GOUT

And Other Crystal Arthropathies

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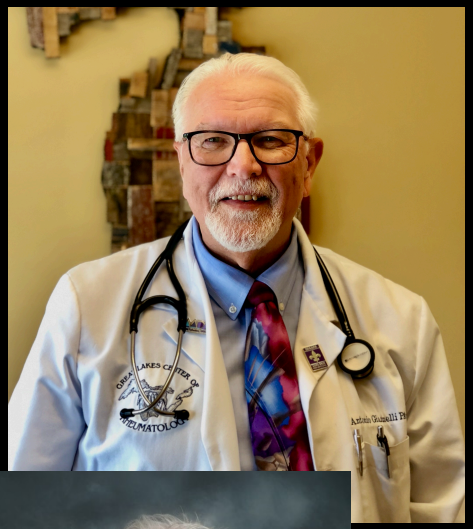

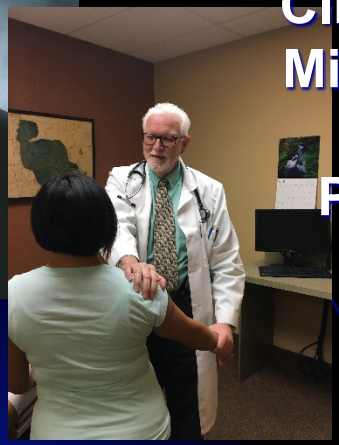

SPAR
Society of Physician Assistants in Rheumatology

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I have NO relevant commercial relationships to disclose

To My Friends:

Christina Starks, PA-C
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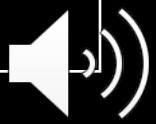
Ben Smith, PA-C, DFAAPA

for their slides and support

Discussion Outline

- Monoarthopathies
- Define Gout / Prevalence
- Differential Dx
- Xray/Lab
- Synovial Fluid Analysis
- Treatment
- Prevention
- Pseudogout (CPPD)

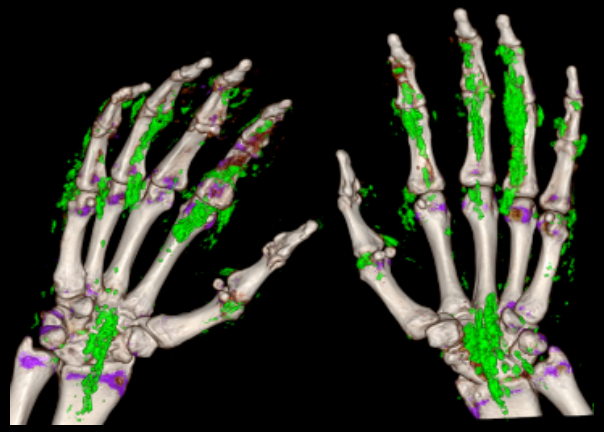


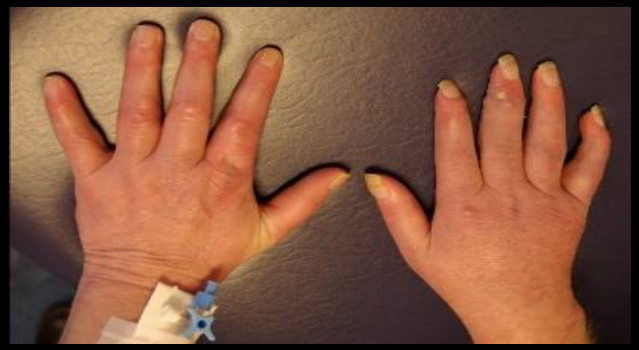
Don’t Miss the PANCE/PANRE Review Slides at the end !

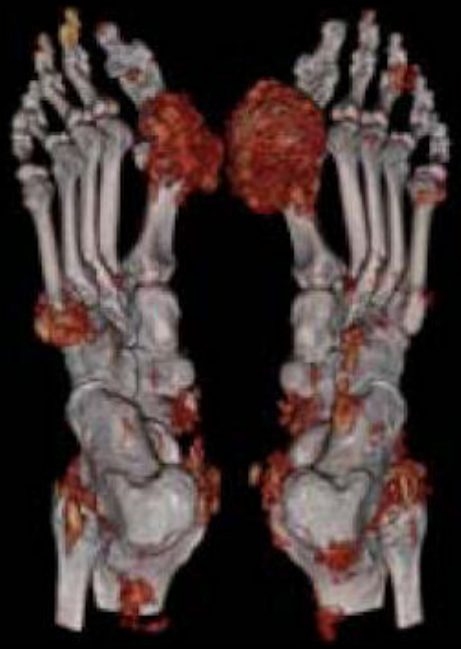
Learning Objectives

1. Recognize Podagra and other types of monoarthropathies
2. Develop a differential diagnosis
3. Understand the value of joint fluid analysis
4. Understand the treatment paradigm for hyperuricemia
5. Be familiar with current gout treatments
6. Discuss prevention with your patients

Urate Burden Extends Beyond Clinically Apparent Tophi







Images courtesy of Dr. Jürgen Rech. Individual patient presentations may vary

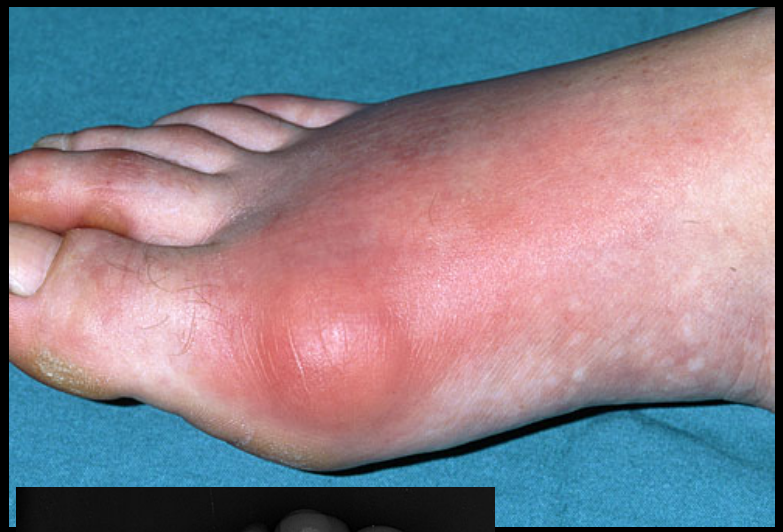
In addition to visible tophi, MSU crystals can accumulate anywhere in the body[1, 2]

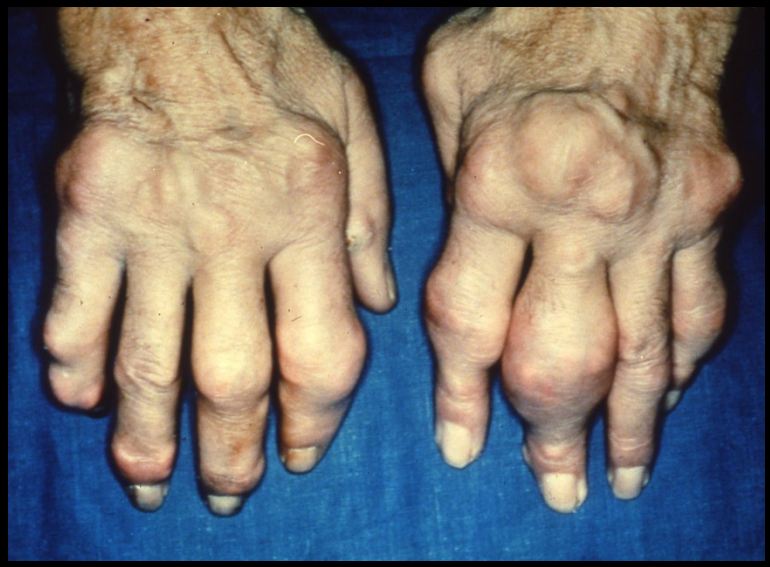
- **Most commonly in the joints, leading to chronic arthropathy**
- **In pressure points (e.g., *ulnar aspect of forearm), bursae, tendons, kidney***
- ***In o*ther connective tissue such as heart valves and spinal column (rarely)**

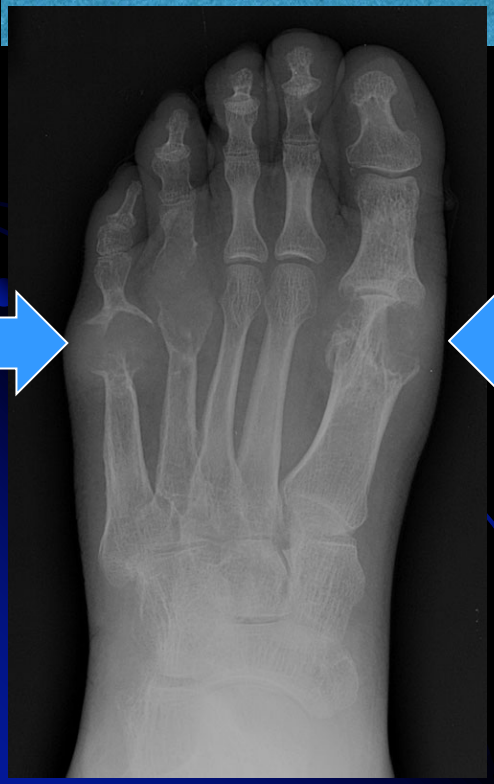
With inadequate treatment, the total body burden of urate can continue to increase[2]

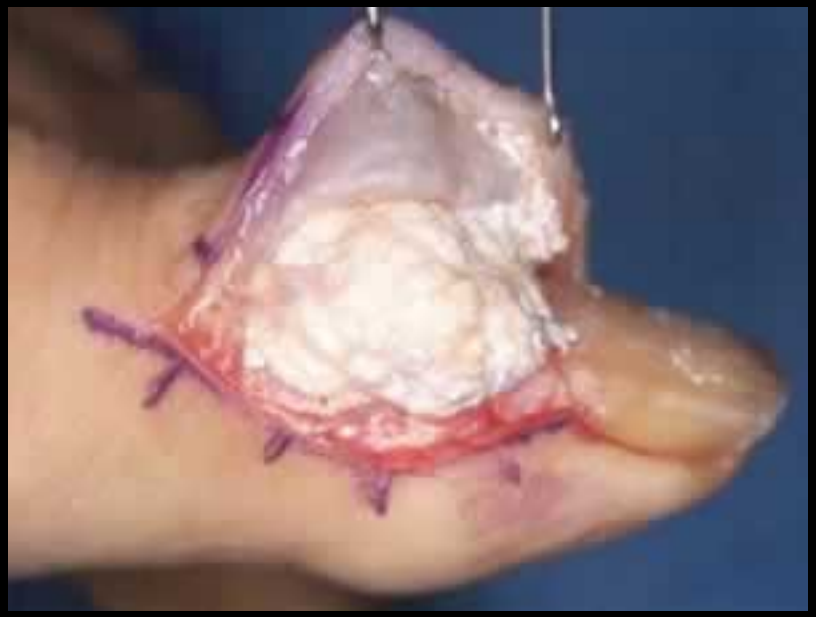
1. Edwards NL, Crystal-Induced Joint Disease in ACP Medicine Textbook, 2012
2. Yu TF, Gutman AB. Am J Med Sci; 1967;254:893–907

