Crystal Arthritis

Farhan Tahir MD, FACR

April 26, 2012

Agenda

- History of crystal arthritis
- Introduction to Gout
- Gout pathophysiology, risk factors, clinical stages, joint destruction and extra articular gout
- Treatment options and targets
- CPPD

Crystal Arthritis

- Gout : oldest diseases in the medical literature
- Greeks authors have written about gout as the result of personal excess
- Association with a diet rich in meat and alcohol gained it the reputation, "the king of diseases and the disease of kings".
- Lowenhook described symptoms of gout in the 1600s.
- In 1848, Sir Alfred Garrod linked gout with hyperuricemia
- Pathophysiology of acute gouty arthritis was not described fully until 1962
- Pseudogout, which may be clinically indistinguishable from gout, was recognized as a distinct disease entity in 1962

Gout

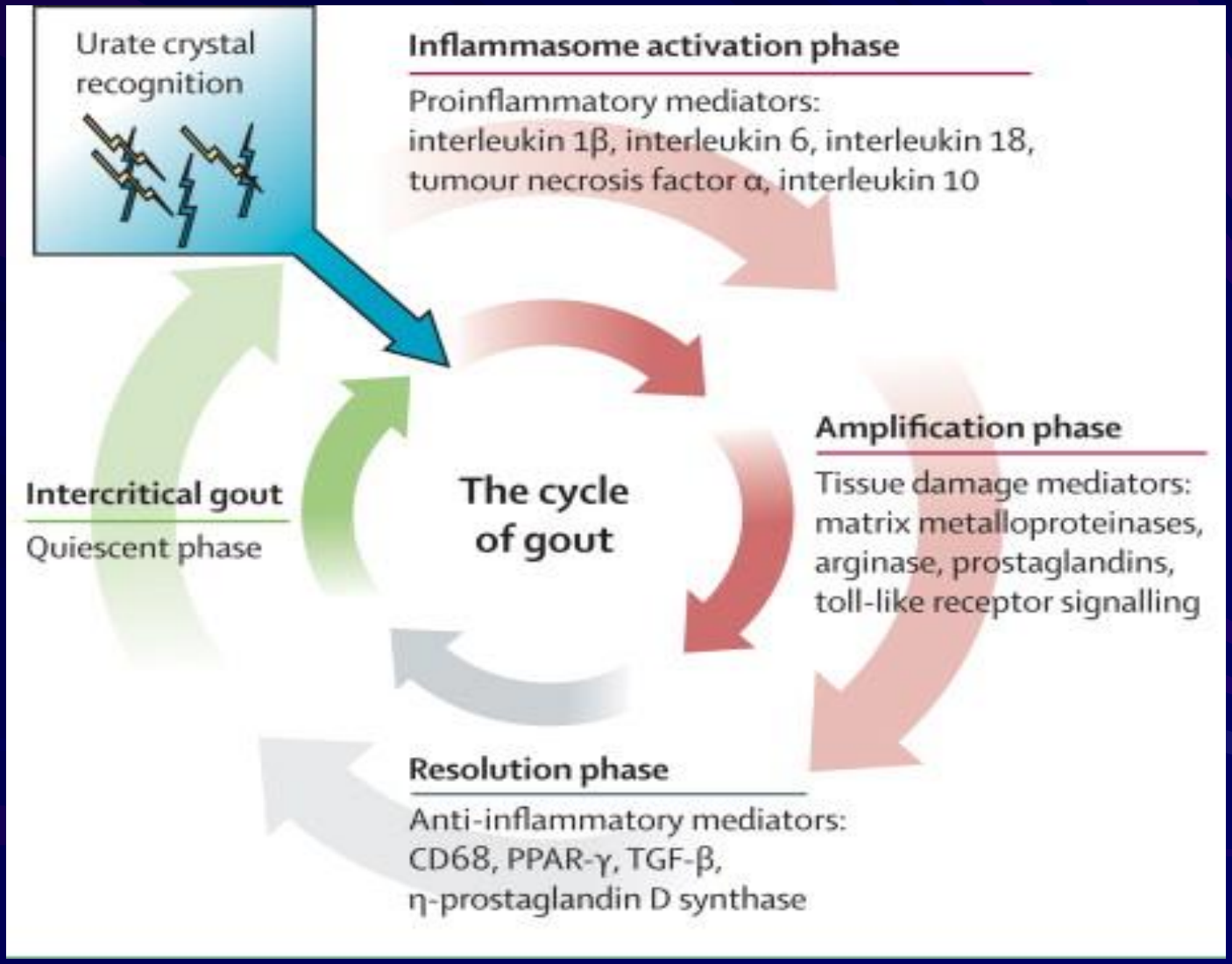
- Inflammatory arthritis mediated by the crystallization of uric acid within joints and soft tissues forming tophi
- Often associated with prolonged periods of undiagnosed hyperuricemia
- Clinically undetectable tophi can only seen on advanced imaging
- Prevalence is rising in US; 8.3 million, only 3.1 million getting treatment

