Gout and Osteoarthritis

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Case history #1

- A 54-year-old man complains of severe pain and swelling in his right first toe that developed overnight. He is limping because of the pain and states that this is the most severe pain he has ever had ('even covering my foot with the bed sheet hurts'). He has had no previous episodes. His only medication is hydrochlorothiazide for hypertension. He drinks 2 to 3 beers a day.

- On examination, he is obese. There is swelling, erythema, warmth, and tenderness of the right first toe. There is also tenderness and warmth with mild swelling over the mid foo

Case history #2

- An 85-year-old man presents with several days of swelling and severe pain in both hands limiting his ability to use his walking frame. He has a history of gout but has not experienced these symptoms before.

- On examination, he has a temperature of 37.8°C (100.1°F). There is diffuse warmth, mild erythema, and pitting edema over the dorsum of both hands. There is tenderness and limited hand grip bilaterally. There are multiple nodules around several of the proximal interphalangeal and distal interphalangeal joints, and effusion and tenderness in his left olecranon bursa with palpable nodules.

SPECTRUUM of crystalline arthritis

Variety of crystals may be associated with joint or soft tissue problems.

Commonly seen Arthropathies: Crystal	Association/Arthropathy
Calcium pyrophosphate dihydrate (CPPD)	Acute pseudogout; A variety of chronic inflammatory or degenerative arthritis; Chondrocalcinosis
Basic calcium phosphate Hydroxy-apatite (BCP)	Calcific periarthritis; Acute/Chronic inflammatory arthritis; Destructive arthropathy; Soft-tissue calcinosis
Monosodium Urate (MSU)	Acute/chronic gouty arthritis; Renal calculi; Tophi

Uncommon Arthropathies: Crystal	Association/Arthropathy
Calcium oxalate aluminum phosphate	Acute arthritis in patients renal dialysis on
Cholesterol	Chronic synovial effusions in RA/OA
Xanthine	Acute arthritis (rare); Renal calculi; Asymptomatic deposition in muscles
Cysteine/cystine	Acute arthritis
Charcot-Leyden, (lysophospho-lipase)	Synovial fluid and tissues with eosinophilia

Gout

•A disease related to the deposition of monosodium urate monohydrate (MSU) crystals in various connective tissues and
•joints.

•The annual incidence of gout in the US in people over 50 years of age is 1.6 per 1000 in men and 0.3 per 1000 in women

•Hyperuricemia: serum or plasma urate concentrations exceeding 6.8 mg/dL (approximate limit of urate solubility).

•Long periods of asymptomatic hyperuricaemia may precede clinical symptoms.

Gout and hyperuricemia

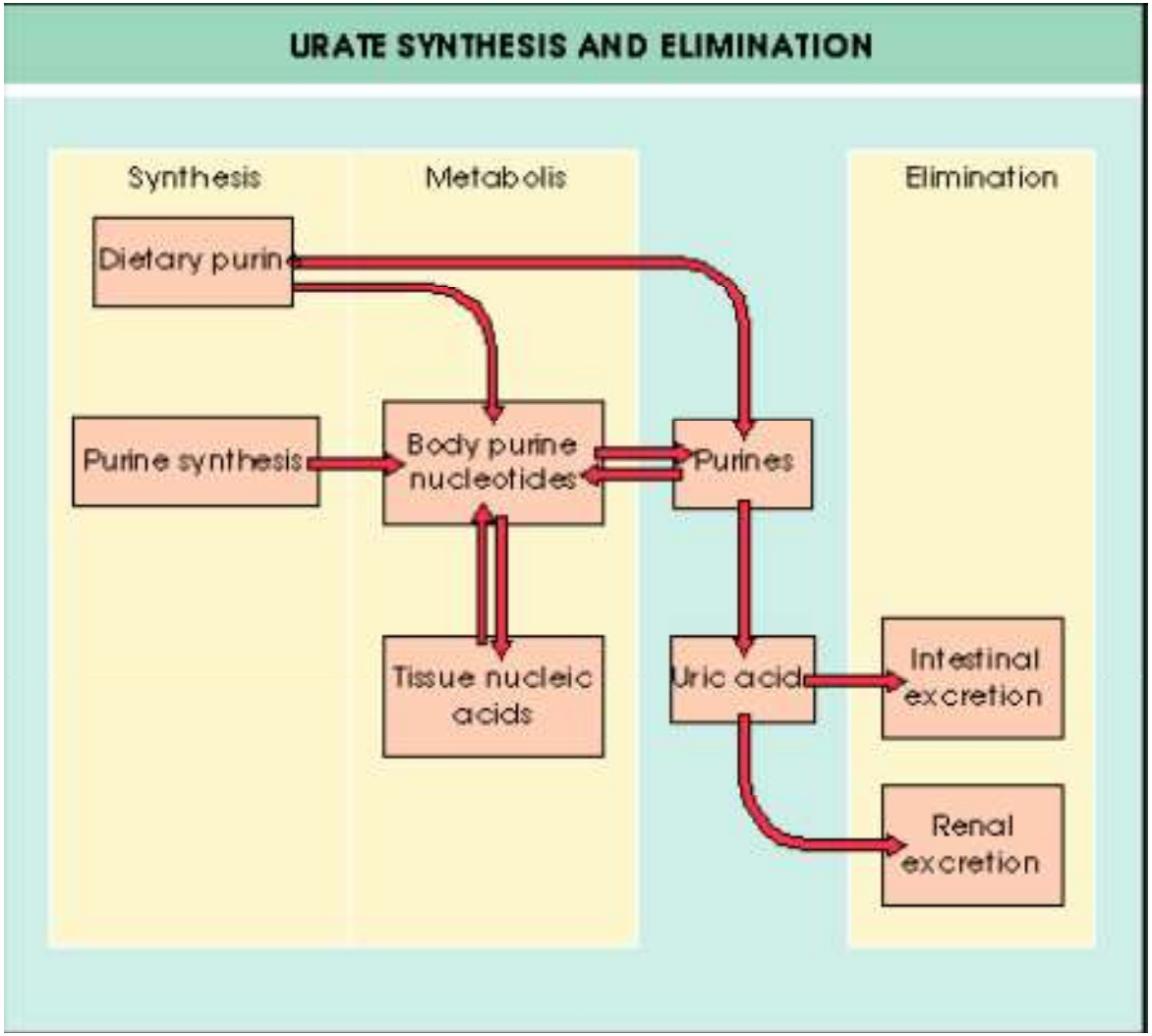
- Urate is a metabolite of purines and the ionised form of uric acid (a weak acid at a physiological pH)>>>uric acid exists mostly as urate.

- Hyperuricaemia is due to renal under-excretion of urate in 90% of cases and to over-production in 10%

- Incidence of gout increases with urate level.

- The annual incidence of gout in men is :

✓ 0.4% for a urate level of 7 to 7.9 mg/dL,
✓ 0.8% for 8 to 8.9 mg/dL,
✓ 4.3% for 9 to 9.9 mg/dL, and
✓ 7% for levels >10 mg/dL.


URATE SYNTHESIS AND ELIMINATION
Synthesis
Metabolis
Elimination
Dietary purine
Purine synthesis
Body purine nucleotides
Purines
Tissue nucleic acids
Uric acid
Intestinal excretion
Renal excretion

Causes of secondary hyperuricemia due to increased purine biosynthesis and/or urate production

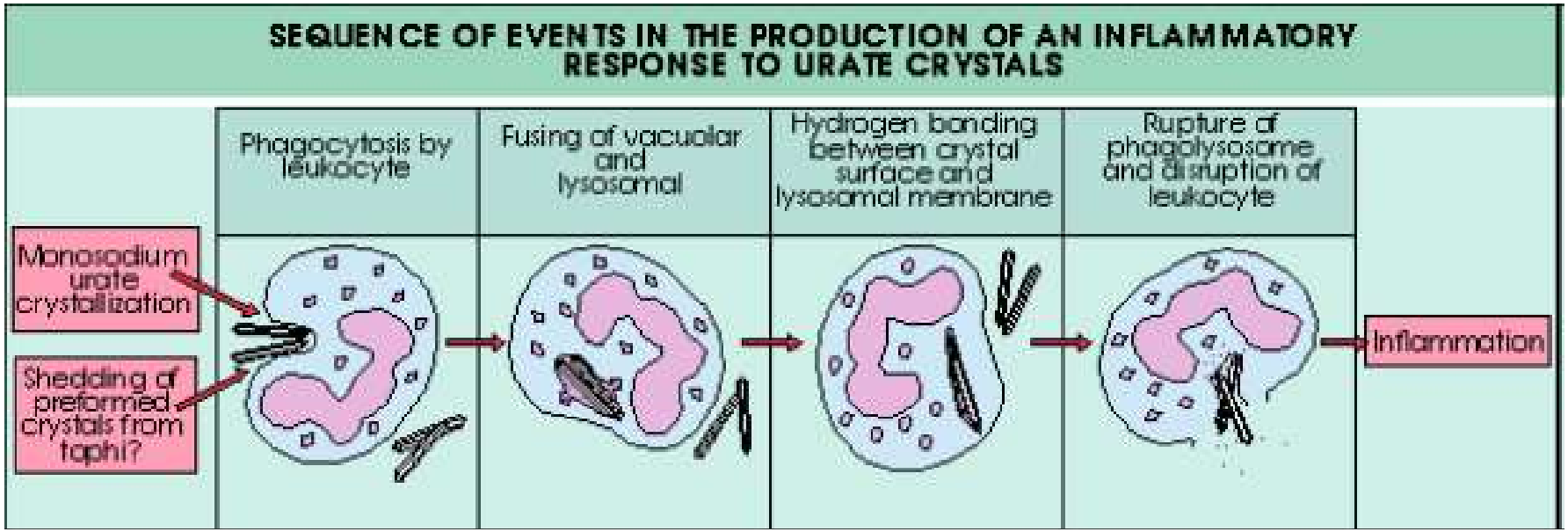
Inherited enzyme defects leading to purine overproduction
Hypoxanthine-guanine phosphoribosyltransferase deficiency
Phosphoribosylpyrophosphate synthetase overactivity
Glucose-6-phosphatase deficiency (glycogen storage disease, type I)
Clinical disorders leading to purine and/or urate overproduction
Myeloproliferative disorders
Lymphoproliferative disorders
Malignancies
Hemolytic disorders
Psoriasis
Obesity
Tissue hypoxia
Down syndrome
Glycogen storage diseases (types III, V, VII)
Drug-, diet-, or toxin-induced purine and/or urate overproduction
Ethanol
Excessive dietary purine ingestion
Pancreatic extract
Fructose
Vitamin B12 deficiency
Nicotinic acid
Ethylamino-1,3,4-thiadiazole
4-amino-5-imidazole carboxamide riboside
Cytotoxic drugs

