Dose of Allopurinol
0	100mg every 3d
10	100mg every 2d
20	100mg
40	150mg
60	200mg
80	250mg
100	300mg
120	350mg
140	400mg

Hande KR, et al. *Am J Med*, 1984.

2012 ACR Management Guidelines for Acute Gouty Arthritis

- The choice of pharmacologic agent depends on severity of the attack
  - Monotherapy for mild/moderate attack
  - Combination therapy for severe attack or those refractory to monotherapy
- Acceptable combination therapy approaches include
  - Colchicine and NSAIDS
  - Oral steroids and colchicine
  - Intra-articular steroids with all other modalities
- Continue current therapy during flare
- Patient education on signs of flare for self treatment

Kanna D, et al. Arthritis Care Res (Hoboken). 2012 Oct;64(10):1447-61

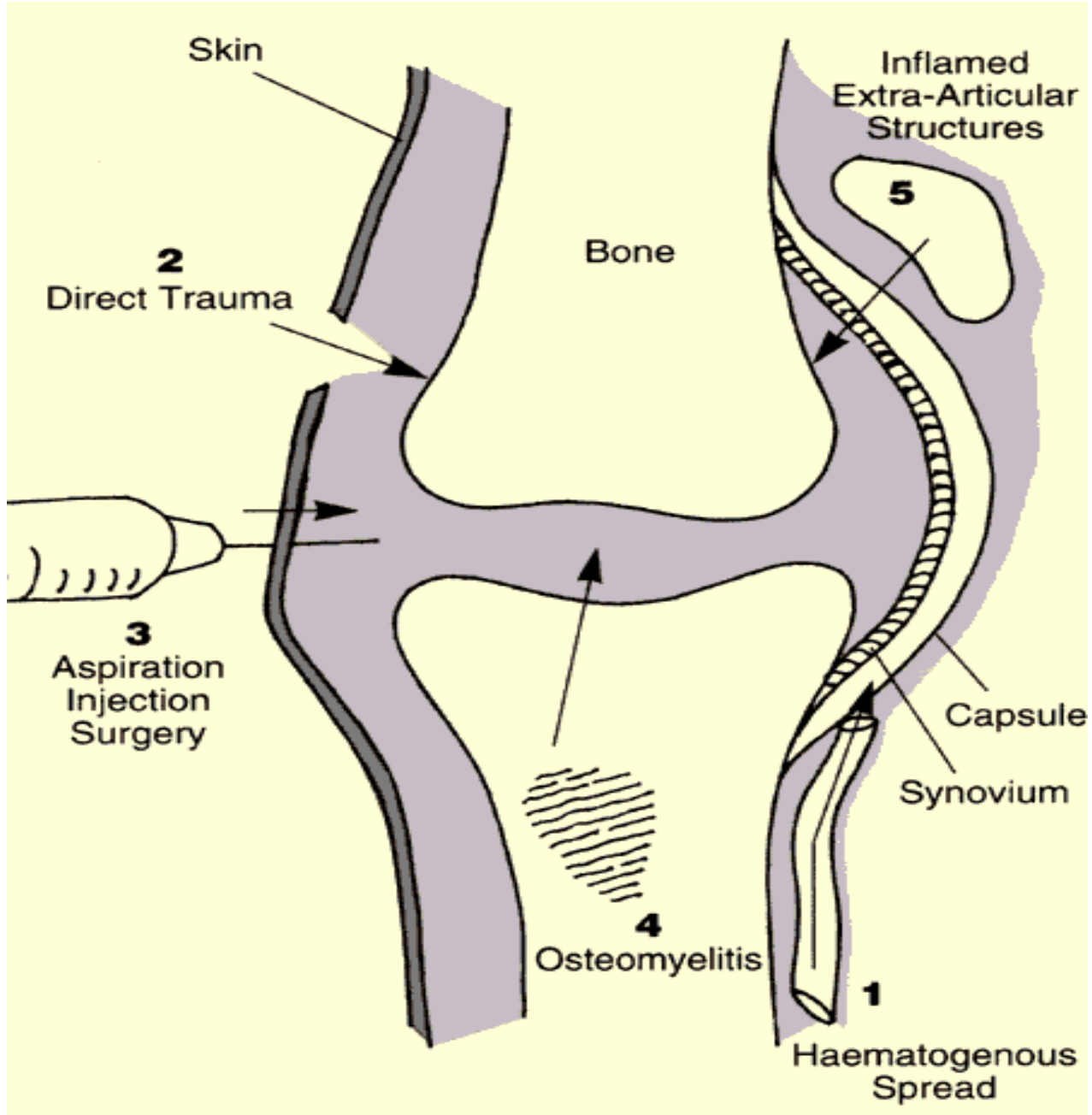
Septic arthritis

septic arthritis

- is an inflammatory joint disease caused by bacterial, viral, and fungal infection.