Gout Epidemiology

- 40% are not adequately treated to bring target uric acid below 6mg/dl
- Poor treatment adherence is a huge problem; 56%
- Most labs list upper limit 8mg or 8.5mg/dl, ignore that, simply the results of changing demographics, obesity
- Asymptomatic hyperuricemia >6.8mg/dl
- Target serum urate <6mg/dl

Figure 1. The Gout *(1799), by the artist James Gillray, depicts the disease as an evil demon attacking a toe.*

The Cycle of Gout



Lancet, Volume 377, Issue 9760, 8–14 January 2011, Pages 165–177

Hyperuricemia – Preclinical Period

- >6.8mg/dl – super saturation
- Onset males age 30, females postmenopausal
- Duration 10-15 yrs before "gout"
- 80% due to under secretion
- 20% due to over production
  - Determined by 24 hr urine collection BUT Spot urine fractional excretion can be used
- Uric acid above 9mg/dl carries 22% risk of future attack in 5 years

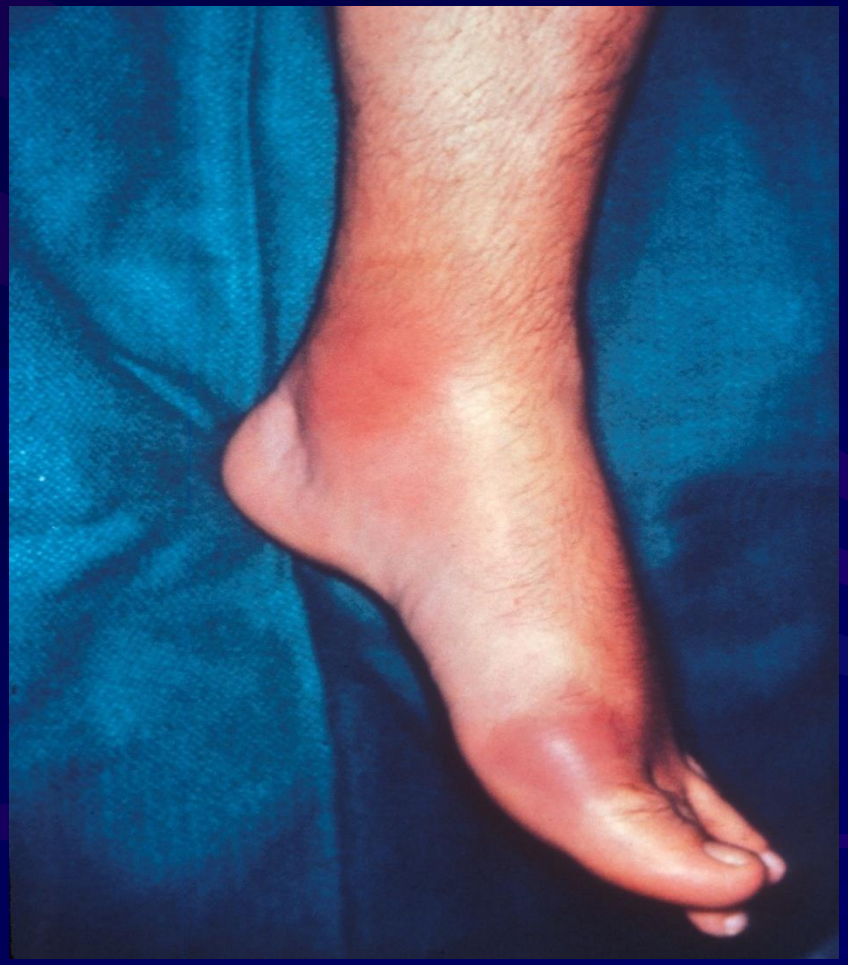
Intercritical Period

- 70% prevalence of MSU crystals remain in the joint so, joint aspiration can be attempted to prove diagnosis if not done in the past
- This phase lasts months to years for 75-80%
- 20% never have another attack