Causes of secondary hyperuricemia due to decreased renal uric acid clearance

Clinical disorders
Chronic renal insufficiency of any form
Lead nephropathy (saturnine gout)
Effective volume depletion (eg, fluid losses, heart failure)
Diabetic or starvation ketoacidosis
Lactic acidosis
Preeclampsia
Obesity
Hyperparathyroidism
Hypothyroidism
Sarcoidosis
Chronic beryllium disease
Familial juvenile hyperuricemic nephropathy (FJHN)
Medullary cystic kidney disease (MCKD)
Glomerulocystic kidney disease
Genetic polymorphisms in urate transporter genes encoding transporters impaired for renal uric acid clearance
SLC2A9
SLC22A12
ABCG2
SLC17A1
Drug- or diet-induced
Diuretics (thiazides and loop diuretics)
Cyclosporine and tacrolimus
Low-dose salicylates
Ethambutol
Pyrazinamide
Ethanol
Levodopa
Methoxyflurane
Laxative abuse (alkalosis)
Salt restriction

Inflammatory response to urate crystals

- The solubility of urate in the joints depends on temperature, pH, non-aggregated proteoglycans, and other factors.
- Urate crystals in the joint interact with undifferentiated phagocytes and trigger an acute inflammatory response by inducing TNF-alpha and activating signal pathways and endothelial cells.
- TNF-alpha, interleukin (IL)-8, and other chemokines lead to neutrophil adhesion to endothelium, influx, and amplification, resulting in neutrophilic synovitis
- Urate crystals activate NALP3 inflammasome that induces the secretion of IL-1beta, which plays a role in the gout inflammatory reaction


SEQUENCE OF EVENTS IN THE PRODUCTION OF AN INFLAMMATORY RESPONSE TO URATE CRYSTALS
Phagocytosis by leukocyte
Fusing of vacuolar and lysosomal
Hydrogen bonding between crystal surface and lysosomal membrane
Rupture of phagolysosome and disruption of leukocyte
Monosodium urate crystallization
Shedding of preformed crystals from tophi?
Inflammation

Inflammatory response to urate crystals

- Spontaneous resolution of gout attack results from clearance of urate crystals by differentiated phagocytes, coating of the crystals with proteins, neutrophilic apoptosis, and inactivation of inflammatory mediators

- Urate crystals can induce chronic inflammation, leading to synovitis, cartilage loss, and bone erosions.

- Urate crystals induce chondrocytes to produce metalloproteinase and nitric oxide, which results in cartilage loss, and may cause bone damage by inhibiting osteoblasts

Clinical presentation of hyperuricemia

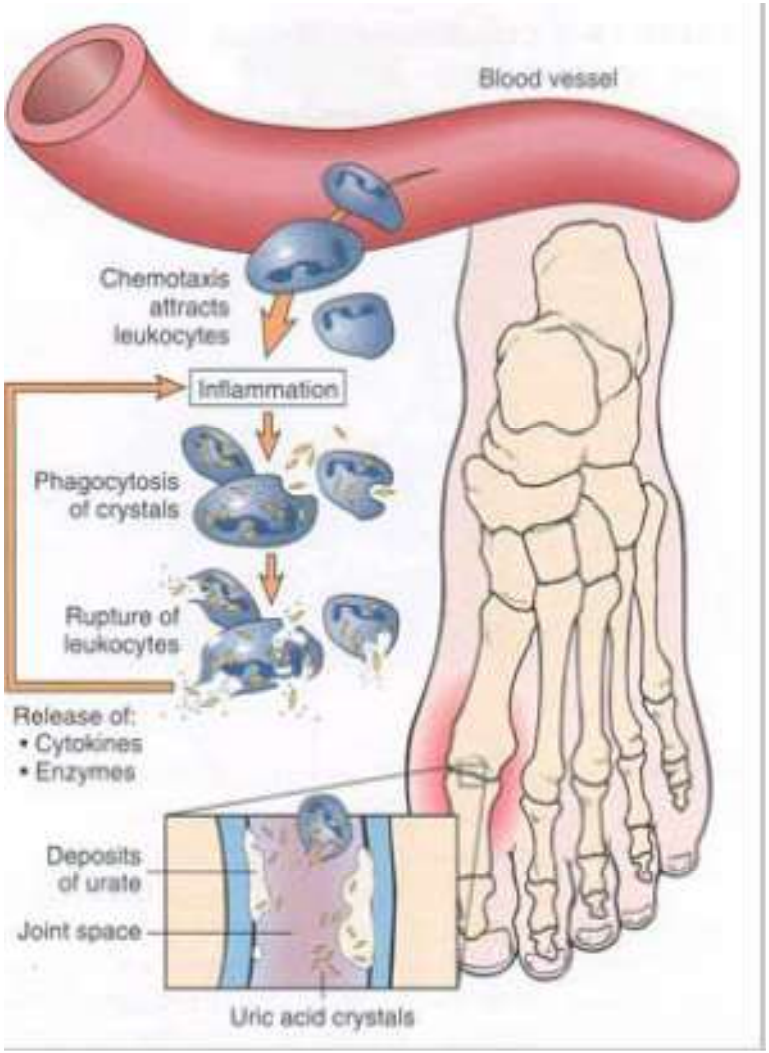
1-Recurrent attacks of acute inflammatory arthritis

2-Chronic arthropathy

3-Accumulation of urate crystals in the form of tophaceous deposits

4-Uric acid nephrolithiasis

- *Chronic nephropathy in gouty patients is most often due to comorbid states*


Blood vessel
Chemotaxis attracts leukocytes
Inflammation
Phagocytosis of crystals
Rupture of leukocytes
Release of:
• Cytokines
• Enzymes
Deposits of urate
Joint space
Uric acid crystals

Renal disease and hyperuricemia

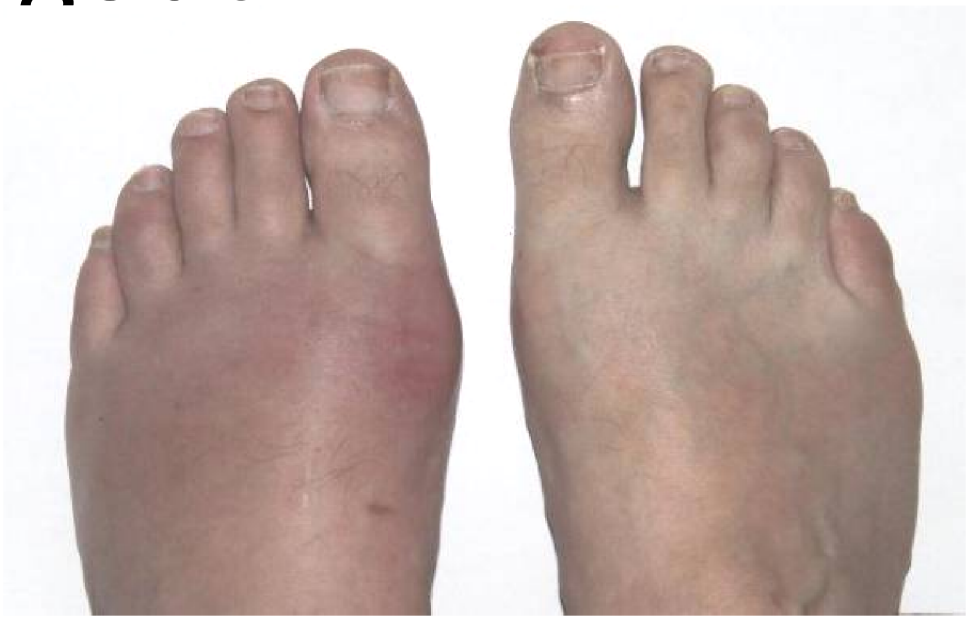
1-Urate nephropathy:
-Formation of urate (MSU) crystals in the renal interstitium associated with renal insufficiency.
Concomitant uncontrolled hypertension often contributed to renal dysfunction.

2. Uric acid nephropathy:
-Acute obstructive uropathy, due to the rapid formation of uric acid crystals in-the collecting tubules
-Acutely ill and dehydrated patient ‘tumour lysis syndrome’.

3. Uric acid nephrolithiasis:
Formation of uric acid calculi in the renal tract.
Elevated urinary uric acid levels, low urine pH and hyperuricaemia

Acute gout

An acute inflammatory arthritis, maybe associated with teno-synovitis; bursitis; and/or cellulitis.



-8X more common in men than women

-The first attack commonly occurs between the third- sixth decades.

-Uncommon in women before the menopause

-Usual course is of recurrent attacks of acute synovitis with inter-critical period in between.