Chronic Tophaceous Gout









Acute Monoarthritis

Noninflammatory

- Trauma
- Hemarthrosis (Fracture)
- Sickle-cell disease
- Mechanical Derangement (Avascular necrosis)

Inflammatory

- Septic arthritis
- **Gout**
- **Pseudogout**
- Viral Arthritis (EBV, Parvo B19, Hep B/C, Rubella, HIV)
- Reiter's Syndrome
- Lyme disease
- Acute rheumatic fever
- Hemarthrosis (Fracture)
- Palindromic (Wax and Wane) rheumatism

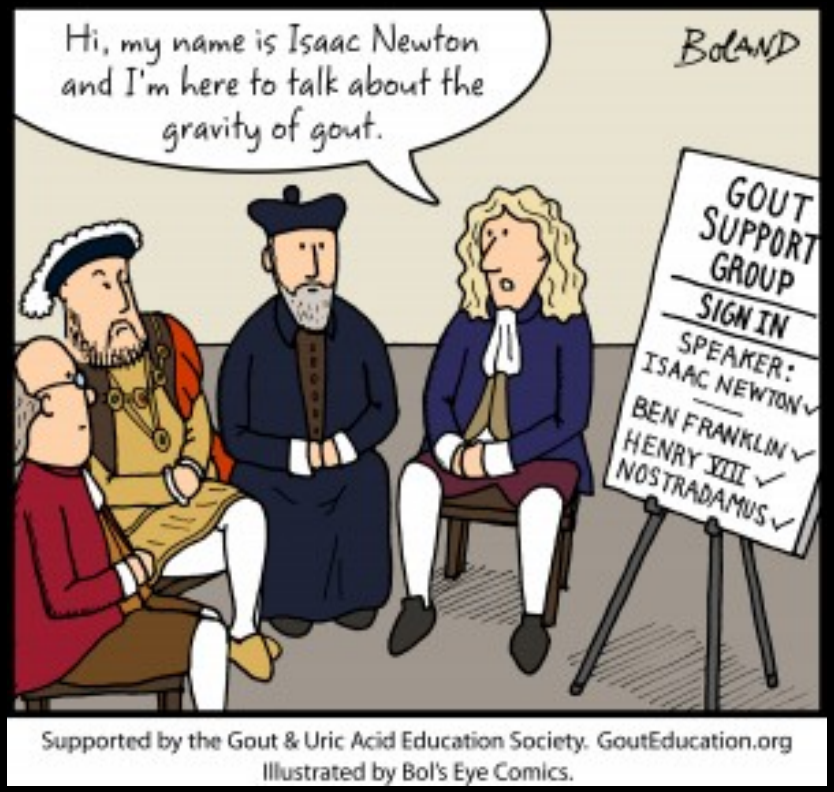
Chronic Monoarthritis

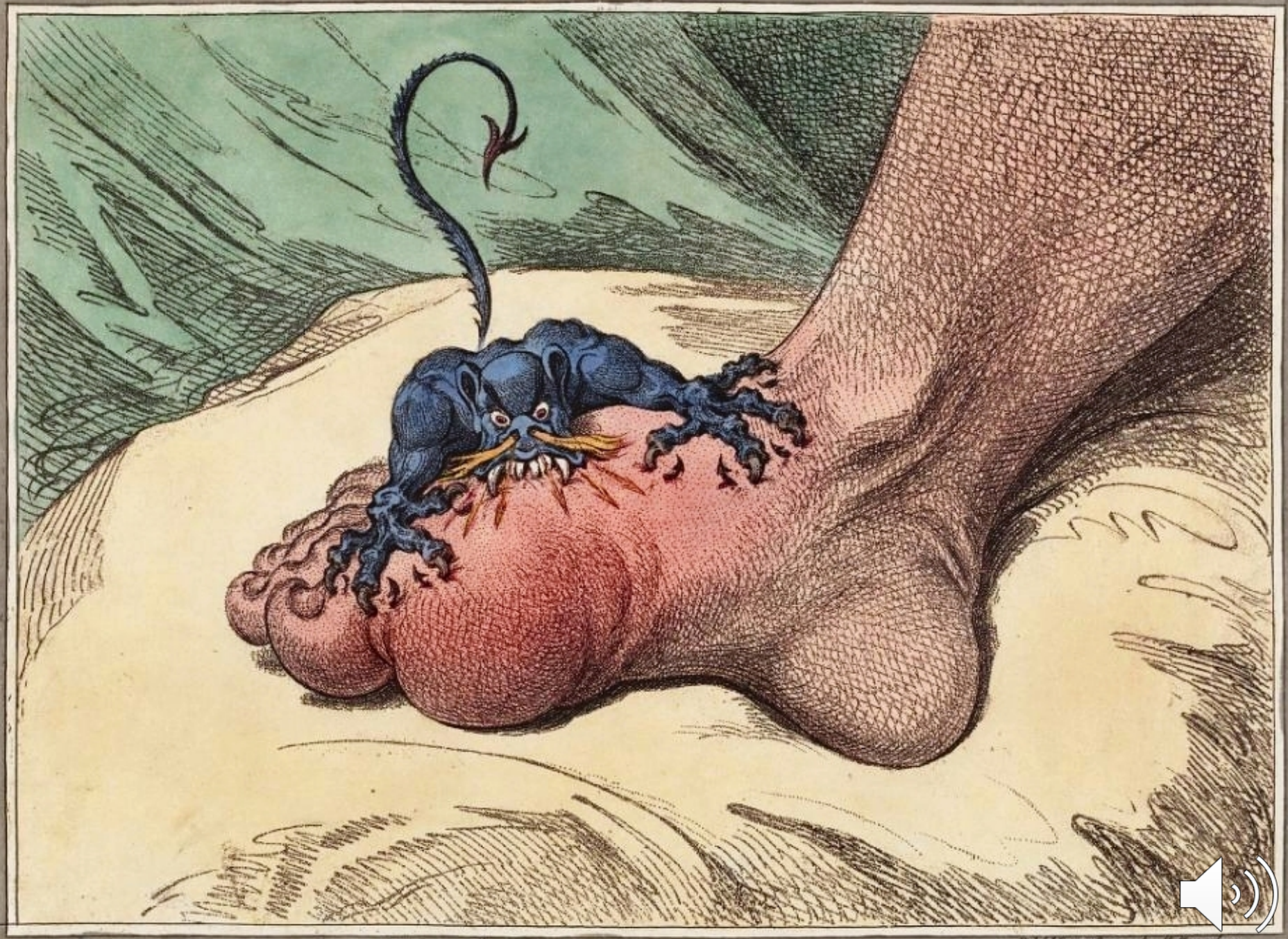
Noninflammatory	Inflammatory
• Hemarthrosis • Pigmented villonodular synovitis (benign tumor) • Neuropathic arthropathy • Osteoarthritis • Osteonecrosis • Foreign body synovitis	• Tuberculosis arthritis • Fungal arthritis • Spondyloarthropathy: Psoriatic arthritis Ankylosing Spondylitis Reactive • **Pseudogout** • Sarcoidosis • Juvenile arthritis

Famous People

Here is a list of famous people – both today and throughout history – who have dealt with and endured the pain of gout

- Ansel Adams, landscape photographer
- Ludwig Van Beethoven, musician
- **Jim Belushi, comedian and actor**
- Maurice Cheeks, basketball player and coach
- Dick Cheney, former Vice President
- **Charles Dickens, English author and social critic**
- Benjamin Franklin, founding father
- King Henry VIII of England
- Samuel Johnson, British author and poet
- Harry Kewell, professional soccer player
- Jared Leto, actor
- Don Nelson, basketball player and coach
- Sir Isaac Newton, English mathematician and physicist
- **Nostradamus, French apothecary**
- Sir Laurence Olivier, actor
- **Luciano Pavarotti, Italian operatic tenor**
- Leonardo da Vinci, Italian painter, sculptor, architect and engineer
- **David Wells, professional baseball player**





The GOUT.

Pub.d May 4.th 1799. by H. Humphrey
27 St.James's Street.

From the *Gout and Uric Acid Education Society*

Stages of Gout

*Only 20% of population with hyperuricemia will progress to acute gout

Stage 1 – Asymptomatic Hyperuricemia

- No symptoms of gout
- Uric acid levels are above 6.0 mg/dL.
- Treatment necessary. May have elevated uric acid levels for years before their first attack
- May need regular monitoring of uric acid levels and advise healthy diet and lifestyle adjustments
- Not everyone with high uric acid gets gout, but the higher the uric acid, the more likely it is

Stage 2 – Acute Gout Attack

- Crystal arthropathy causes episodes of intense pain and swelling in the joint, subside, even without treatment, within three to 10 days
- Another gout attack may not occur for months or years, but chances are good that more will come
- Regular monitoring of uric acid levels and ongoing treatment is important

Stage 3 – Intercritical Gout

- The time between gout flares. It is a symptom-free time, when their joints are functioning normally. However, even when symptoms are absent, ongoing deposits of uric acid crystals continue to accumulate, silently.
- Additional and more painful attacks of gout are likely to continue unless the uric acid is lowered to below 6.0 mg/dL.