Chronic Gout

- > 10 years
- Tophi
- Chronic joint swelling
- Joint destruction
- Frequent and more severe attacks
- Urate precipitation leads to acute gouty arthritis
  - Local factors – temperature, pH, trauma
  - Systemic factors – hydration state, infections, meds, alcohol, co-morbid conditions-CHF
- Don’t ignore asymptomatic hyperuricemia, needs notice and monitoring for metabolic conditions but literature doesn't support uricosuric therapy

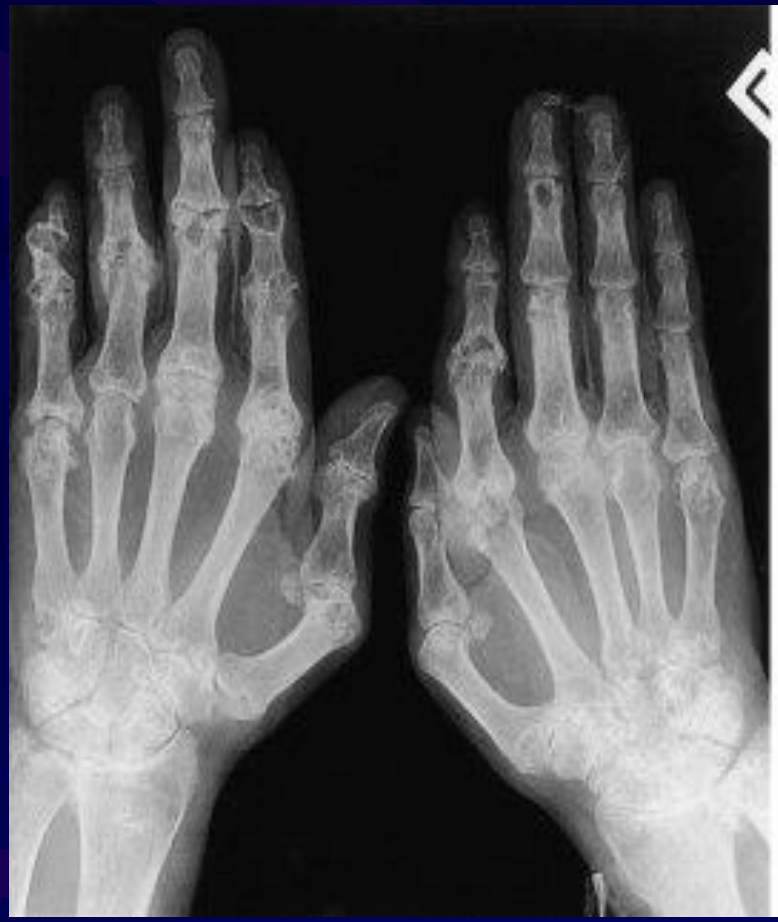