ACUTE GOUT

- The most common joint to be affected is the first metatarso-phalangeal (1st MTP) joint(podagra)
- The initial presentation may be polyarticular in up to 10% of patients especially in elderly females on thiazide diuretics.
- A typical attack is sudden in onset and awakens the patient from sleep.
- The joint becomes red, hot, and swollen mimicking a local infection

ACUTE GOUT

- May be associated with constitutional symptoms
- Labs may show leucocytosis and elevated ESR
- Elevated uric acid may be present
- Occasionally acute attacks may occur with normal serum uric acid levels

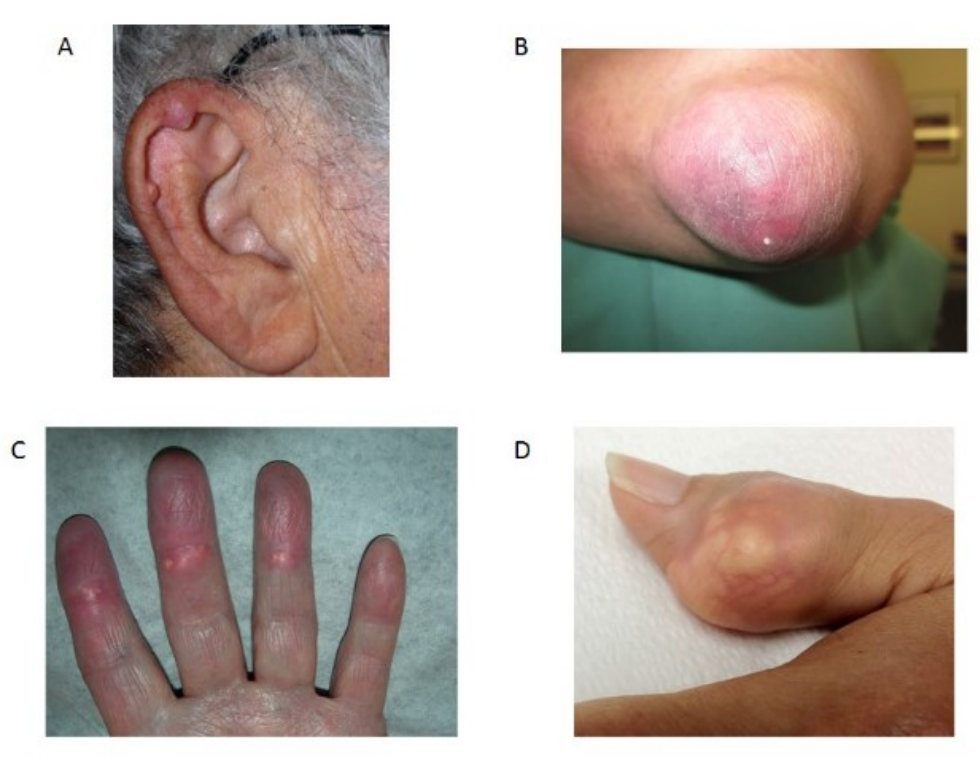
Triggers of gout attacks

Disturbances in extracellular fluid urate concentrations :

Trauma, surgery, starvation, fatty foods, dehydration, and ingestion of drugs affecting (raising or lowering) serum urate concentrations (eg, allopurinol, uricosuric agents, thiazide or loop diuretics, and low-dose aspirin).

4. Clinical Evidence of Tophus

- Draining or chalk-like subcutaneous nodule under transparent skin with overlying vascularity
- Typical locations:
  - Ears, olecranon bursa, finger pads, tendon (e.g., Achilles)

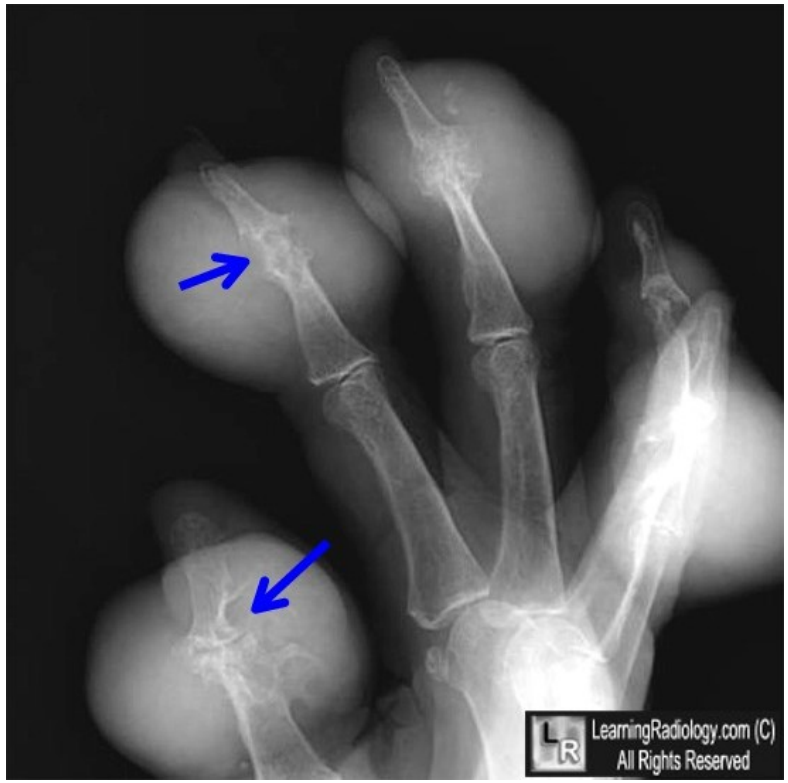


Scored as absent or present

Tophaceous gout

A chronic, erosive, deforming arthritis, associated with peri-articular and subcutaneous urate deposit(tophi)

May cause dactylitis



Diagnosis of gout

- Crystal identification is the golden standard test
- Serum urate level
- Radiology
- Synovial fluid: MSU crystals
- Histology: MSU crystals

Serum Urate level

- Higher in males than in females
-
- Falsely low during an acute attack

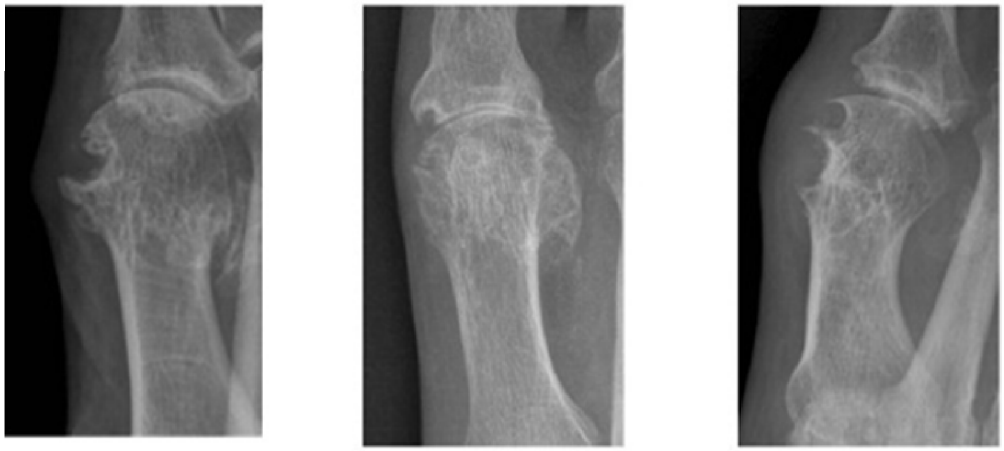
- Aim: reduce level to < 6 mg/dl

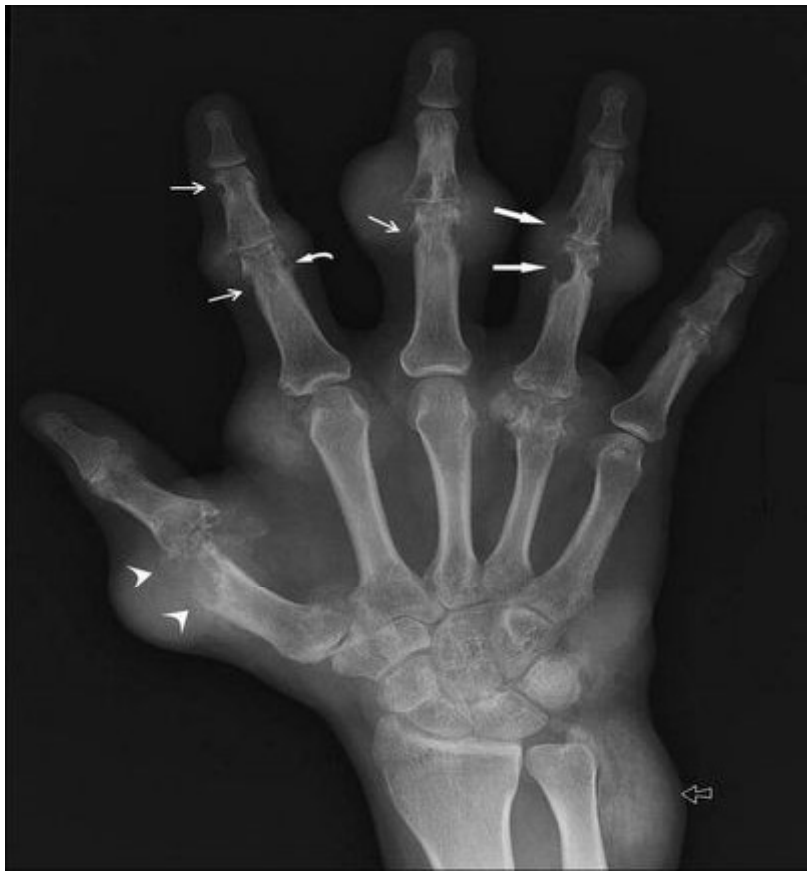
- The risk of gout increases with the degree and duration of hyperuricaemia.

Radiographic changes in gout

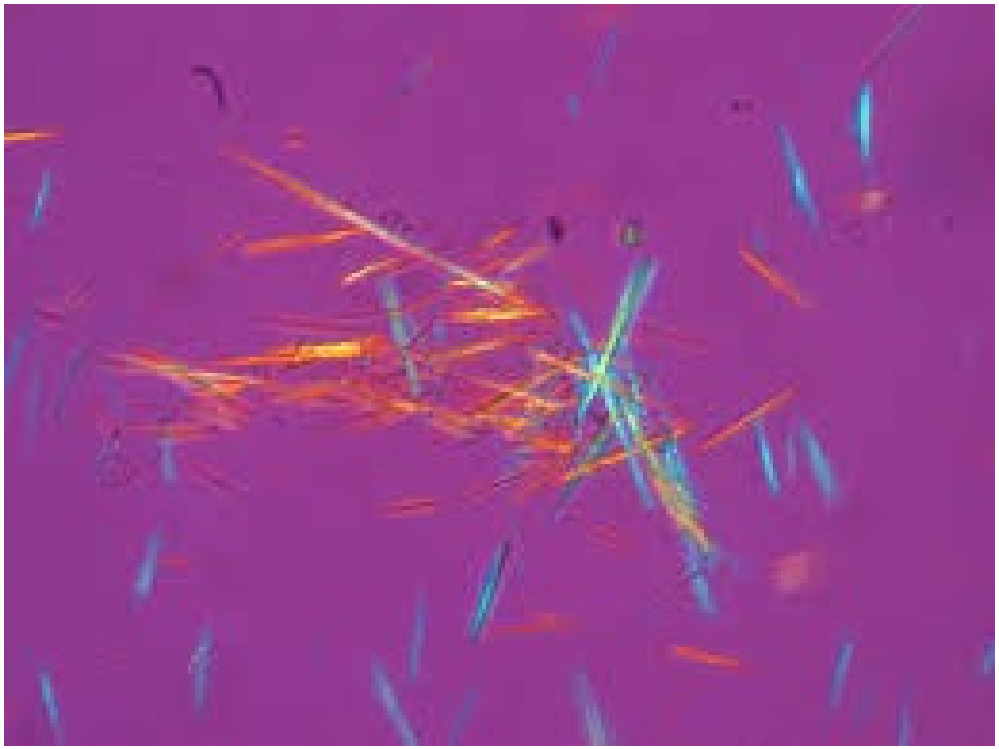
- X-rays are usually normal early in the disease.