Stage 4 – Chronic Tophaceous Gout

- Late stage of gout, now a chronic arthritis, often results in deformity and destruction of bone and cartilage
- Ongoing, destructive inflammatory process is active, kidney damage is also possible. With proper medical attention and treatment, most gout patients will not progress to this advanced, disabling stage

* From American College of Rheumatology Guidelines 2020

Risk Factors

The more risk factors a person has, the greater the risk for developing gout

Hyperuricemia – High levels of uric acid, above 6.8 mg/dL, can lead to gout attacks. The best range for uric acid is below 6.0 mg/dL regardless of age or gender

Family History – One in four people with gout has a family history of the disease

Age – Gout can occur in anyone at any age, but it typically develops in people age 45 and older

Gender – Gout affects men more than women, although once women are post-menopausal, their rates of gout increase almost (but not quite) to the same level as men

Obesity – Someone with a Body Mass Index (BMI) of 30 or higher is considered obese

Other Health Issues – Gout is associated with high blood pressure, heart disease, diabetes and kidney disease. It is important to receive a prompt diagnosis and ongoing treatment to manage these conditions

Joint Injury – People with previously damaged joints are more likely to get gout

A High-Fructose Diet – High-fructose corn syrup is added to many foods and drinks. It causes uric acid to go up. Sweetened soft drinks and juices; certain cereals and pastries; ice cream and candy; and processed foods at fast food restaurants often contain high-fructose corn syrup

Use of Certain Medications – This especially includes diuretics or water pills and certain anti-rejection medications used in transplant patients

From the *Gout and Uric Acid Education Society*

Common Triggers

Alcohol – This includes excessive intake of alcohol or binge drinking. This includes > 2 plus drinks (beer, spirits, wine)

Purine-Rich Foods – Eating large amounts of foods high in purines, including red meat, organ meat and shellfish, can trigger attacks

Dehydration, Crash Diets – This especially includes high-protein fad diets

Starting Uric-Acid Lowering Medicines – Although treating gout with uric acid-lowering medications is important for many gout sufferers – and is often the best long-term solution for controlling gout – starting a new medication can actually trigger attacks. If gout symptoms seem to be developing after starting medication, call the medial professional who prescribed the medication before stopping or dismissing treatment

Trauma, Surgery or Sudden Illness – Those who are in bed or stationary for a long period of time are at higher risk

Radiation or Chemotherapy

From the *Gout and Uric Acid Education Society*

Gout triggers can differ from one person to another. Once a person identifies his or her specific trigge[illegible], gout can be easier to manage

Epidemiology
Prevalence

- The most common form of inflammatory arthritis of both men and women in the US
- Gout is predominately a disease of adult men, 9.2 million total population
- Risk increases with age. Peak incidence in the fifth decade
- Rarely occurs in men before adolescence or in women before menopause
- Prevalence 3.9% (5.9% men and 2% women)

Rheumatology Secrets, 2020

Total US Population for Some Rheumatic Conditions

- 1.3 M Rheumatoid Arthritis
- 2.7 M Carpel Tunnel Syndrome
- 5.0 M Fibromyalgia
- 7.1 M Back Pain with Limited Activity
- 9.2 M Gout

Prevalence of Gout and Hyperurecemia in the US General Population, *Arthritis and Rheumatism*, 201
and American College of Rheumatology Guidelines Database, 2020

HYPERURICEMIA

- Present in 5% of asymptomatic Americans on at least one occasion
- 15% will develop clinical gout, higher if uric acid > 9 mg/dL
- Duration and magnitude of hyperuricemia directly related to likelihood of subsequent attacks
- Current ACR 2020 recommendation* is to treat if uric acid > 9 mg/dL (with/without strong FHx), or tophi and/or joint erosions present, or acute gout flares ≥ 2/yr. Treat to target (< 6.0 mg/dL,< 5.0 mg/dL for tophi)

*Arthritis Care & Research Vol 72, No 6, June 2020, pp 744-760

Also See Rheumatology Secrets, 2020

GOUT
Differential Diagnosis

- Septic arthritis
- Cellulitis
- Acute pseudogout (CPPD)
- Rheumatoid arthritis
- Fracture

GOUT AND HYPERURICMIA
Classification

GOUT

- **Primary**

  - Most are undersecretors (90%), few are oversecretors

- **Secondary**

  - Undersecretion

  e.g. obesity, drugs (diuretic therapy), renal failure, lead nephropathy, thyroid disease, respiratory acidosis

  - Overproduction

  e.g. excess purine diet, ETOH abuse, lymph or myeloproliferative disorders

HYPERURICEMIA

- **Dietary excess of purines**
- **Overproduction of urate – purine precursor abnormality**
- **Undersecretion – abnormal handling of urate**

Optional Rheum Evaluation

Measurement of 24 hour urine for uric acid:
> 800 mg/24 hrs suggests OVER
< 800 mg suggests UNDER

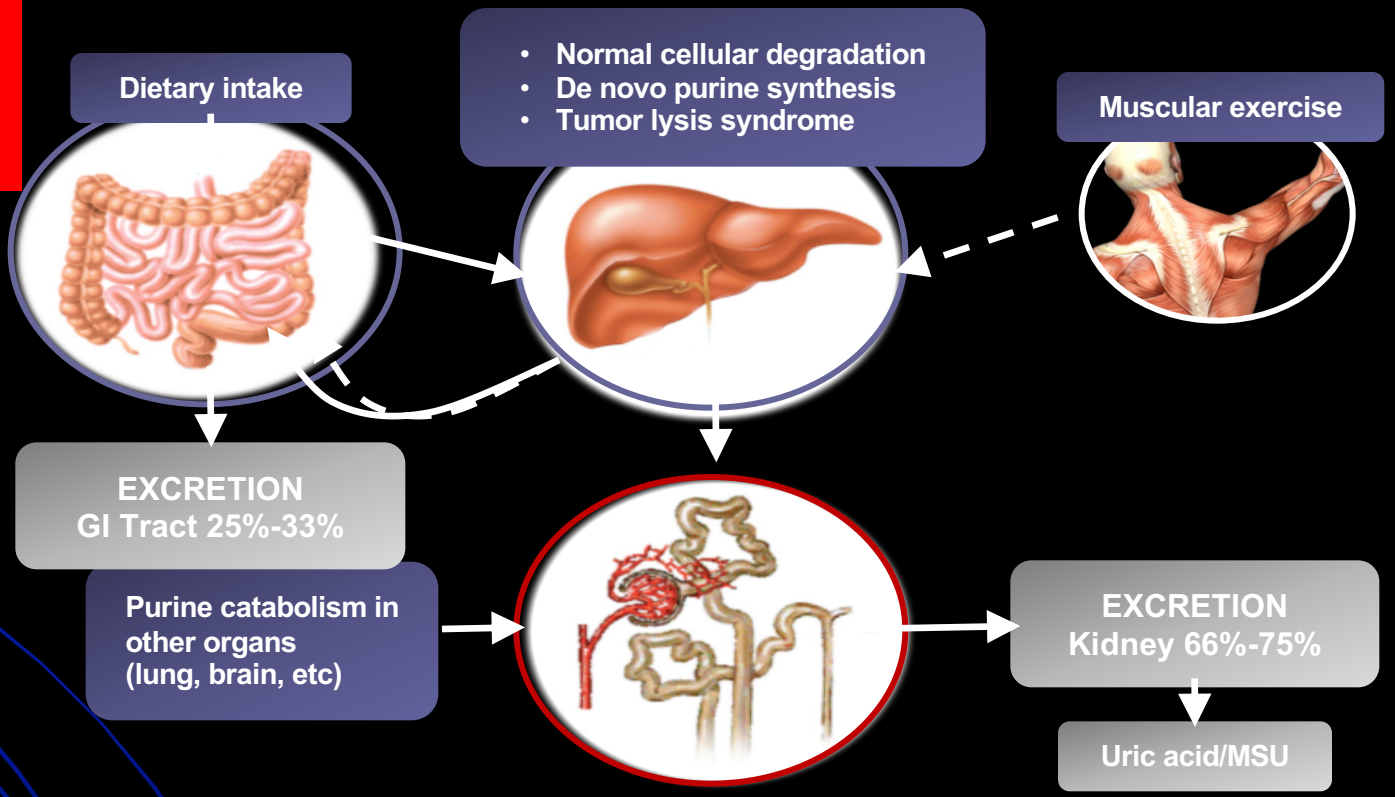
If elevated in urine, more aggressive treatment should be undertaken to prevent urate renal stones (Nephology Referral)

Rheumatology Secrets, 2020

The kidneys play an important role in the regulation of serum uric acid

Defective renal handling of uric acid is a frequent pathophysiologic factor sustaining hyperuricemia and gout[1]

Normal Human Urate Turnover[2-4]



1. Bobulescu IA, Moe OW. *Adv Chronic Kidney Dis.* 2012;19(6):358-371;
2. Hediger MA, et al. *Physiology*. 2005;20(2):125-133;
3. Bishop C, et al. *J Clin Invest.* 1951;30(8):879-888;
4. Terkeltaub R, et al. *Arthritis Res Ther.* 2006;8(suppl 1):S4.

The Typical Gout Patient Has Multiple Comorbidities

Demographics[1]

- Advanced age
- Male
- Postmenopausal women[2]

Comorbidities[2]

- Hypertension (74%)
- Chronic kidney disease (71%)
- Diabetes mellitus (26%)/ Obesity (53%)

Drugs that increase serum urate and may precipitate gout[1]

- Aspirin (low dose)
- Cyclosporine
- Chemotherapeutic cytotoxics
- Diuretics (especially thiazides and loops)
- Ethambutol
- Ethanol
- Levodopa
- Nicotinic acid
- Pyrazinamide
- Tacrolimus

Lifestyle[3]

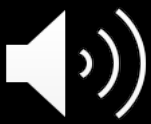
- Obesity (high BMI)
- Diet rich in meat and seafood
- High alcohol intake
- Fructose

1. McLean L. The pathogenesis of gout. In: Hochberg MC, Silman AJ, Smolen JS, Weinblatt ME, Weisman MH, eds., *Rheumatology*. 3rd ed. Edinburgh: Mosby; 2003:1903-1918.
2. Zhu et al. *Am J Med.* 2012; 125, 679-687.
3. Gout. The Arthritis Society of Canada. Accessed May 8, 2011. www.arthritis.ca/local/files/.../TAS%20Gout%20sheet_eng.pdf

Meds to Watch: Mostly from Reduced Elimination of Urate

- Diuretics (loop, thiazides)
- ACE Inhibitors - the "-il " drugs (ie, Lisinopril, Enalapril)
- Pyrazinamide (TB Rx)
- ASA
- Ethambutol (TB Rx)

- Angiotension Receptor Blockers (ARBs) – the "-tan" drugs ie, candesartan (Atacand), valsartan (Diovan)
- **Exception:** Losartan (Cozaar) – lowers uric levels