Route of infection

- dissemination of pathogens via the blood, from distant site…. (most common)
- dissemination from an acute osteomylitic focus
- dissemination from adjacent soft tissue infection,
- entry via penetrating trauma
- entry via iatrogenic means


Skin
Inflamed
Extra-Articular
Structures
5
Bone
2
Direct Trauma
3
Aspiration
Injection
Surgery
Capsule
Synovium
4
Osteomyelitis
1
Haematogenous
Spread

Etiology

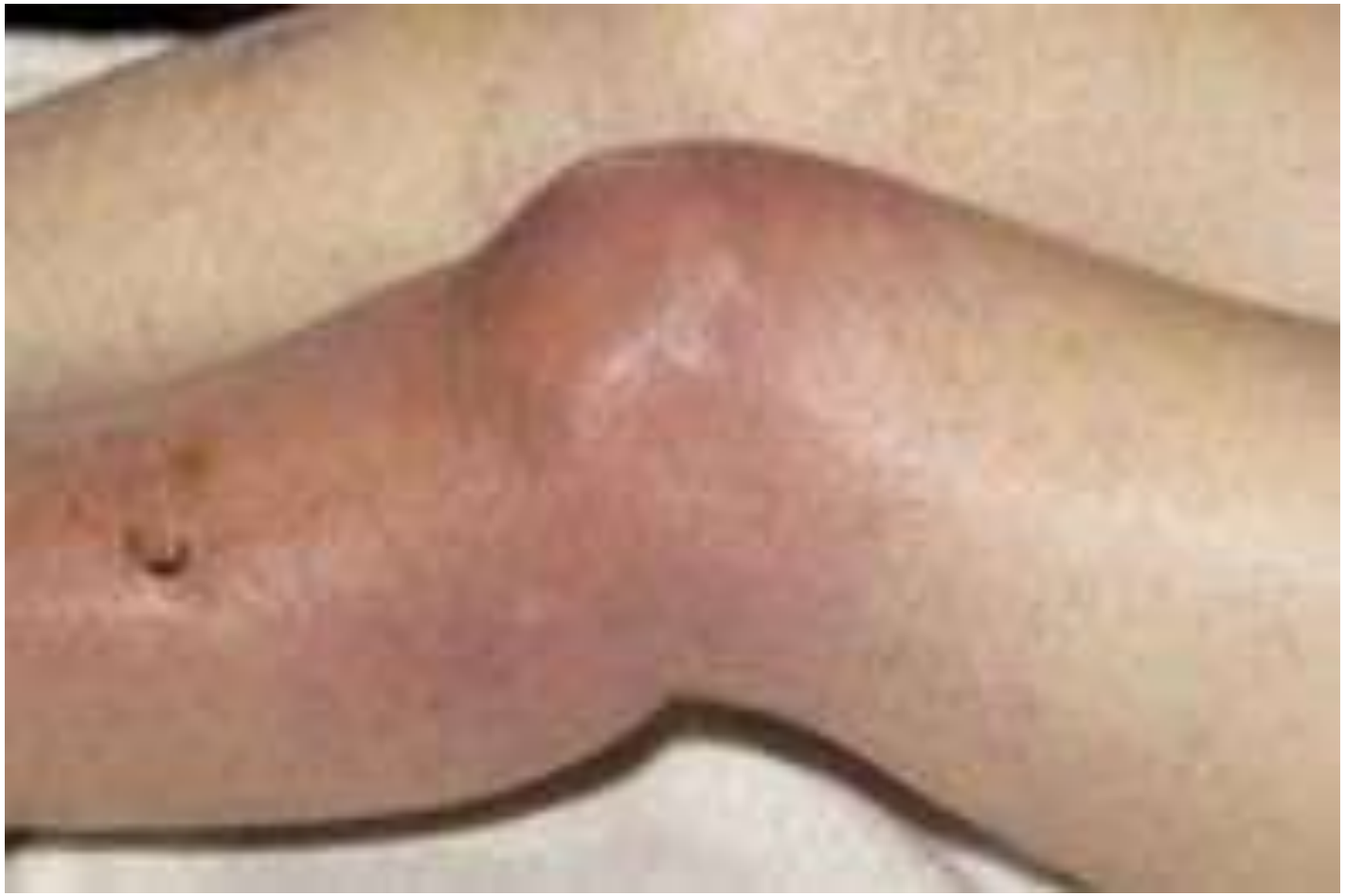
- *Staphylococcus aureus* (40-70% of all cases)
- *Streptococcus* group A
- Other group *Streptococcus*
- *Haemophilus influenzae*
- *Salmonella*
- *Mycobacteria*