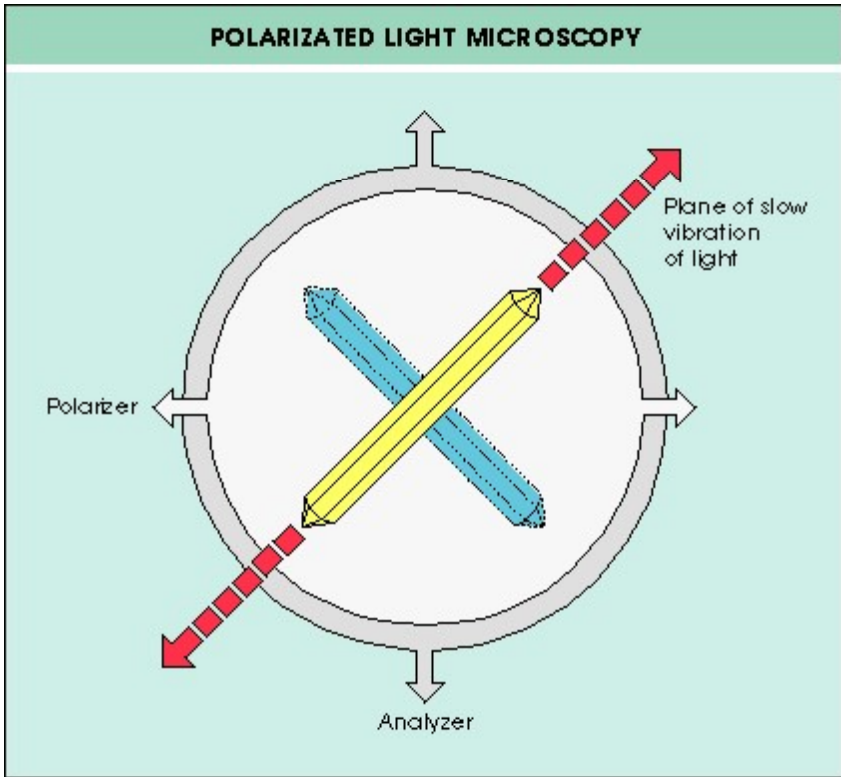
- After a few repetitive acute attacks: punched out erosion, with sharp margins and overhanging edges





Crystal identification





Polarising light microscopy

A contrast-enhancing technique to allow the evaluation of the composition and three-dimensional structure of anisotropic specimens

A regular light microscope uses unpolarised white light. This is the type of light that we see, and its waves vibrate in random directions.

Polarised light has waves that vibrate only in one direction, and cannot be seen by us normally.

Anisotropic substances are “direction-dependent” : they do not behave the same way in all directions.

Polarising light microscopy uses polarizing filters to make use of polarised light, to improve image quality when examining birefringent (doubly-refracting) anisotropic materials.

Crystal identification

Even during the asymptomatic inter-critical period, urate crystals are present in previously affected joints in virtually all untreated gouty patients and in approximately 70 percent of those receiving uric acid-lowering therapy

2015 ACR-EULAR Gout Classification Criteria (1)

Criteria (to be used if Sufficient Criterion not met): *Score ≥8 required for classification as gout*		Categories	Score
CLINICAL	**Pattern of joint/bursa involvement during symptomatic* episode(s) ever**	Joint(s) or bursa(e) other than ankle, midfoot or 1st MTP (or their involvement only as part of a polyarticular presentation)	0
		Ankle OR midfoot (as part of monoarticular or oligoarticular episode without MTP1 involvement)	1
		MTP1 (as part of monoarticular or oligoarticular episode)	2
	Characteristics of symptomatic episode(s) ever: i) Erythema overlying affected joint (patient-reported or physician-observed) ii) can't bear touch or pressure to affected joint **iii)** great difficulty with walking or inability to use affected joint	No characteristics	0
		One characteristic	1
		Two characteristics	2
		Three characteristics	3
	Time-course of episode(s) ever: Presence (ever) of ≥2, irrespective of anti-inflammatory treatment: i) Time to maximal pain <24 hours ii) Resolution of symptoms in ≤14 days **iii)** Complete resolution (to baseline level) between symptomatic episodes	No typical episodes	0
		One typical episode	1
		Recurrent typical episodes	2
	Clinical evidence of tophus: Draining or chalk-like subcutaneous nodule under transparent skin, often with overlying vascularity, located in typical locations: joints, ears, olecranon bursae, finger pads, tendons (e.g., Achilles).	Absent	0
		Present	4

2015 ACR-EULAR Gout Classification Criteria (2)

LAB	**Serum urate:** Measured by uricase method. Ideally should be scored at a time when the patient was not taking urate-lowering treatment and patient was beyond 4 weeks of the start of an episode (i.e., during intercritical period); ***if*** practicable, retest under those conditions. The highest value irrespective of timing should be scored.	<4mg/dL [<0.24mM] †	**-4**
		4-<6mg/dL [0.24-<0.36mM]	0
		6-<8mg/dL [0.36-<0.48mM]	2
		8-<10mg/dL [0.48-<0.60mM]	3
		≥10mg/dL [≥0.60mM]	4
	Synovial fluid analysis of a symptomatic (ever) joint or bursa:** Should be assessed by a trained observer.	Not done	0
		MSU negative	**-2**
IMAGING‡	**Imaging evidence of urate deposition in symptomatic (ever) joint or bursa:** Ultrasound evidence of double-contour sign¶ ***or*** DECT demonstrating urate deposition§.	Absent OR Not done	0
		Present (either modality)	4
	Imaging evidence of gout-related joint damage: Conventional radiography of the hands and/or feet demonstrate at least one erosion.‡‡	Absent OR Not done	0
		Present	4
		TOTAL SCORE	

Maximum score is 23. **Threshold to classify as gout is ≥8.**

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ABBREVIATED VERSION

Criteria		Categories	Score
CLINICAL	Pattern of joint/bursa involvement	Ankle OR midfoot (mono-/oligo-)	1
		MTP1 (mono-/oligo-)	2
	Characteristics of episode(s) ever	One characteristic	1
		Two characteristics	2
		Three characteristics	3
	Time-course of episode(s) ever	One typical episode	1
		Recurrent typical episodes	2
	Clinical evidence of tophus	Present	4
LAB	Serum Urate	<4mg/dL [<0.24mM]	-4
		6-<8mg/dL [0.36-<0.48mM]	2
		8-<10mg/dL [0.48-<0.60mM]	3
		≥10mg/dL [≥0.60mM]	4
	Synovial Fluid examination for MSU crystals	negative	-2
IMAGE	Imaging evidence of urate deposition	Present	4
	Imaging evidence of gout-related joint damage	Present	4
		Maximum Possible Total Score	**23**

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DRUGS MODIFYING RENAL EXCRETION OF URATE

Increased excretion	Decreased excretion
Aspirin (high dose)	Aspirin (low dose)
Phenylbutazone (high dose)	Phenylbutazone (low dose)
Chlorothiazide (intravenous)	Thiazide diuretics
Probenecid	Furosemide
Sulfinpyrazone	Ethacrynic acid
Benzbromarone	Ethambutol
Diflunisal	Pyrazinamide
Azapropazone	Nicotinic acid
Radiographic contrast media	
Oral anticoagulants	
Adrenal steroids	
Glycopyrrolate	
Glyceryl guaiacolate	
Chlorprothixene	

Drugs such as outdated tetracyclines may also cause hypouricemia by inducing a Fanconi-like syndrome.

Case history #1

- A 54-year-old man complains of severe pain and swelling in his right first toe that developed overnight. He is limping because of the pain and states that this is the most severe pain he has ever had ('even covering my foot with the bed sheet hurts'). He has had no previous episodes. His only medication is hydrochlorothiazide for hypertension. He drinks 2 to 3 beers a day.

- On examination, he is obese. There is swelling, erythema, warmth, and tenderness of the right first toe. There is also tenderness and warmth with mild swelling over the mid foo

Case history #2

- An 85-year-old man presents with several days of swelling and severe pain in both hands limiting his ability to use his walking frame. He has a history of gout but has not experienced these symptoms before.

- On examination, he has a temperature of 37.8°C (100.1°F). There is diffuse warmth, mild erythema, and pitting edema over the dorsum of both hands. There is tenderness and limited hand grip bilaterally. There are multiple nodules around several of the proximal interphalangeal and distal interphalangeal joints, and effusion and tenderness in his left olecranon bursa with palpable nodules.

Treatment of gout

TREATMENT OF ACUTE ATTACK:

NSAIDs, colchicine and steroids (intra-articular or systemic)

The first line of therapy is to rest the affected joint and to use full doses of a non-steroidal anti-inflammatory drug (NSAID):

indomethacin 50mg 8 hrly (or maximum 50mg

6 hrly); naproxen 500mg 12 hrly; diclofenac 50mg 8 hrly, provided the patient's renal function is normal.

Colchicine can be a useful adjunct to NSAIDs if the acute attack does not settle rapidly.

0.5 mg twice or three times a day

Treatment of gout

- Intra-articular : if NSAID use is contra-indicated and one or at most two joints are inflamed.
- Systemic steroids for polyarticluar disease or if NSAIDs or colchicine contraindicated(CKD, PUD, CHF)
- Allopurinol or uricosuric drugs should not be commenced during an acute attack of gout.

Non-pharmacological treatment

Avoid diuretic therapy; weight gain; alcohol consumption; Aspirin therapy at low doses

Specific Recommendations:
GENERAL HEALTH, DIET, AND LIFESTYLE MEASURES FOR GOUT PATIENTS#:

Evidence Grades for Recommendations:
Level A: Supported by multiple (ie, more than one) randomized clinical trials or meta-analyses
Level B: Derived from a single randomized trial, or nonrandomized studies.
Level C: Consensus opinion of experts, case studies, or standard-of-care.