GOUT:
Clinical Presentation

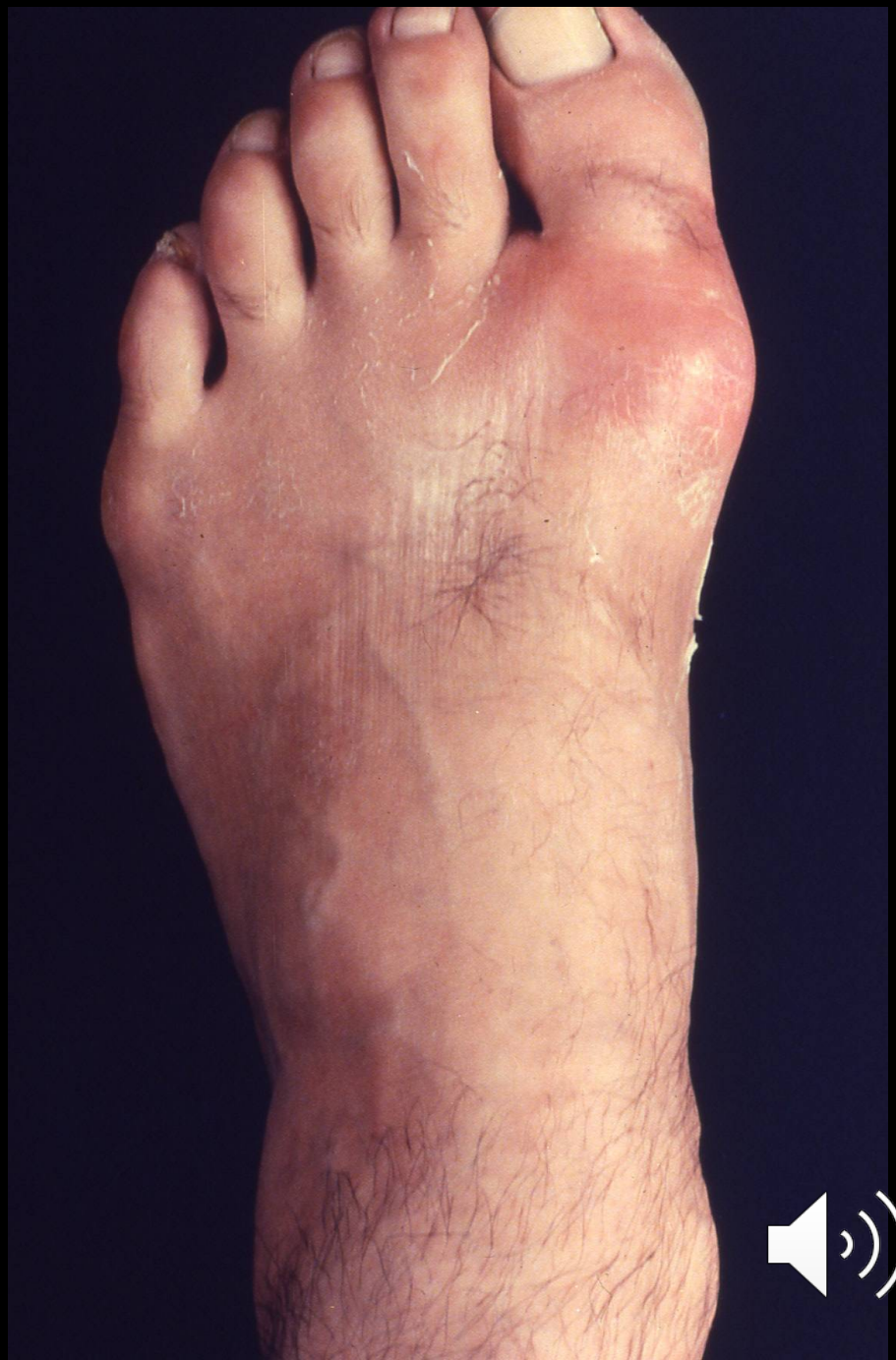
- Sudden onset of severe pain, swelling, and heat in one joint (usually)
- Often starts at night
- Joint is RED hot to the touch
- Precipitated by trauma, alcohol, or illness such as kidney disease
- 90 % Monoarticular
- 50 % are Big Toe (Podagra)
- Flares Can Occur More Often Over Time

Podagra (first MTP arthritis)

Gout was personified as one of the Greek deities, Podagra, born of Dionysus (Bacchus), the god of wine, and Aphrodite (Venus), the goddess of love

Podagra was the foot-torturer, a terrible-tempered virgin goddess

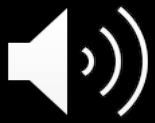
The idea was that gout resulted from service to these gods and was a consequence of overindulgence in sex, food and wine, as recorded by ancient Roman authors, and persisted into the Christian era



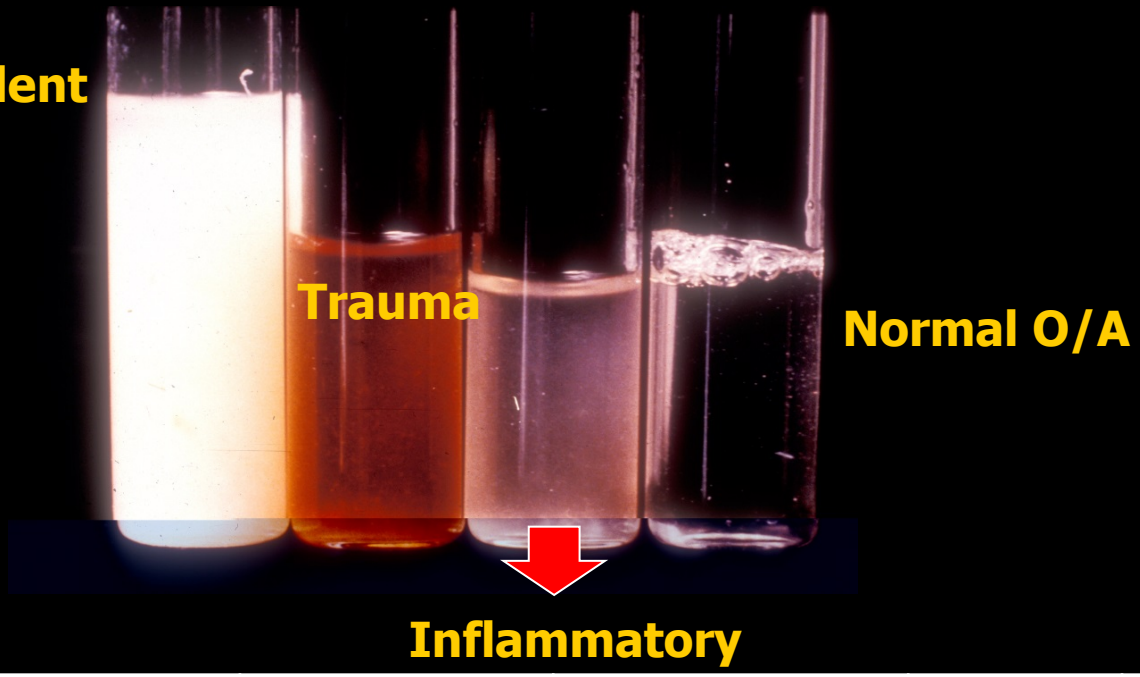
GOUT
Laboratory Findings

- **Uric acid levels-variable (< 8.0 mg/dL is "normal") but attacks can occur > 6.7 mg/dL**
- **Joint fluid will contain intracellular Monosodium Urate Crystals**
- **White cell counts in synovial fluid fall between 5,000-50,000 WBCs/mm²**

Serum urate levels alone **cannot** be used to Dx gout



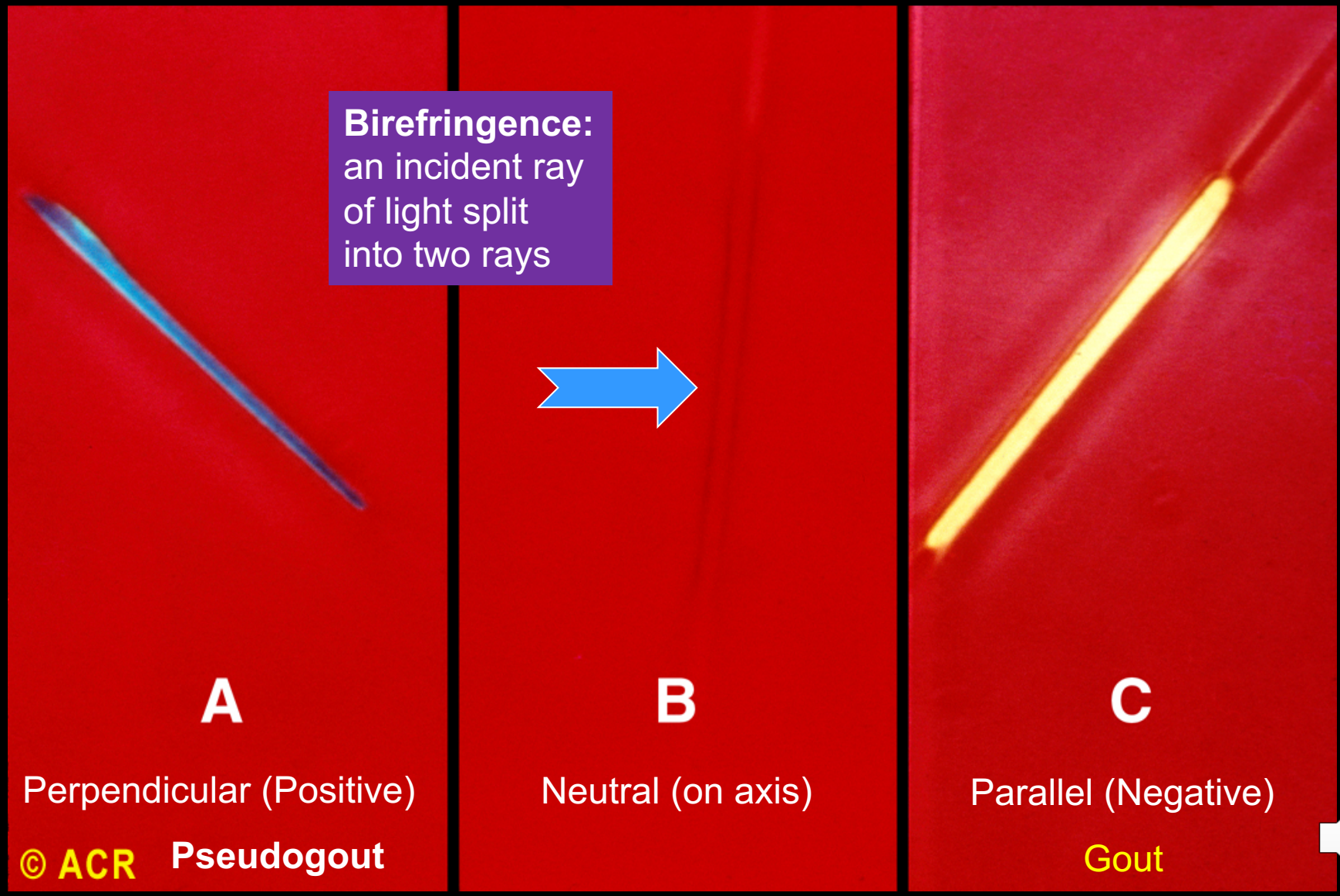
SYNOVIAL FLUID ANALYSIS*



Condition	Color	Clarity	* WBC	Cryst	C&S
OSTEO	Amber	Clear	200 -2,000	–	–
TRAUMA	Pink Red	Clear-opaque	<2,000	–	–
INFLAM	Yellow	Cloudy	2000-100,000	– +	–
BACTERIA	Purulent	Opaque	>50,000 (>95%PMNs)	– +	+