Pathology

- There is an acute synovitis with a purulent joint effusion and Synovial membrane becomes edematous, swollen and hyperemic, and produces increase amount of cloudy exudates contains leukocytes and bacteria
- As infection spread through the joint, articular cartilage is destroyed by bacterial and cellular enzymes.
- If the infection is not arrested the cartilage may be completely destroyed.
- Pus may burst out of the joint to form abscesses and sinuses.
- The joint may be become pathologically dislocated.

Physical examination

1. More common at lower extremities (50% knee)
2. Decreased or absent rang of motion.
3. Signs of inflammation: joint swelling, warm, tenderness and erythema.
4. Risk factors : chronic rheumatic disease, prosthetic joint, prior joint injection, and chronic disease (DM, CKD, liver cirrhosis, alcoholism, cancer etc)



Investigation

Lab studies:

- The diagnosis can usually be confirmed by joint aspiration and immediate microbiological investigation of the fluid.
- Blood culture may be positive in about 50% of proven cases.
- Non specific features of acute inflammation-leucocytosis,ESR,CRP-are suggestive but not diagnostic .

SYNOVIAL FLUID FINDINGS

	Normal	Osteoarthritis	Rheumatoid and other inflammatory arthritides	Septic arthritis
Gross appearance	Clear	Clear	Opaque	Opaque
Volume (ml)	0–1	1–10	5–50	5–50
Viscosity	High	High	Low	Low
Total white cell count/mm^3	<200	200–10,000	5000–75,000	>50,000
% Polymorphonuclear cells	<25%	<50%	>50%	>75%

Imaging studies

1-Plain x-ray:

- The appearance of significant x-ray findings depends upon the duration and virulence of infection.
- Plain radiography findings are generally nonspecific and may reveal only soft tissue swelling ,widening of the joint space (due to the effusion), and periarticular osteoporosis during the first 2 weeks.
- Later ,when the articular cartilage is attacked ,the joint space is narrowed.(persistent subluxation, destructive arthritis).

DIFFERENTIAL DIAGNOSIS

- ***Osteomyelitis***: near a joint may be indistinguishable from septic arthritis ;the safest is to assume that both are present.
- ***An acute haemarthrosis*** :either post-traumatic or due to a haemophilic bleed ,can closely resemble infection. The history is helpful and joint aspiration will resolve any doubt.
- ***Transient synovitis(irritable joint)*** in children: causes symptoms and signs which are less acute ,but there is always the that this is the beginning of an infection.
- ***Gout and pseudogout*** in adults :aspirated fluid may look turbid but the presence of urate or pyrophosphate crystals will confirm the diagnosis.
- ***Rheumatic fever***

complication

- ***Dislocation:*** a tense effusion may cause dislocation
- ***Epiphyseal destruction***: in neglected infants the largely cartilaginous epiphysis may be destroyed ,leaving an unstable pseudarthrosis.
- ***Growth disturbance:*** physeal damage may result in shortening or deformity
- ***Ankylosis:*** if articular cartilage is eroded healing may lead to ankylosis
- ***Secondary osteoarthritis***
- ***Osteomyleitis/abcess/sinus***

Treatment

- ***General Measures:***

The first priority is to aspirate the joint and examine the fluid, treatment is then started without further delay.

- Analgesics and splinting of the involved joint in the position of maximal comfort alleviate pain.
- Fluid replacement and nutritional support may be required.
- Other foci of infection and any coexisting medical conditions must be identified and treated appropriately.

Treatment

- Adequate intravenous antibiotic 2 weeks or signs improve, then 4 weeks orally
- Pus drainage (needle aspiration, arthroscopy or surgical)
- Protected from weight bearing 6 weeks, early passive motion

Drainage:

Indication of Surgical Drainage:

1-Joints that do not respond to antimicrobial therapy and daily arthrocentesis

2-. Any joint with limited accessibility, including the sternoclavicular or the hip joint

3-Patients with underlying disease, including diabetes, rheumatoid arthritis, immunosuppression, or other systemic symptoms, should be treated more aggressively with earlier surgical intervention

Thank you