- **Weight loss for obese patients, to achieve BMI that promotes general health**
- **Healthy overall diet ^**
- **Exercise (Achieve physical fitness)**
- **Smoking cessation**
- **Stay well hydrated**

C

Avoid	Limit	Encourage >
• Organ meats high in purine content (eg, sweetbreads, liver, kidney) B	Serving Sizes of: • Beef, Lamb, Pork • Seafood with high purine content (eg, sardines, shellfish) B	• Low-fat or non-fat dairy products B
• High fructose corn syrup-sweetened sodas, other beverages, or foods C	• Servings of naturally sweet fruit juices • Table sugar, and sweetened beverages and desserts • Table salt, including in sauces and gravies C	• Vegetables C
• Alcohol overuse (defined as more than 2 servings per day for a male and 1 serving per day for a female) in all gout patients B • Any alcohol use in gout during periods of frequent gout attacks, or advanced gout under poor control C	• Alcohol (particularly beer, but also wine and spirits) in all gout patients B	

^Without a specific task force panel (TFP) vote, adherence to diets for cardiac health and control of co-morbidities such as obesity, metabolic syndrome, diabetes, hyperlipidemia, and hypertension was stressed for gout patients, as appropriate.

> The TFP recommendation to "encourage" intake was not intended to advocate excesses in consumption of specific dietary items. There was a lack of TFP voting consensus on: Cherries and Cherry Products, Ascorbate (In Supplements or Foods), Nuts, Legumes. The TFP did not specifically vote on the question of limits on consumption of purine-rich vegetables and legumes.

Case history #1

- A 60-year-old woman presents complaining of bilateral knee pain on most days of the past few months. The pain was gradual in onset. The pain is over the anterior aspect of the knee and gets worse with walking and going up and down stairs. She complains of stiffness in the morning that lasts for a few minutes and a buckling sensation at times in the right knee.
- On exam, there is a small effusion, diffuse crepitus, and limited flexion of both knees. Joint tenderness is more prominent over the medial joint line bilaterally. She has a steady but slow gait, slightly favoring the right side.

Case history #2

- A 55-year-old woman has been complaining of pain and swelling in several fingers of both hands for the past 2 months. She describes morning stiffness lasting 30 minutes. Her mother tells her that she had a similar condition at the same age.
- She denies any other joint pain or swelling.
- On exam, she has tenderness, slight erythema, and swelling in one PIP joint and two DIP joints in each hand. She has squaring at the base of her right thumb (the first carpometacarpal joint). There is no swelling or tenderness in her MCP joints.

Osteoarthritis (OA)

A degenerative joint disease, occurring primarily in older people and characterized by erosion of the articular cartilage, hypertrophy of bone at the Margins,subchondral sclerosis, and a range of biochemical and morphologic alterations of the synovial membrane and joint capsule

Osteoarthritis (OA)

- The commonest form of arthritis
- Leading cause of disability in the elderly
- OA is uncommon in adults under age 40 and highly prevalent in those over age 60
- Much more common in women than in men
- OA ranges from an asymptomatic, incidental finding on clinical or radiologic examination to a progressive disabling disorder eventually culminating in "joint failure.

Osteoarthritis (OA)

- Most frequently affected are the spine, knees, hips, interphalangeal joints of the hands, and first metatarsophalangeal (MTP) joints

- Uncommon in the elbows, glenohumeral joints, ankles, and wrists

Primary Vs Secondary OA

- **Primary OA** : the most common type , has no identifiable etiology or predisposing cause.
- **Secondary OA**: has an identifiable underlying cause

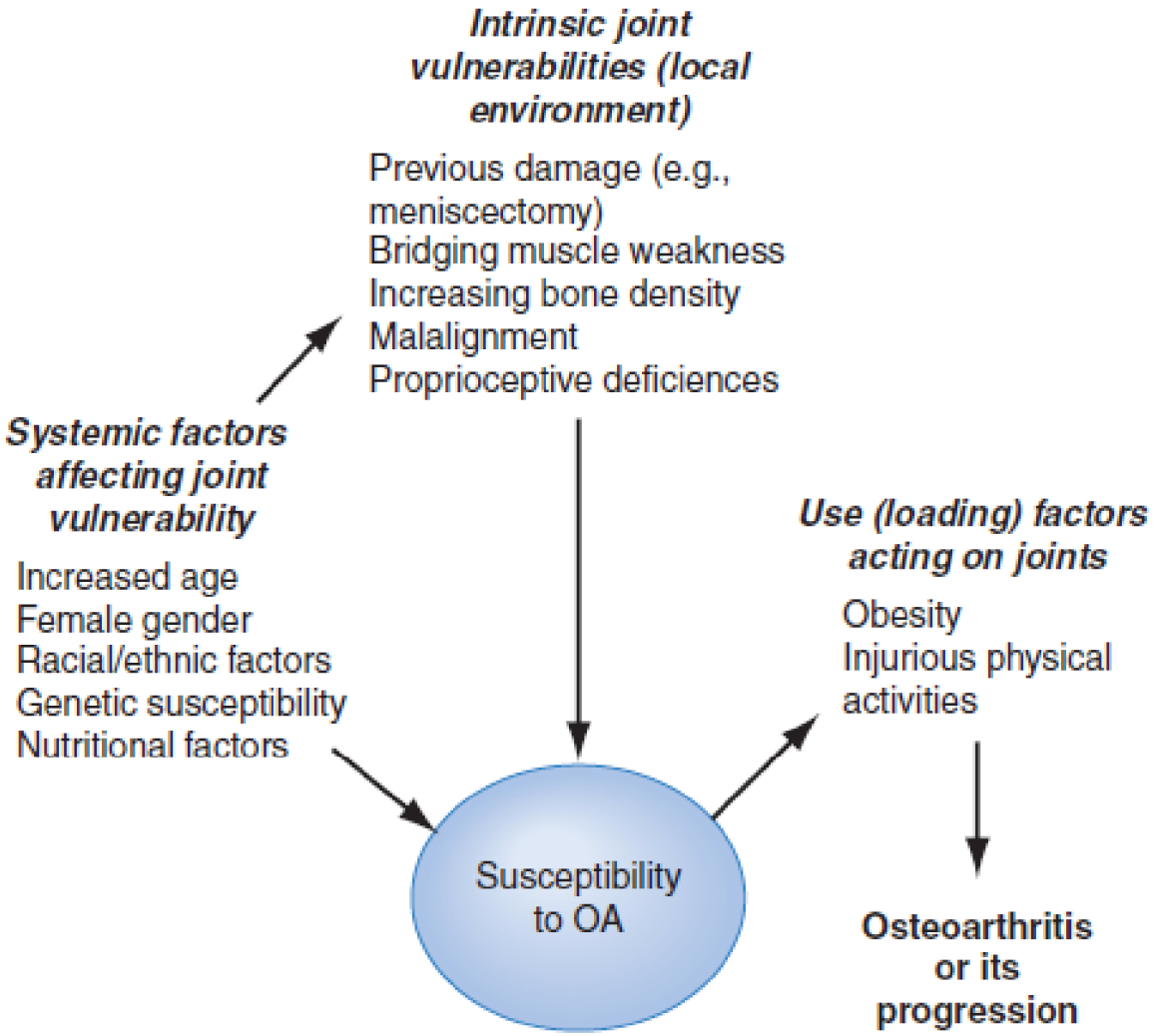
✓ The most common causes of secondary OA are metabolic conditions OR anatomic factors

✓ Pathologically indistinguishable from primary OA.

TABLE 99-3 Causes of Secondary Osteoarthritis

Metabolic
Crystal-associated arthritis
Calcium pyrophosphate or apatite deposition
Acromegaly
Ochronosis
Hemochromatosis
Wilson's disease
Hyperparathyroidism
Ehlers-Danlos syndrome
Gaucher's disease
Diabetes
Mechanical/Local Factors
Slipped capital femoral epiphysis
Epiphyseal dysplasias
Legg-Calvé-Perthes disease
Congenital dislocation
Femoroacetabular impingement
Congenital hip dysplasia
Limb-length inequality
Hypermobility syndromes
Avascular necrosis/osteonecrosis
Traumatic
Joint trauma (e.g., ACL tear)
Fracture through joint
Prior joint surgery (e.g., meniscectomy, ACL)
Charcot joint (neuropathic arthropathy)
Inflammatory
Rheumatoid arthritis or other inflammatory arthropathies
Crystalline arthropathy (gout)
History of septic arthritis