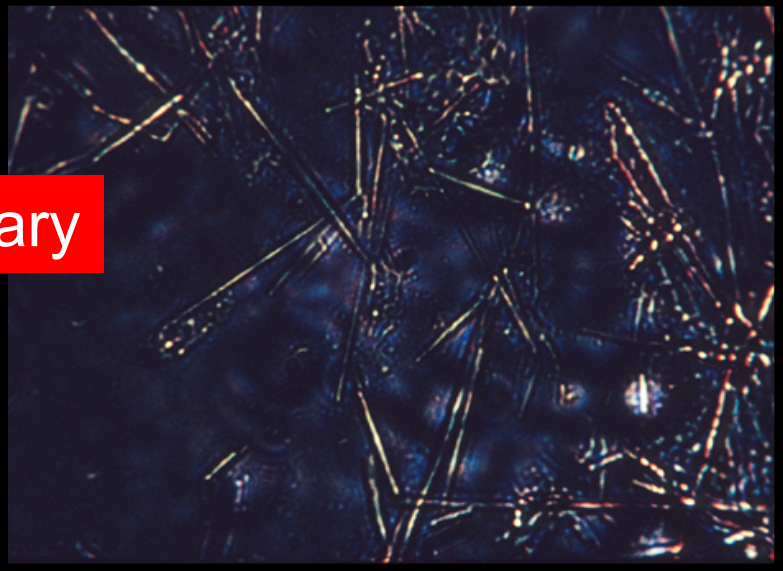
* REPEAT FLUID EXAM IMPROVES DIAGNOSTIC SENSITIVITY

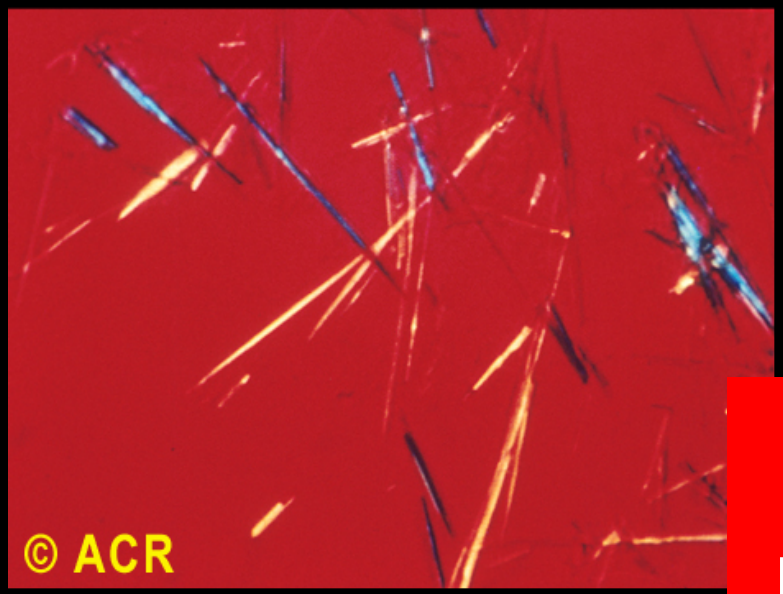
Urate Crystal: Gout vs Pseudogout (polarized light microscopy)

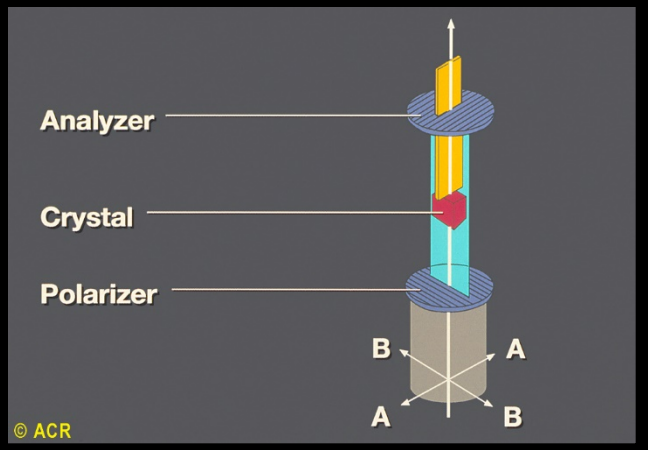


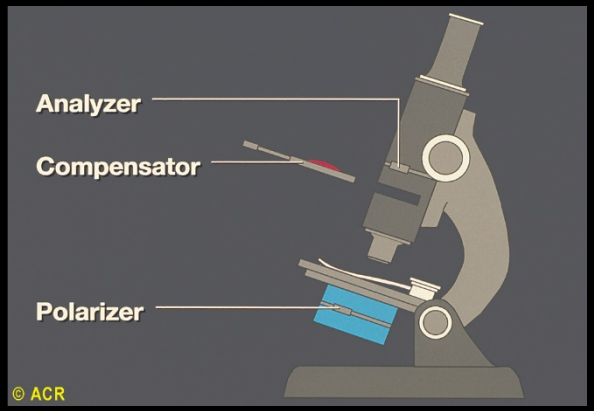
Gout: urate crystals (ordinary and polarized light microscopy)

Ordinary





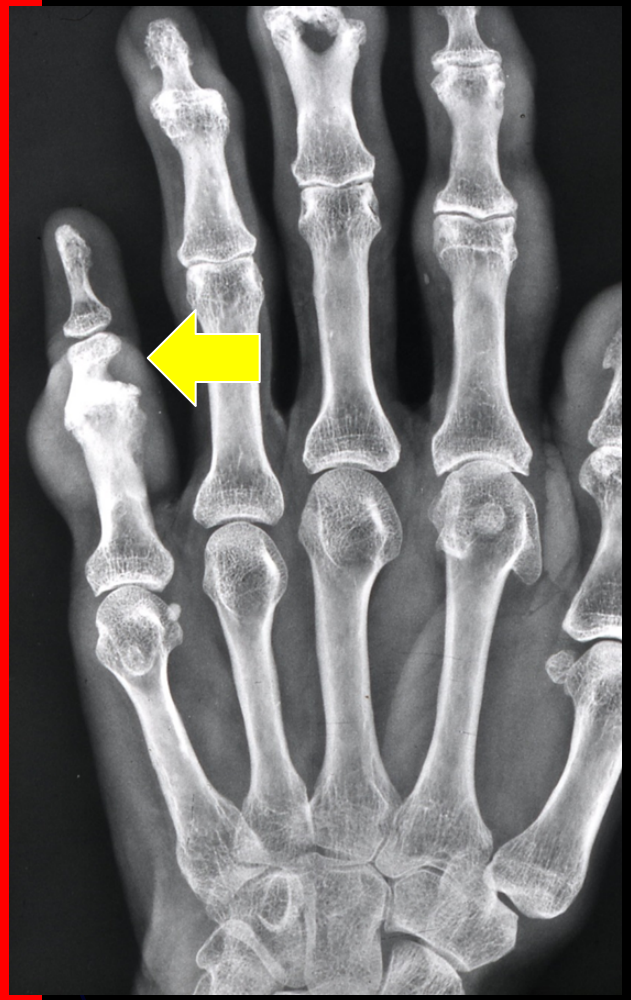


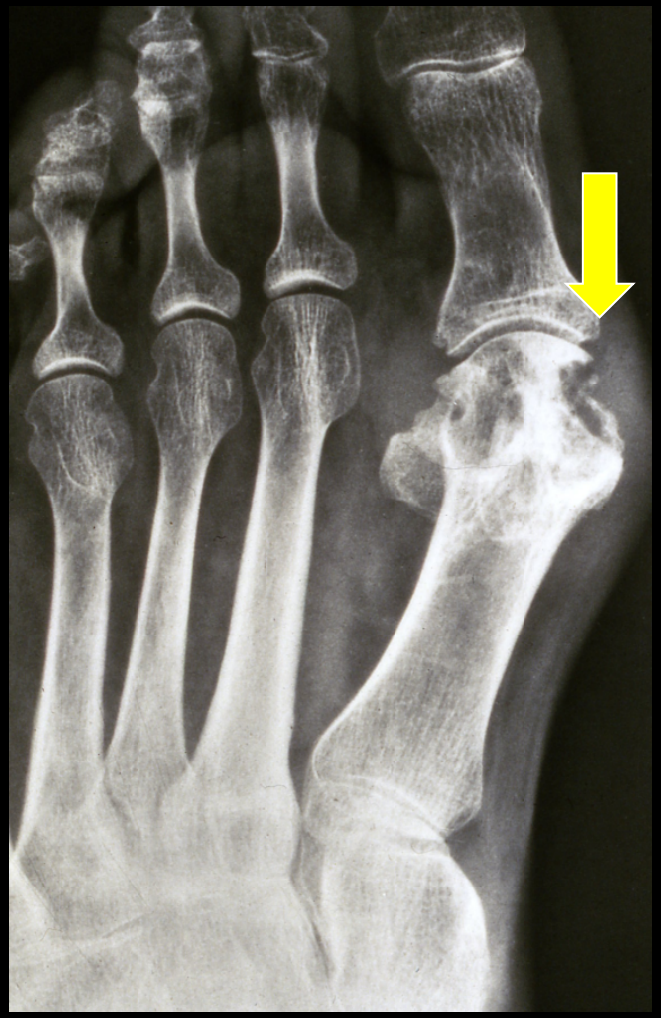


Monosodium urate:
Negatively
Birefringent Crystals

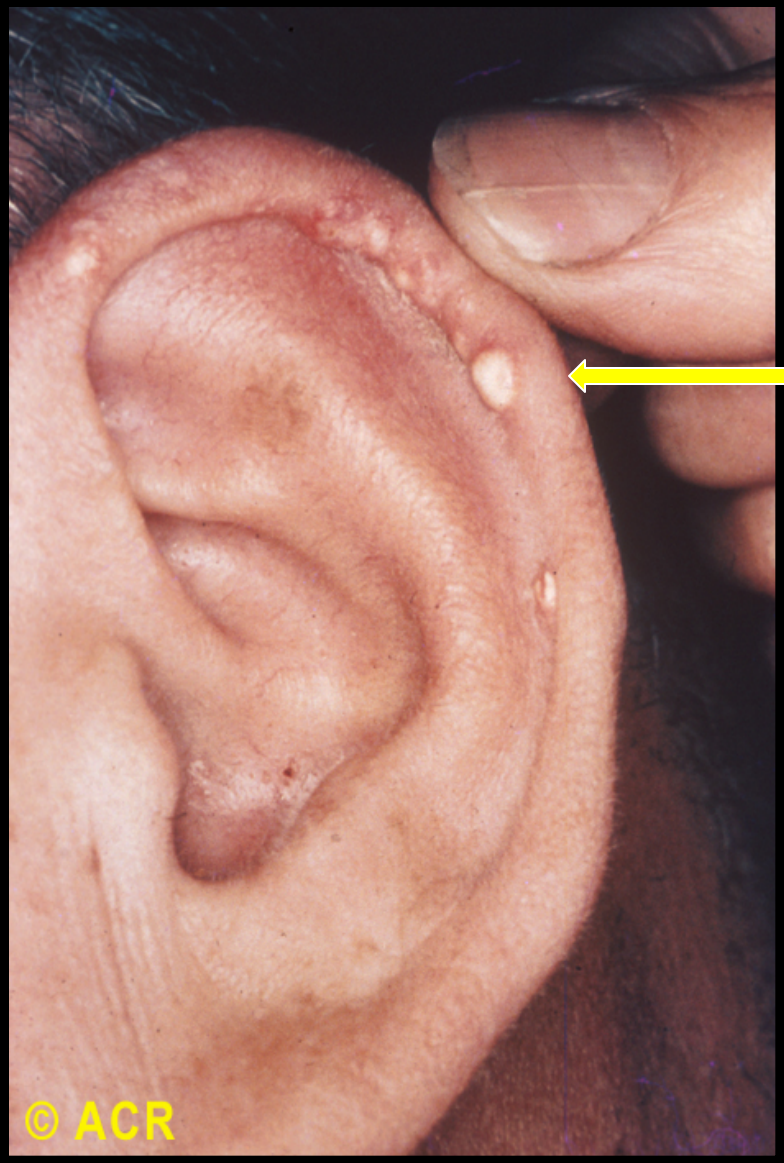
GOUT X-rays

- Early-soft tissue swelling
- Changes occur after years of disease
- Locations:
  - feet, hands
  - wrists
  - elbows
  - knees
- Gout erosions are slightly removed from the joint





"Mouse/Rat bite" and "punched-out" erosions



Gout Tophi

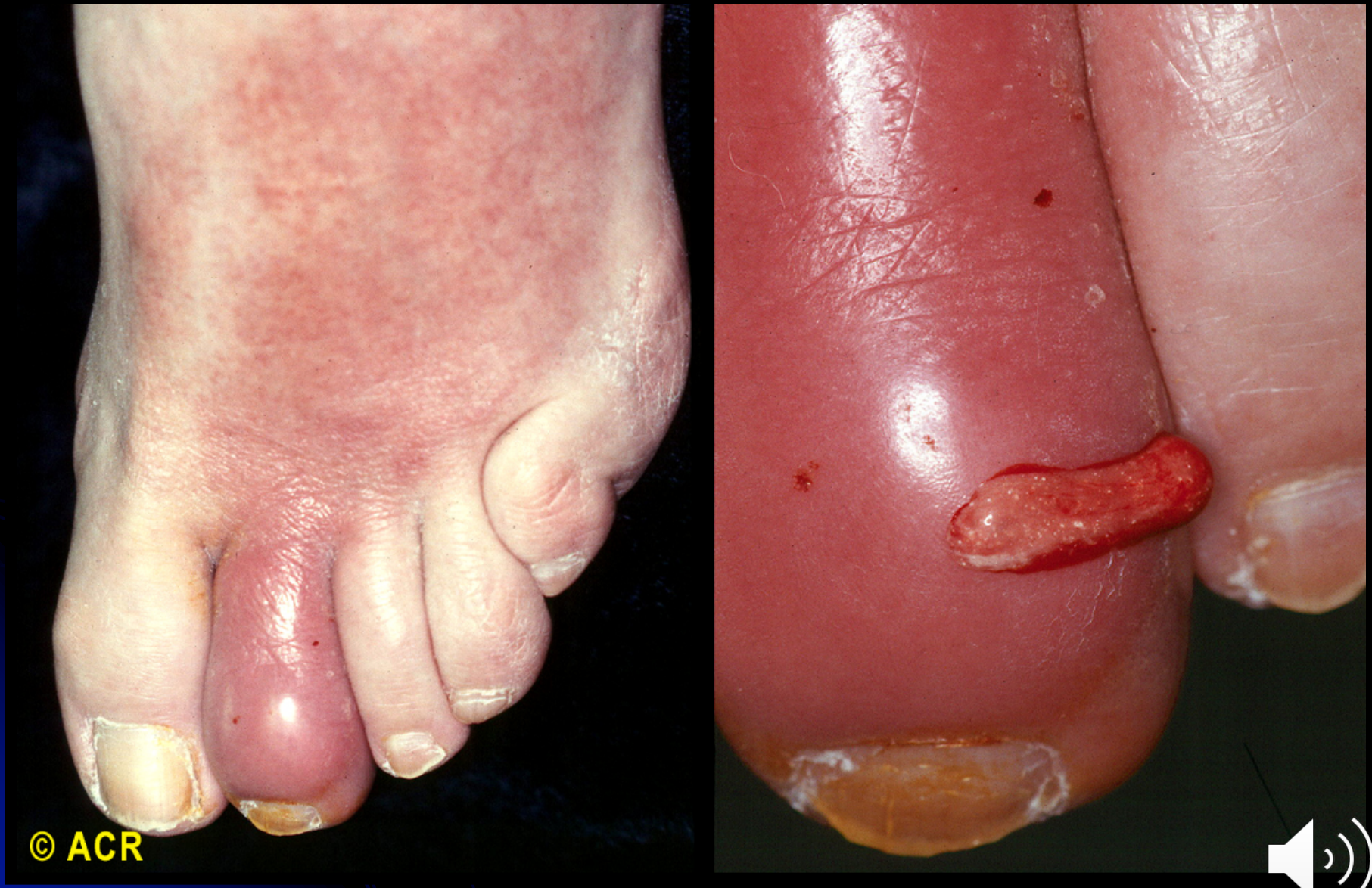
Uric acid solids seen in chronic gout (10-20 yrs) that collect in soft tissues such as:

Ear

Achilles tendon

Eyelids

OUCH!!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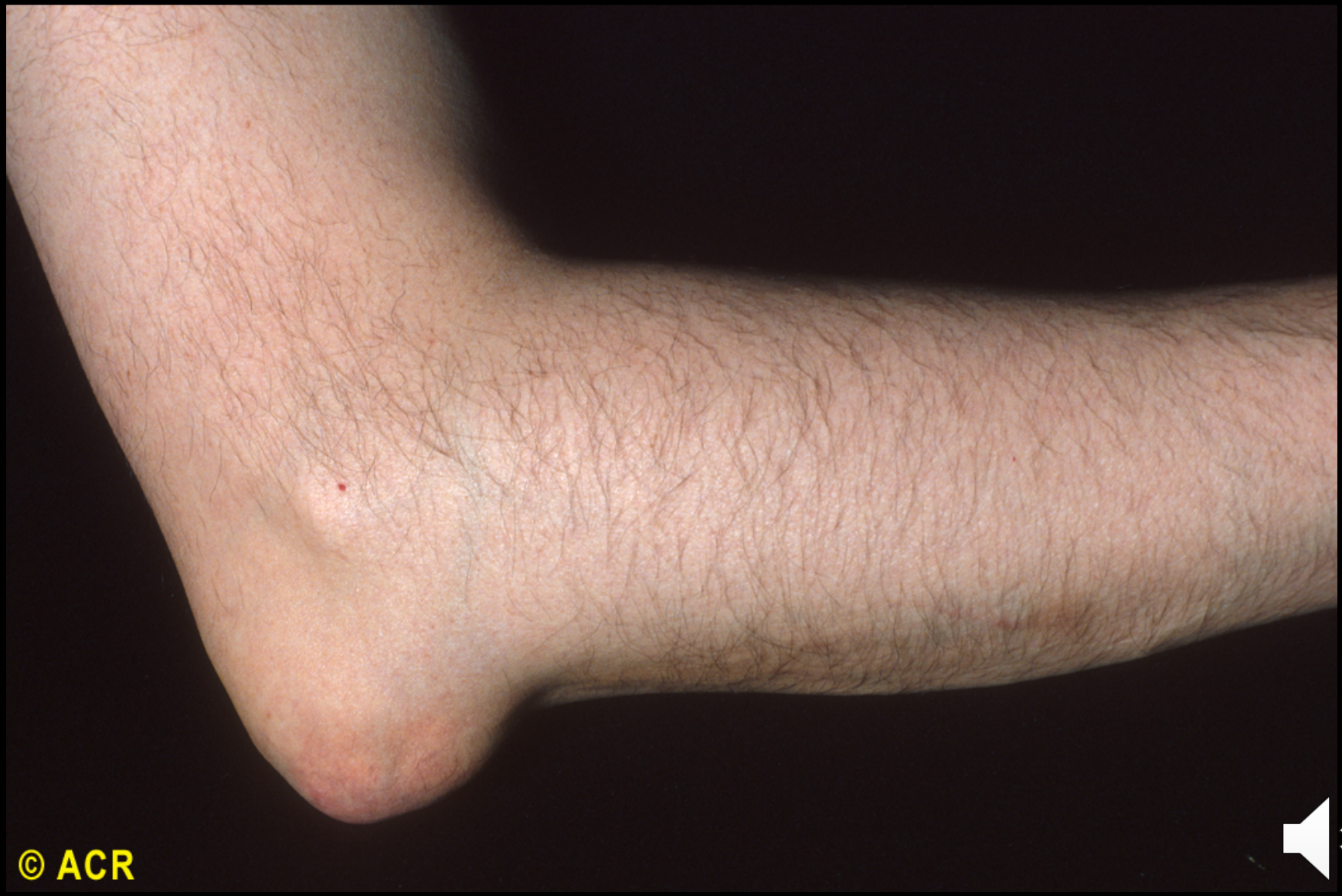
Gout: Olecranon Bursitis