Risk factors for OA



Pathogenesis of OA

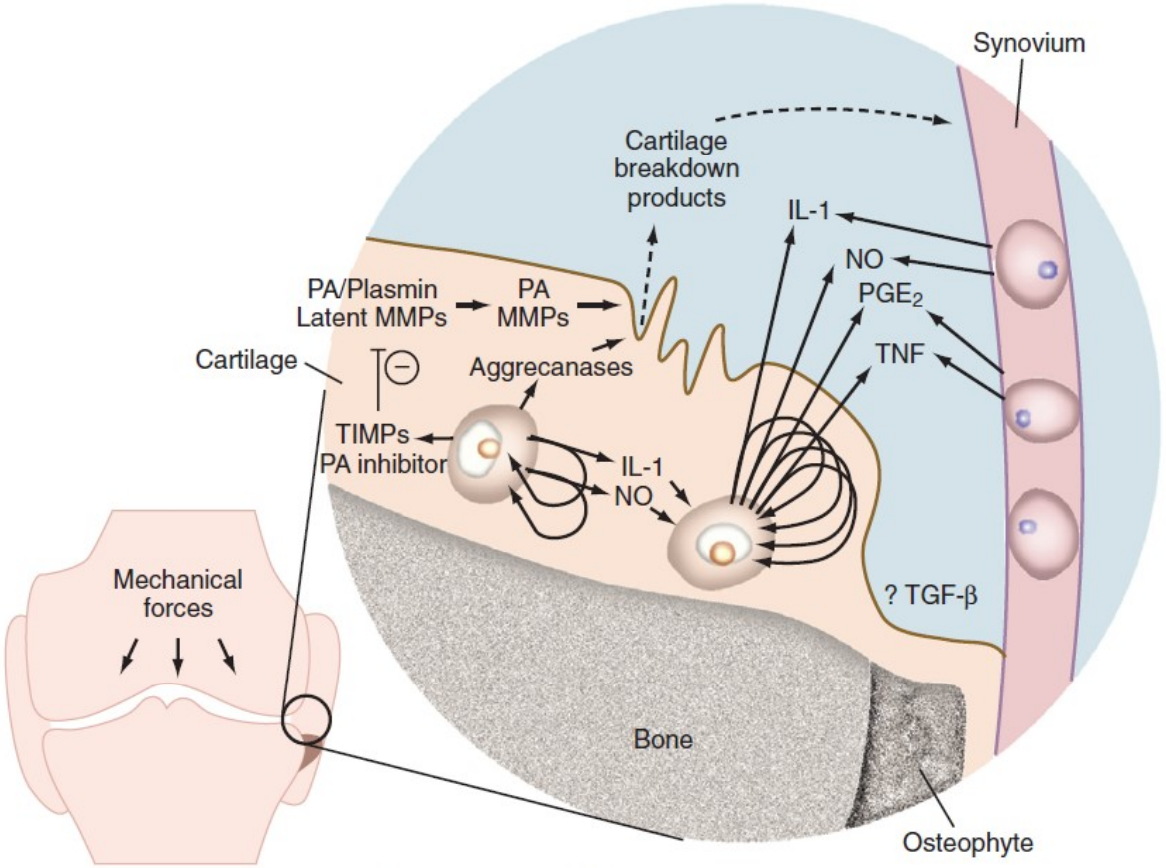


Figure 98-2 Schematic of pathogenic mechanisms of osteoarthritis. Mechanical stress initiates altered metabolism characterized by the release of matrix metalloproteinases (MMPs), pro-inflammatory cytokines, and mediators such as nitric oxide (NO) and prostaglandin E_2 (PGE_2). Cartilage breakdown products play a role by stimulating the release of cytokines from synovial lining cells and by inducing MMP production by chondrocytes. Perpetuation of cartilage damage is amplified by the autocrine and paracrine actions of interleukin (IL)-1β and tumor necrosis factor (TNF) produced by chondrocytes. PA, Plasminogen activator; TGF-β, transforming growth factor-β; TIMPs, tissue inhibitors of metalloproteinases.

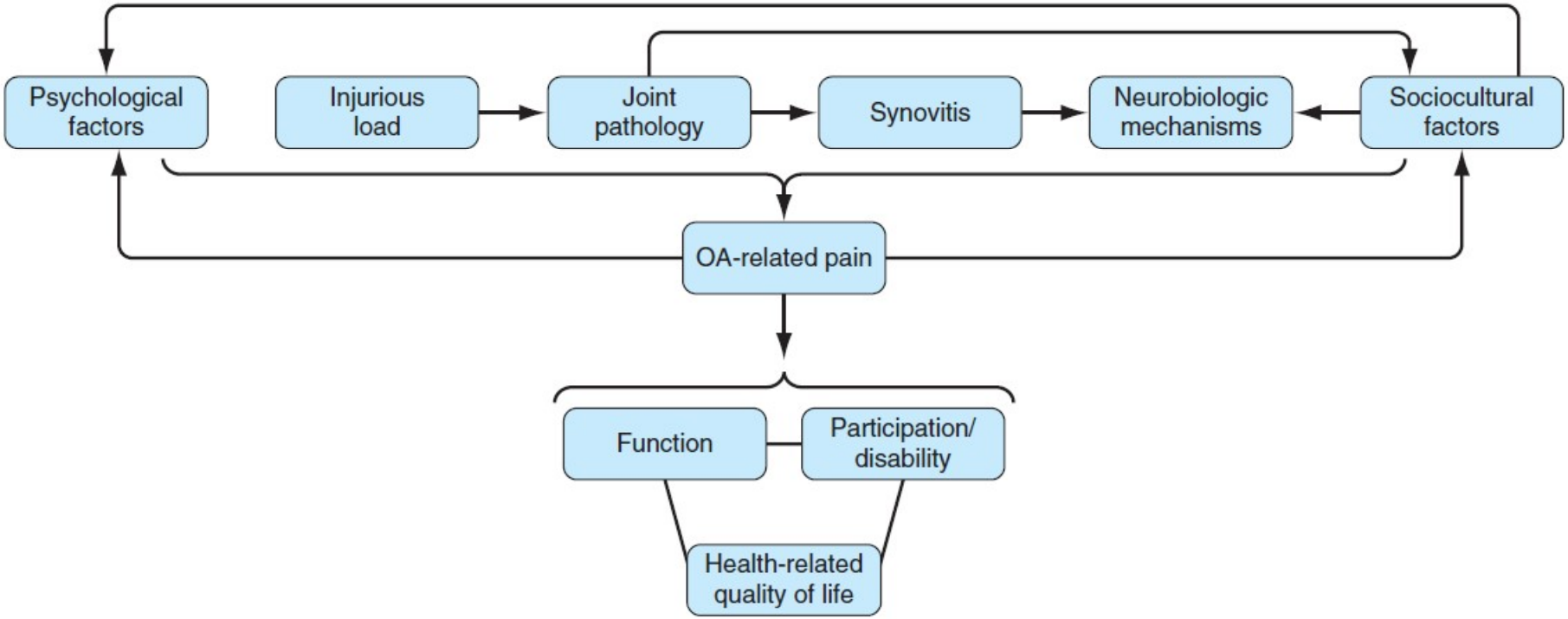


Figure 100-1 Pathophysiology of osteoarthritis (OA)-related pain and its consequences. *(Modified from Neogi T: The epidemiology and impact of pain in osteoarthritis.* Osteoarthritis Cartilage *21[9]:1145-1153, 2013.)*

Approach to the diagnosis of OA
History

- Osteoarthritis is primarily a clinical diagnosis
- More common in people >50 years and in women.
- M/C joints : asymmetric ;hands, knees, hips, and spine
- Hand OA is more common in women with a FHx of hand OA.
- The most common symptom is joint pain : worse with activity and weight-bearing, especially following a period of rest(gelling phenomenon) . +- Joint swelling, joint locking or joint instability.

Approach to the diagnosis of OA

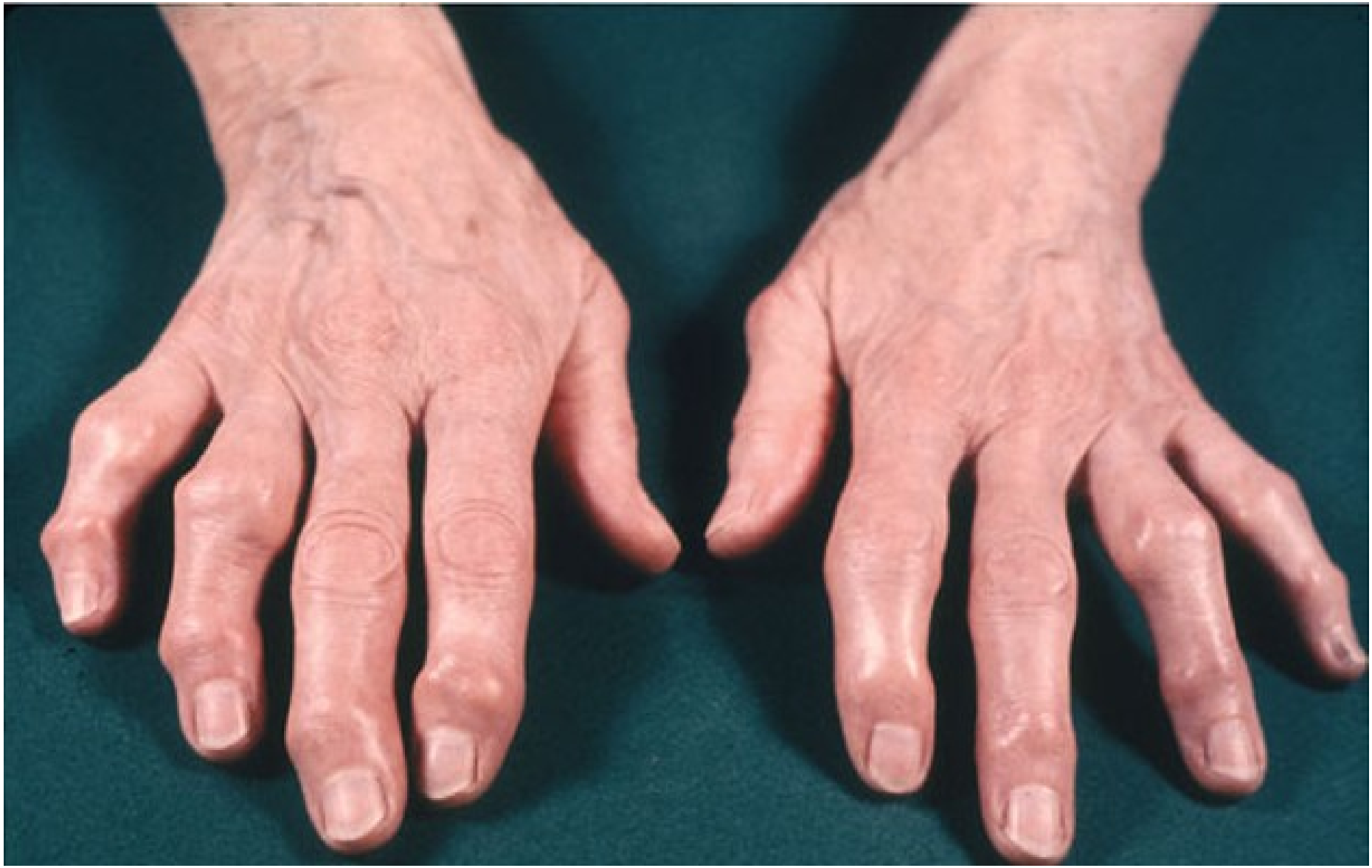
- Morning stiffness, less than 30 minutes
- Night symptoms only in advanced OA
- Loss of function resulting in limitation of daily activities

Approach to the diagnosis of OA
Physical examination

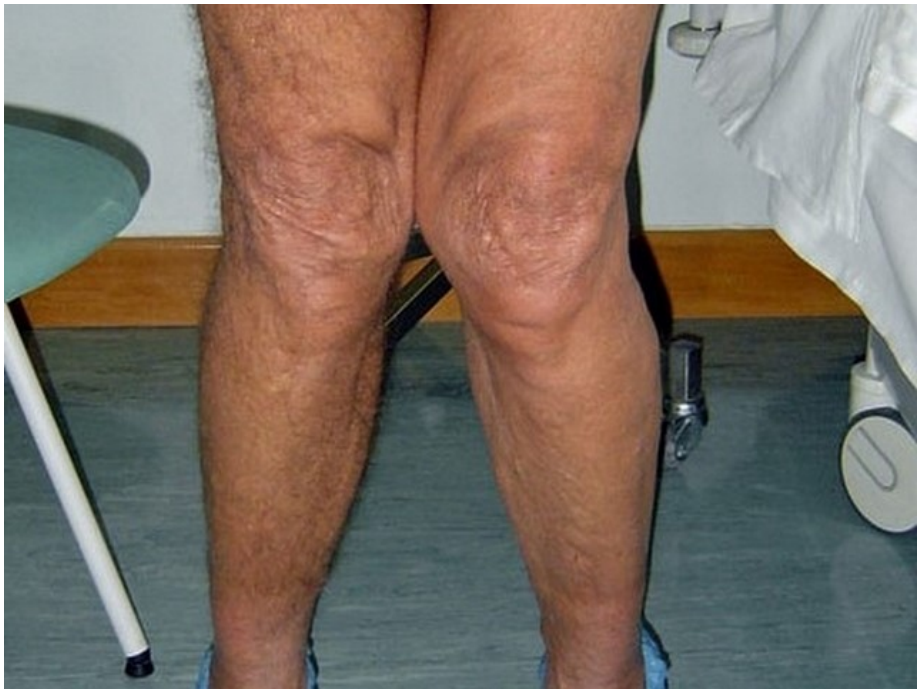
- Weight and BMI are important, as knee OA and, to a lesser degree, hip OA are common in overweight patients.
- Pain on range of motion and limitation of range of motion are common to all forms of osteoarthritis

- Swelling may be observed, with bony deformities and malalignment of the affected joint.

- Bony deformities are particularly common in the hands and lead to enlargement of the PIP joints (Bouchard nodes) and DIP joints (Heberden nodes), as well as squaring at the base of the thumb (the first carpometacarpal joint).



- knee OA: new bone formation with bony enlargement
- Palpate crepitus during the range of motion of the joint.
- Limited range of motion, small effusions, and joint line tenderness may be elicited.
- An abnormal gait can be observed.



Approach to the diagnosis of OA Imaging

- OA is essentially a clinical diagnosis. Plain radiographs may be done in the initial workup to help confirm the diagnosis in moderate to advanced OA, but they are not sensitive in detecting early disease. They may also help rule out less common etiologies for pain, such as a bone tumor, pigmented villonodular synovitis, or avascular necrosis, when suspicion exists.
- They are poorly correlated with the symptoms
- Computed tomography or magnetic resonance imaging, are rarely needed

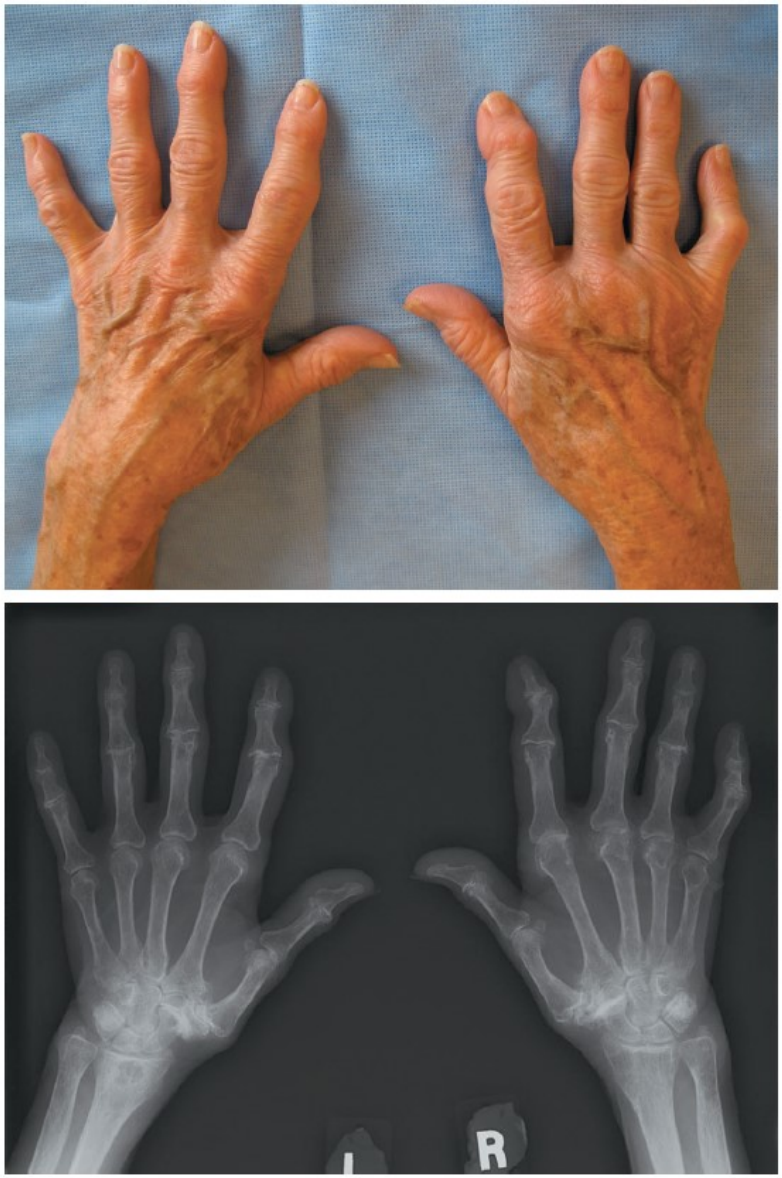


Figure 99-2 The hands of a 79-year-old woman showing clinical *(top)* and radiographic *(bottom)* features of osteoarthritis (OA). This patient has Heberden's and Bouchard's nodes of multiple digits; radiographs show osteophytes, joint space narrowing, and cysts typical of OA, as well as "gull-wing" deformities at the third proximal interphalangeal joints suggestive of erosive OA.