Baseline Treatment

- **Diet:** avoid organ meats, high fructose corn syrup (POP !!), alcohol > 2 servings/day male, 1 serving/day female, limit beef, lamb, pork, sugar, shellfish
- **Comorbid causes:** DM, metabolic syndrome, drugs (diuretics), kidney disease
- **Evaluate:** for tophi, frequency (≥ 2 attacks/yr) and severity of acute disease (ie, joint erosions)

Health, Diet and Lifestyle Measures

Avoid:

- Organ meats high in purine content
- High fructose corn syrup (soda)
- ETOH overuse (male > 2/day, female > 1/day), avoid all use during acute flares or if in poor condition

Limit:

- Serving sizes of beef, lamb, pork, some seafoods (sardines, shellfish)
- Servings of natural sweet juices, table sugar, beverages, desserts
- ETOH

Encourage:

- Low or nonfat dairy
- Vegetables
- Weight loss
- Exercise
- Plenty of fluids
- Stop smoking
- Overall Healthy Lifestyle

Acute (Initial) Treatment Options ACR Guidelines 2020

Colchicine (low dose), or NSAIDs, or steroids as first line agents

- PO **Colchicine** (Colcrys) 0.6 mg 1-2 x day x 3 days (inhibits neutrophil motility and activity, leading to a net anti-inflammatory effect)
- **Naproxen** 500 mg 2 x day. Can use celecoxib and sulindac as alternatives. AVOID ASA.
- PO Steroids 40-60 mg/day x 3 days, taper by 10-15 mg/day q 3 days until off. Intra-articular steroid injection (Dose by joint)
- Cold compresses, rest

Structural Classes/Risks of NSAIDs

Proionic acid

Ibuprofen
Fenoprofen
Naproxen*
Oxaprosyn
Ketoprofen
Flurbiprofen

* No cardiac Risk

Acetic/Fenamic Acid

Sulindac
Indomethacin
Diclofenac
Etodolac
Tolmetin
Meclofenamate

Enolic/ Carboxylic Acid

Piroxicam
Meloxicam
Ketorolac

Nonacidic Comp

Nabumetone

Salisalates

ASA*
Na Salic
Salsalate
CholMgTrisal
Diflunisal

Cox-2 Inhibitors

Celecoxib
Etoricoxib and
Lumiracoxib
(not FDA Approved)

Risks

Previous GI NSAID complications
Previous ulcer
Smoking
Use with Corticosteroids
Use with Anticoagulants
Age
Multiple medical problems:
- **Hypertension**
- **Edema**
- **Renal**

Colchicine (Colcrys™) 0.6mg

- Indication for the acute treatment of gout

  SIG: At first onset of symptoms

  1.2 mg followed by 0.6mg in one hour, then q12h
- Side effect profile: Diarrhea 23% Nausea 4%
- **Caution:** may be used in mild to moderate renal or hepatic impairment. Not indicated for severe renal or hepatic impairment who are on concurrent strong inhibitor of P-gp inhibitors or CYP3A4.
- Indication: for the prophylaxis of gout when starting ULT. Sig: 0.6 QD to BID

Glopera available as a liquid option, from 0.1 - 0.6 mg

Colcrys ™ Package Insert

GOUT Urate Lowering Agents

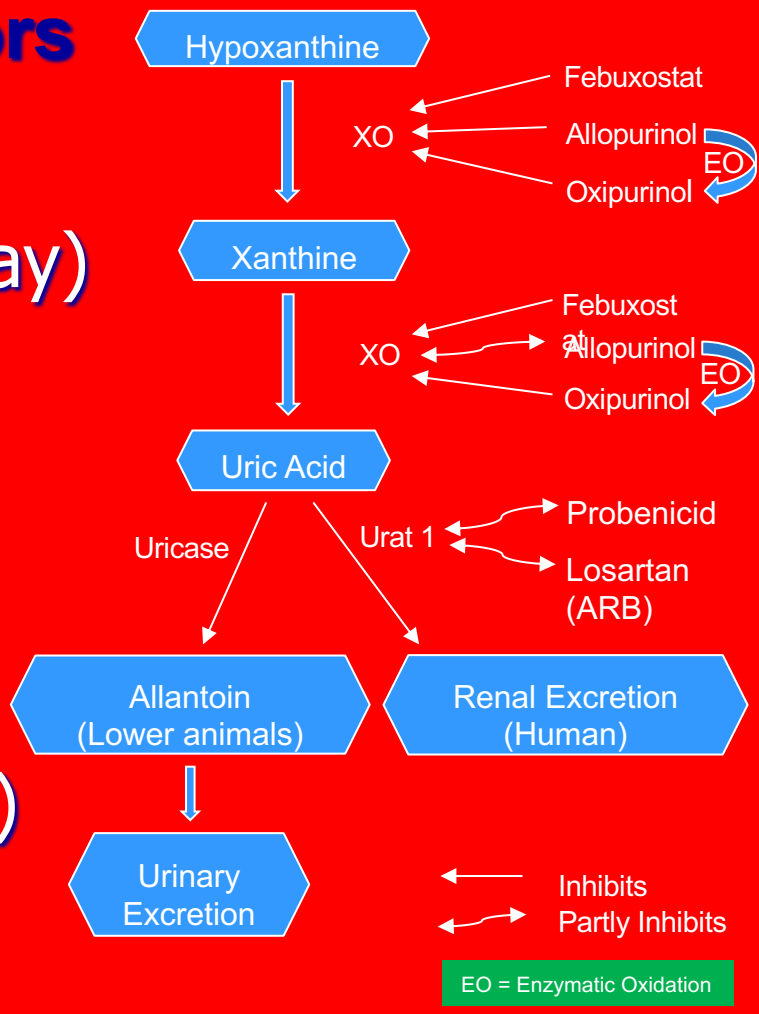
- **Xanthine Oxidase Inhibitors**

Allopurinol (100-300 mg/day)

Febuxostat** (40 or 80 mg/day)

- **Uricosuric Agents**

Probenecid (250-500 mg BID x 1 week then 500 mg BID, may need to go higher depending on serum uric acid)



Lesinurad (Zurampic) (NEWER)
200 mg QD; needs to be used with an XO Inhibitor. Caution in patients with a creatinine clearance < 45. Monitor kidney function routinely
NOTE: Lesinurad not available in US as of 2/1/2019

** **BLACK BOX WARNING** for increased risk of cardiovascular death compared to Allopurinol
FDA Nov, 2017
www.thelancet.com Published online November 9, 2020 https://doi.org/10.1016/S0140-6736(20)32234-0

*ACR 2020 Guidelines: Urate Lowering Treatment (ULT) for the gout patient with renal impairment

Considerations for patients with renal impairment	
Medication	**Notes**
Allopurinol	• Recommended as a first-line pharmacologic approach • **Patient should not be started on a dose >100 mg/day** • For any patient with CKD in stage 4 (Cr_{Cl} 15-30mL/min or worse), start at 50 mg/day, titrate up every 3-4 weeks • Dose can be increased up to 800 mg daily, even with renal impairment, as long as it is accompanied by **adequate patient education and monitoring for drug toxicity (eg, pruritus, rash, elevated hepatic transaminases)** • These patients may be at higher risk of **allopurinol hypersensitivity syndrome (AHS) – watch for a rash, looks like measles**
Febuxostat	• Recommended as a second-line pharmacologic approach **No dosage adjustment in patients with mild to moderate renal impairment (Cr_{Cl} 30-89 mL/min)** **≥ Stage 3 start with ≤ 40 mg/day** **Contraindicated in pts on:** **-azathioprine** **-mercaptopurine** **-theophylline**
Probenecid	• **Probenecid is NOT recommended as a first-line ULT** in patients with a Cr_{Cl} <50 mL/min, stage 3 or higher

*Arthritis Care & Research Vol 72, No 6, June 2020, pp 744-760

ACR 2020 Guidelines: flare prevention and long-term management of gout

Recommendation	PICO question	Certainty of evidence
For patients starting any ULT, we strongly recommend allopurinol over all other ULT as the preferred first-line agent for all patients, including in those with CKD stage ≥3.	10	Moderate
We strongly recommend a xanthine oxidase inhibitor over probenecid for those with CKD stage ≥3.		
For allopurinol and febuxostat, we strongly recommend starting at a low dose with subsequent dose titration to target over starting at a higher dose (e.g., ≤100 mg/day [and lower in patients with CKD] for allopurinol or ≤40 mg/day for febuxostat).	7	Moderate
For probenecid, we conditionally recommend starting at a low dose (500 mg once or twice daily) with dose titration over starting at a higher dose.		
We strongly recommend initiating concomitant antiinflammatory prophylaxis therapy (e.g., colchicine, NSAIDs, prednisone/prednisolone) over no antiinflammatory prophylaxis. The choice of specific antiinflammatory prophylaxis should be based upon patient factors.	9	Moderate
We strongly recommend continuing prophylaxis for 3–6 months rather than <3 months, with ongoing evaluation and continued prophylaxis as needed if the patient continues to experience flares.	9	Moderate
When the decision is made that ULT is indicated while the patient is experiencing a gout flare, we conditionally recommend starting ULT during the gout flare over starting ULT after the gout flare has resolved.	6	Moderate
We strongly recommend *against* pegloticase as first-line therapy.	10	Moderate†

Strongly recommend | Conditionally recommend | Strongly recommend against | Conditionally recommend against

* PICO = population, intervention, comparator, outcomes; CKD = chronic kidney disease; NSAIDs = nonsteroidal antiinflammatory drugs.
† Moderate evidence is in support of the efficacy of pegloticase, but due to cost, safety concerns, and favorable benefit-to-harm ratios of other untried treatment options, the recommendation is *against* using pegloticase as first-line agent.

Continue gout flare prophylaxis if there are ongoing symptoms or signs

Regularly monitor for sUA and ULT side effects

After palpable tophi and all acute and chronic gouty arthritis symptoms have resolved, continue all measures, as needed, to maintain sUA <6 mg/dL

Note: ACR 2020 Guidelines may conflict with other specialty recommendations, esp treat-to-target

Refer to a specialist when there is:

- **–Unclear etiology of hyperuricemia**
- **–Refractory signs and symptoms of gout**
- **–Difficulty in reaching target sUA, particularly with renal impairment and a trial of xanthine oxidase inhibitor treatment**
- **–Multiple and/or serious adverse events from pharmacologic ULT**

IV Gout Treatment: Pegloticase (Krystexxa)

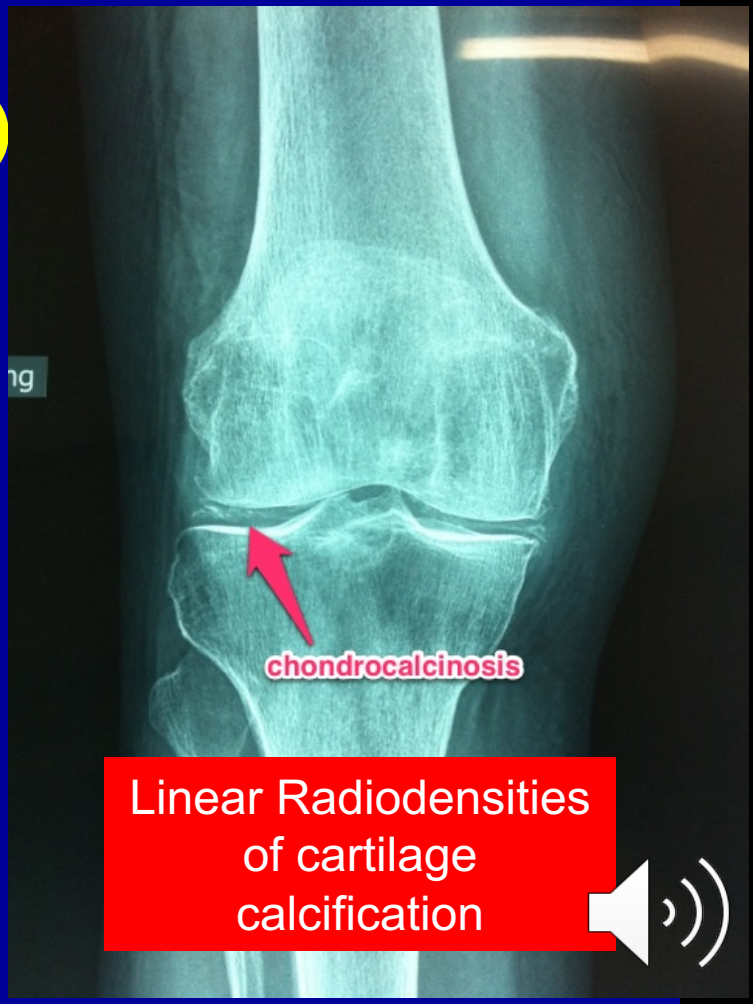
- PEGylated uric acid enzyme
- IV Infusion Only (Need Rheumatology Referral)
- Use only if xanthine oxidase inhibitors fail
- Significant Tophus Resolution
- Important to screen for G6PD Deficiency (Glucose-6-phoshate dehydrogenase)
- Most Common SEs: gout flares(77%), infusion reactions (26%), nausea (12%), contusion (11%), nasopharyngitis (7%)

GOUT:
Lowering Serum Uric Acid

- **Be careful if treating elevated serum uric acid during acute attack. May get repeat attacks!**
- If patient is on uric acid agent (ie, Zyloprim) leave the dose the same
- When attack has abated then use agents to lower uric acid
- Start dose low and go slow! Need lab <6.0. Can check it every 2-3 weeks

PSEUDOGOUT

- Acute pseudogout refers to acute gout-like attacks of inflammation that occur in one or more joints lasting for several days to weeks
- **Calcium PyroPhosphate Dihydrate crystals (CPPD) in synovial fluid is pathopneumonic for Dx**
- ~ 50% of attacks occur in the knees
- Like gout attacks, can occur spontaneously or be provoked by trauma, surgery, severe illness
- Population is usually older than gout pts



PSEUDOGOUT

Prevalence

Diseases Strongly Associated with CPPD:

Aging **Osteoarthritis**
Gout Hemochromatosis
Hyperparathyroidism Hypermagnesemia
Hypophosphatasia

8% of the adult population (> 60 yo) have articular CPPD deposits on knee radiographs

and

Prevention

- **Colchicine**
- **Low dose NSAIDS**
- **Low dose steroids in resistant cases**

Acute Treatment: Steroid joint injection initially, NSAIDs, colchicine
Chronic Treatment: NSAIDs, colchicine

Rheumatology Secrets 4th Ed, 2020

“Atypical presentations of common diseases are usually something else”

-Thomas Ignaczak, MD
Rheumatologist

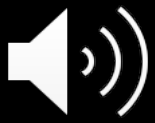
Take Home Points

1. Gout should be recognized and managed as a chronic disease, not an acute condition
2. Knowing the stages, triggers and early intervention are key in the prevention of joint damage
3. Chronic gout deformities can have a terrible cost in a patients lifestyle

ARS Question One

A joint positive for gout most likely will have what negatively birefringent needle-shaped crystals seen on synovial fluid analysis?

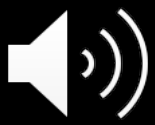
A. Monosodium Urate Crystals
B. Calcium Pyrophosphate Crystals
C. Dilithium Crystals



ARS Question Two

The recommended first line (initial) treatment for acute gout is:

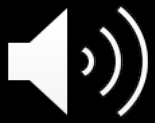
A. Allopurinol
B. Colchicine or NSAIDs or Steroids
C. Allopurinol and Steroids



ARS Question Three

What xray finding is often pathognomonic for CPPD Disease?

A. Sclerosis and Osteophytes Formation
B. “Punched out” or “Mouse bite”erosions
C. Chondrocalcinosis



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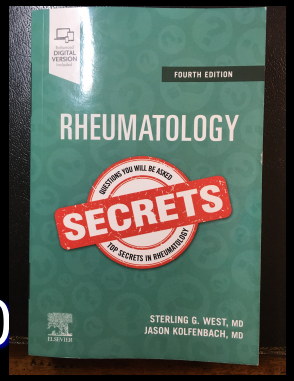

- Rheumatology Secrets
  4rd Ed
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- American College of Rheumatology Slide Series
- ACR Gout Management Guide
  Arthritis Care & Research
  Vol 72, No 6, June 2020
  pp 744-760

- Thomas Ignaczak, MD
- PANCE/PANRE Slide Info from PANCE Prep Pearls 2nd ED (Williams) 2017

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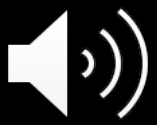
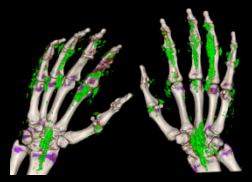


Remember:
Autoimmune Disease Affects All Age Groups

Cheesesteak anyone ?

PANCE/PANRE Review 1

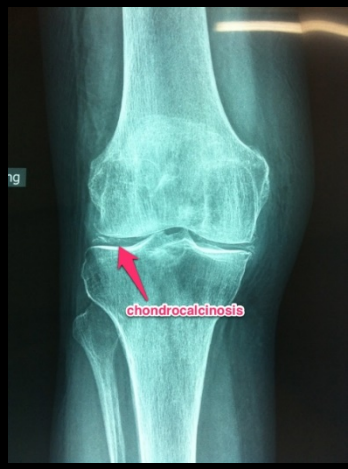
Gout Arthritis: Crystal-induced arthropathy



- **Gout:** Uric acid deposition (Mono Sodium Urate) in soft tissue, joints and bones caused by rapid changes in uric acid levels from purine rich foods (ETOH, liver, oily fish, yeasts) or meds (loop/thiazide diuretics, ARBs, ACEi, TB Rx, ASA). Most common in men > 30 y/o
- **Acute Flare:** 80% monoarthropathy (severe pain, redness, swelling). Knees, feet, ankles common. **Podagra - 1st MCP involvement.** Chronic causes tophi deposition (ie, ears, Achilles). Can cause uric acid stones (low urine vol/acidic pH)
- **Dx: negatively birefringent needle-shaped urate crystals.** Xrays: "Mouse/rat bite" "punched-out" erosions. Lab: uric acid ⇧
- **TX: Acute:** Colchicine, NSAIDs, steroids first drugs of choice(ie Indocin, Naprosyn). Adjust doses for CKD (Acute and Chronic)
- **Chronic:** Allopurinol (xanthine oxidase inhibitor), Febuxostat (Uloric), Colchicine, Probenecid (Urocosuric drug), Vit C, Losartan (for HTN)
- **Lab:** Lower Uric acid < 6.0 mg/dL, < 5.0 mg/dL if tophi present
- **SE:** GI irritation, Stephen-Johnson Syndrome

PANCE/PANRE Review 2

CPPD Arthritis: Crystal-induced arthropathy



- **CPPD (Pseudogout):** Calcium pyrophosphate deposition in soft tissue, joints causing inflammation and destructive bone lesions. Often associated with other diseases (ie, OA, DM, hyperthyroidism). Most common in elderly > 60 y/o, females
- **Acute Flare:** (pain less severe than gout, redness, swelling). Knees most common. Chronic disease resembles RA
- **Dx: positively birefringent, rhomboid-shaped crystals.** Xrays: **Chondrocalcinosis** (cartilage calcification) shows linear radiodensities Lab: synovial fluid analysis only
- **TX: Acute:** Intraarticular Steroids 1st line, NSAIDs, Colchicine (Both A&C) **Chronic:**Colchicine. No treatment if asymptomatic

Thank You
for Your Time and Interest

Elfreths Alley

My Friend Julia Swafford PA-C, SPAR HOD Rep

2016 AAPA Annual Conference

San Antonio, TX

See Extra Slides of Interest

gnle852@gmail.com

Extra Slides of Interest

Rheumatology Pearls
Pseudogout

- CPPD diseases are thought to be diseases of the elderly with increasing frequency after the age of 55.
- There is no gender or ethnic/racial predilections.
- Prevalence in the population is not increasing as it is with gout.
- Unfortunately correcting underlying metabolic conditions does not resolve the crystals deposited.
- CPPD should be considered in the elderly who are diagnosed with seronegative RA involving the wrists, MCPs, and shoulders

Clinical Presentations of CPPD Deposition

1) Acute mono-arthritis (pseudogout)
2) Polyarticular noninflammatory arthritis (pseudoOA)
3) Polyarticular inflammatory arthritis (pseudoRA)
4) Neuropathic-like joint destruction (pseudo-Charcot)
5) Asymptomatic

Rosenthal AK. In, Crystal Induced Arthropathies (ed Wortmann et al), 99-116, 2006