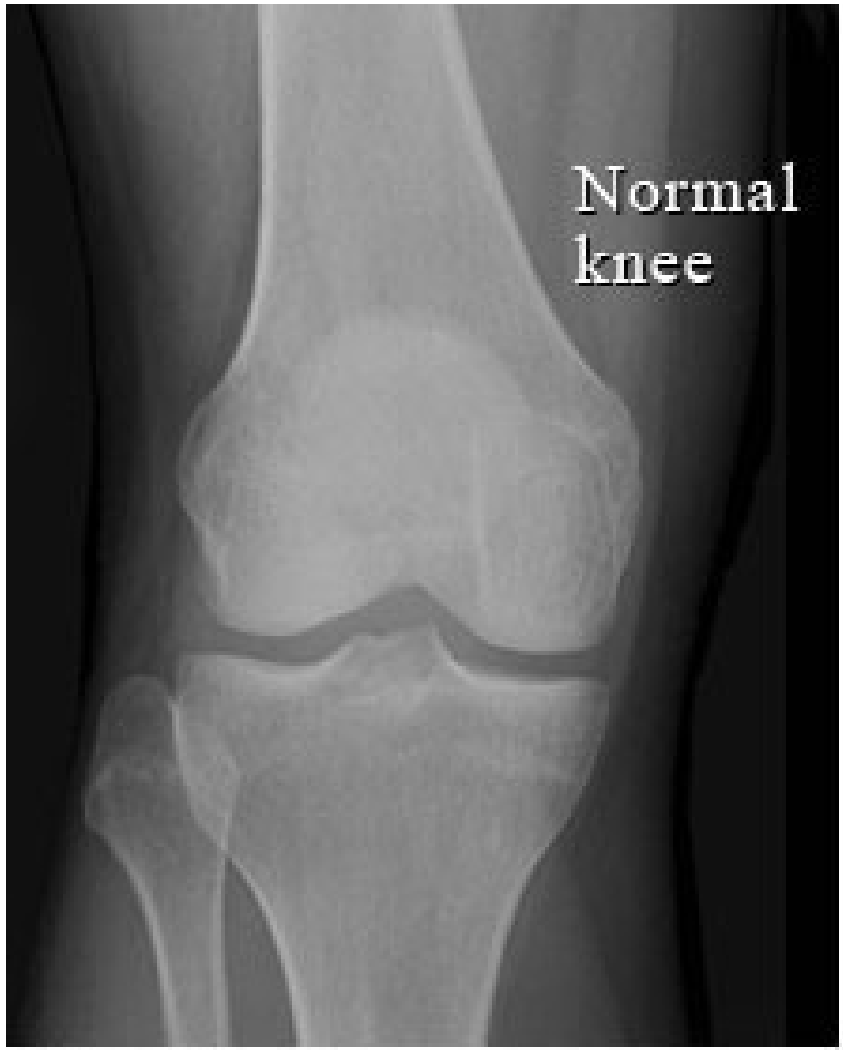


Figure 1

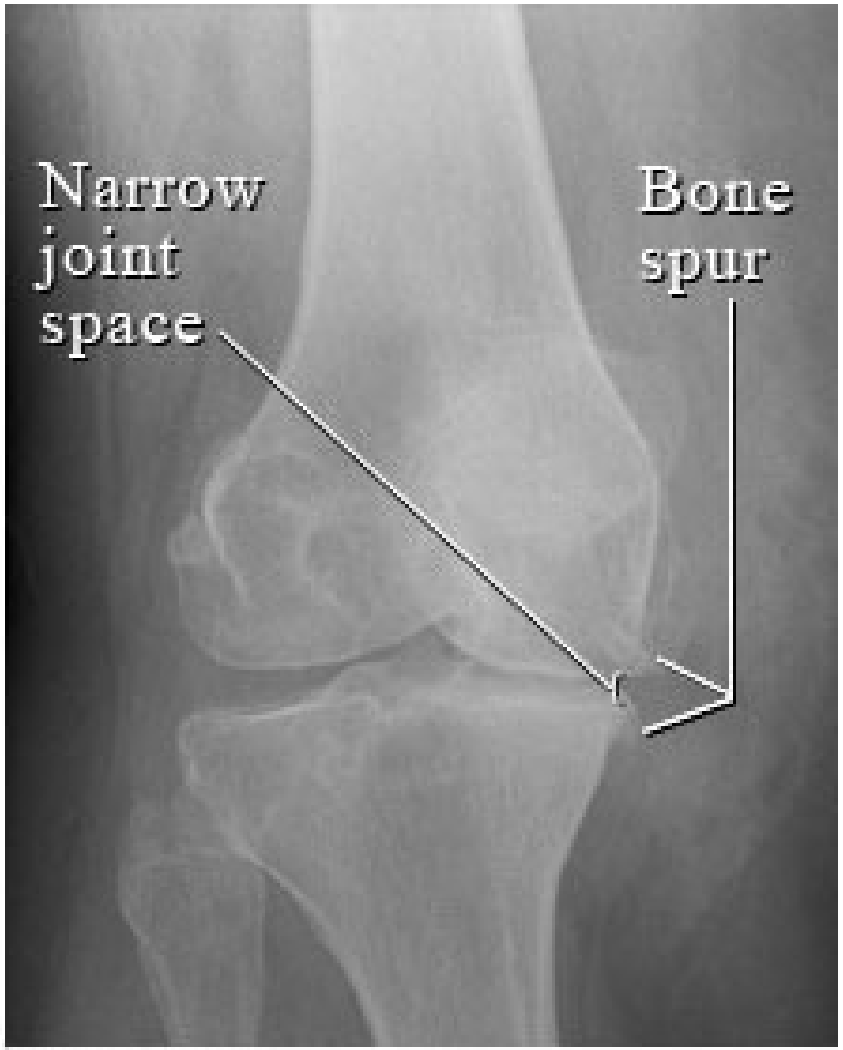
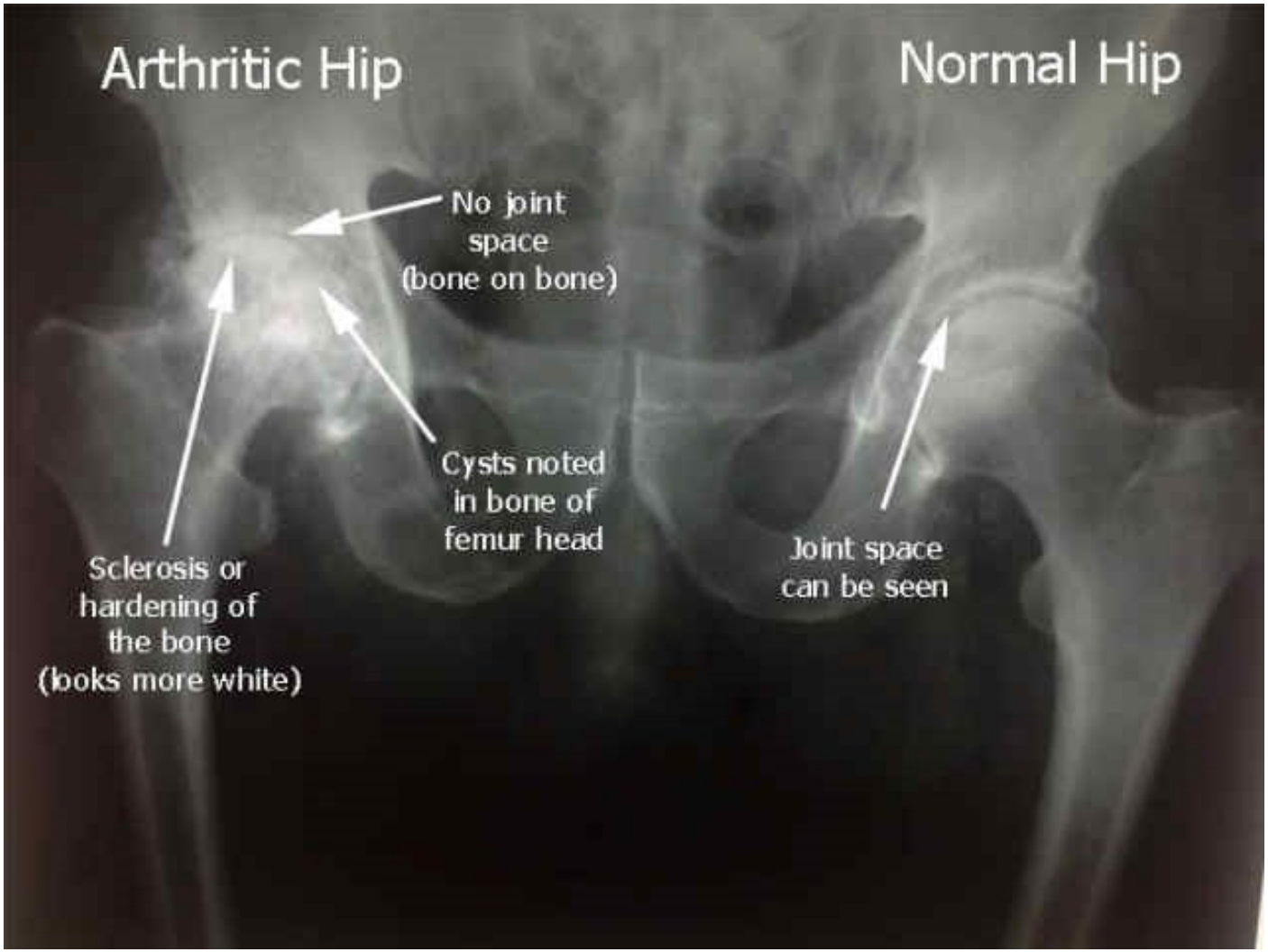


Figure 2


Femur
osteophyte
sclerosis
reduced
joint
space
Tibia


Arthritic Hip
Normal Hip
No joint space (bone on bone)
Cysts noted in bone of femur head
Sclerosis or hardening of the bone (looks more white)
Joint space can be seen

Approach to the diagnosis of OA
Labs

- Laboratory testing usually is not required to make the diagnosis

- Inflammatory markers (CRP, ESR) should be ordered in the initial workup if inflammatory arthritis, such as rheumatoid arthritis, is a differential diagnosis. These tests are normal in OA.

TABLE 99-1 American College of Rheumatology Radiologic and Clinical Criteria for Osteoarthritis

Hand[5]

1. Hand pain, aching, or stiffness on most days of prior month
2. Hard tissue enlargement of ≥2 of 10 selected joints*
3. Fewer than 3 swollen MCP joints
4. Hard tissue enlargement of ≥2 DIP joints
5. Deformity of ≥2 of 10 selected joints*

Diagnosis requires items 1-3 and either 4 or 5

Knee: Clinical[6]

1. Knee pain for most days of prior month
2. Crepitus with active joint motion
3. Morning stiffness lasting ≤30 min
4. Bony enlargement of the knee on examination
5. Age ≥38 yr

Diagnosis requires 1 + 2 + 4, or 1 + 2 + 3 + 5, or 1 + 4 + 5

Knee: Clinical and Radiographic[6]

1. Knee pain for most days of prior month
2. Osteophytes at joint margins
3. Synovial fluid typical of osteoarthritis
4. Age ≥40 yr
5. Morning stiffness lasting ≤30 min
6. Crepitus with active joint motion

Diagnosis requires 1 + 2, or 1 + 3 + 5 + 6, or 1 + 4 + 5 + 6

Hip: Clinical and Radiographic[4]

1. Hip pain for most days of the prior month
2. ESR ≤20 mm/hr
3. Radiographic femoral and/or acetabular osteophytes
4. Radiographic hip joint space narrowing

Diagnosis requires 1 + 2 + 3, or 1 + 2 + 4, or 1 + 3 + 4

Treatment of Osteoarthritis

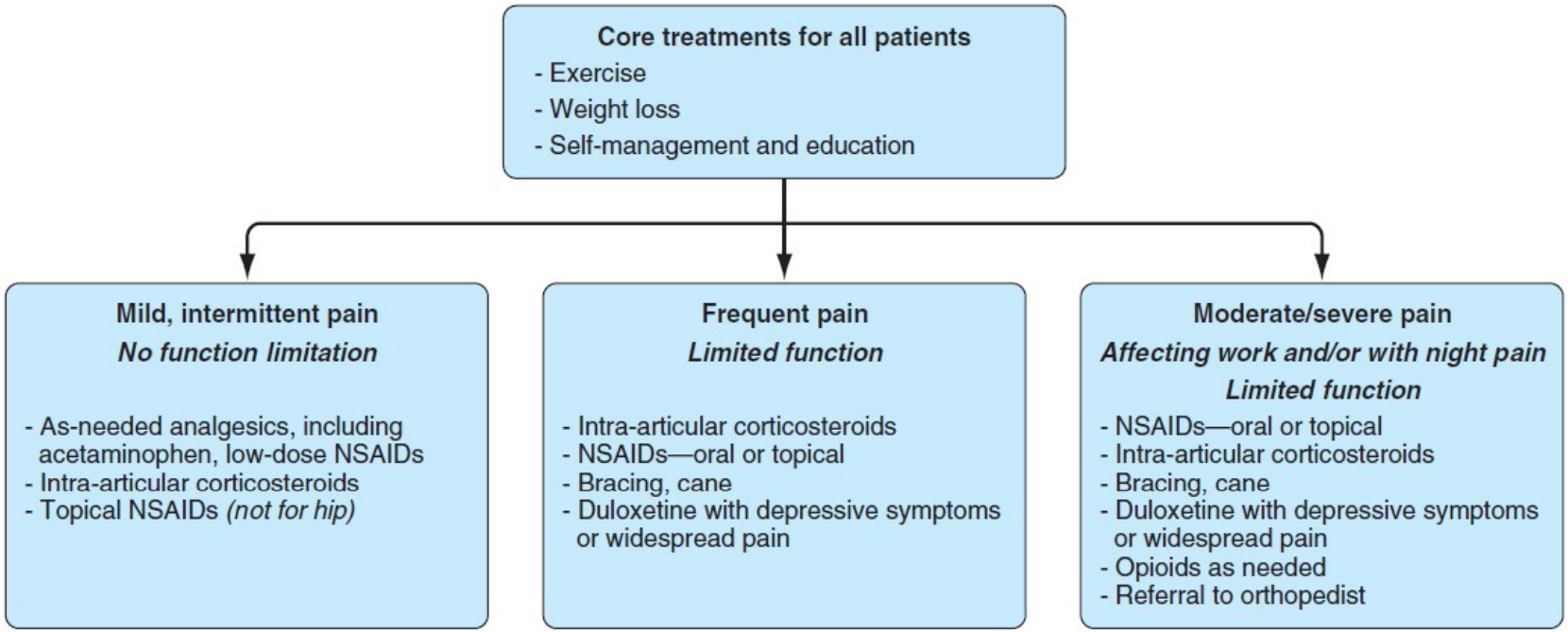


Figure 100-2 Algorithm for management of knee and hip osteoarthritis.

THE END