Typical Acute Attack

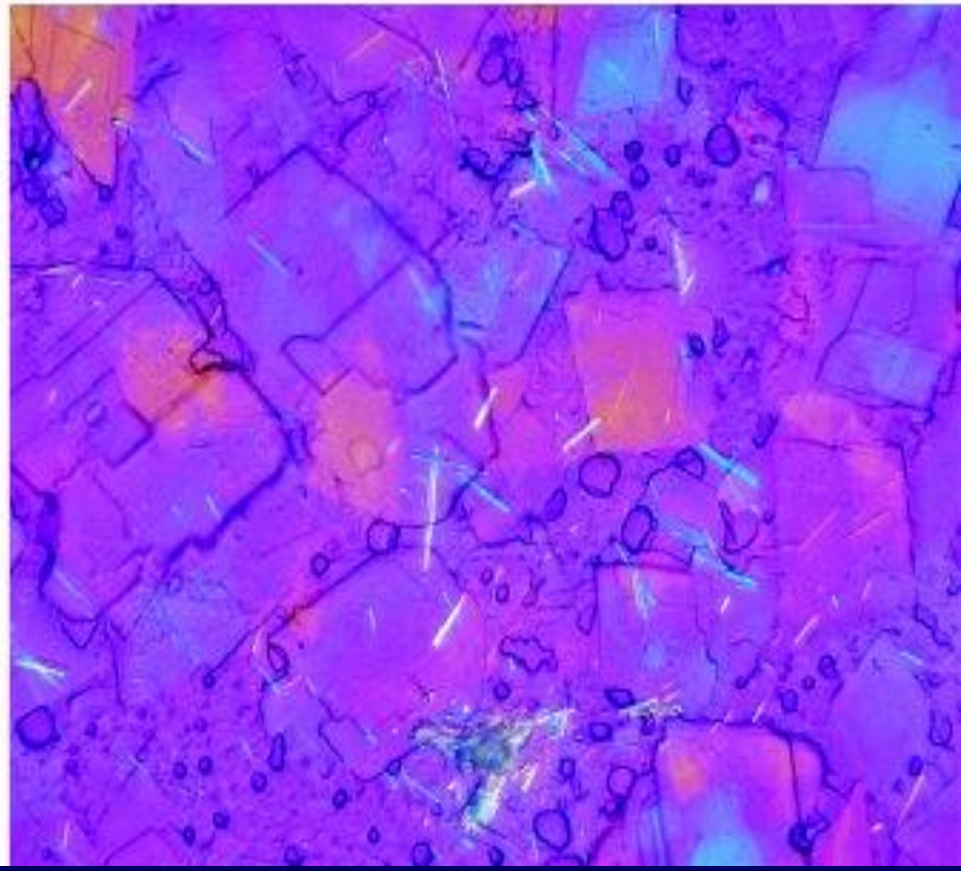


Atypical is not very uncommon Pseudo-rheumatoid gout

- The patient, a 68-year-old man with a 30-year history of seronegative yet nodular rheumatoid arthritis, presented for reevaluation
- Hand radiography revealed soft tissue swelling and extensive joint destruction with erosions
- Most of the erosions were “punched out” or overhanging

Pseudorheumatoid gout

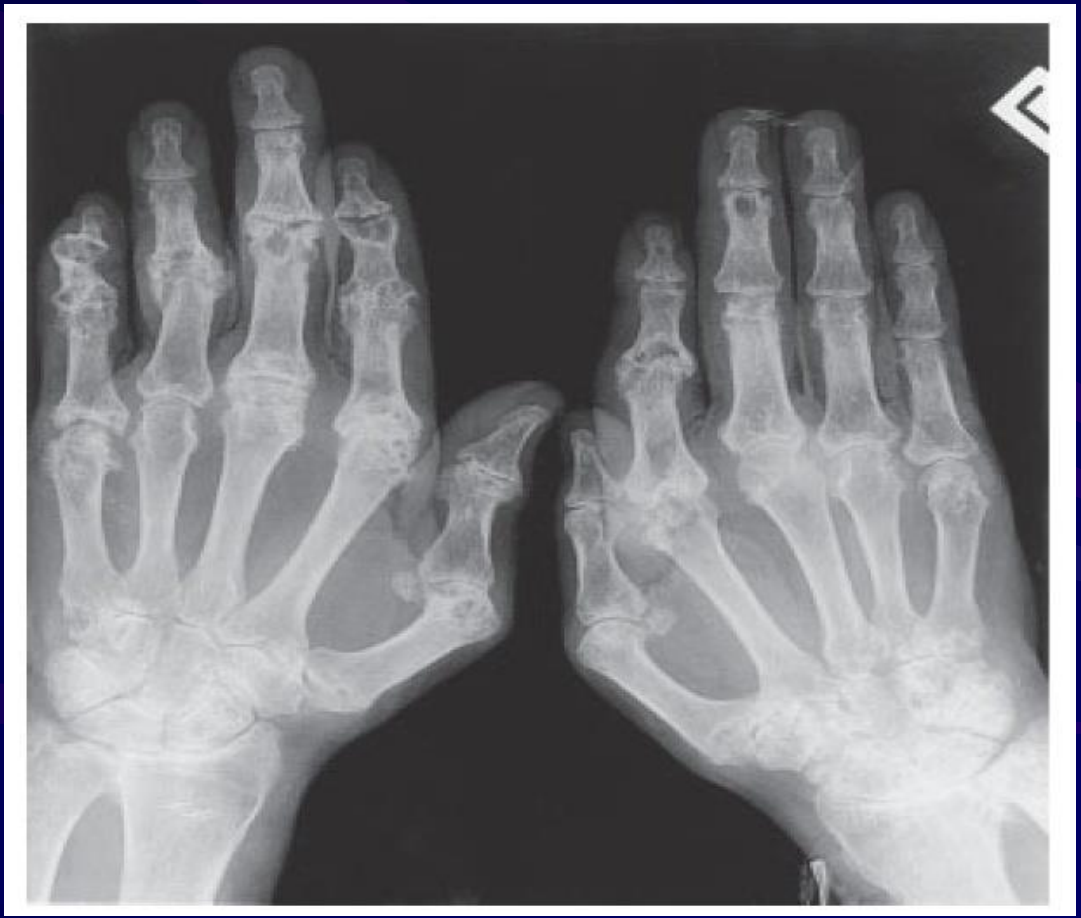


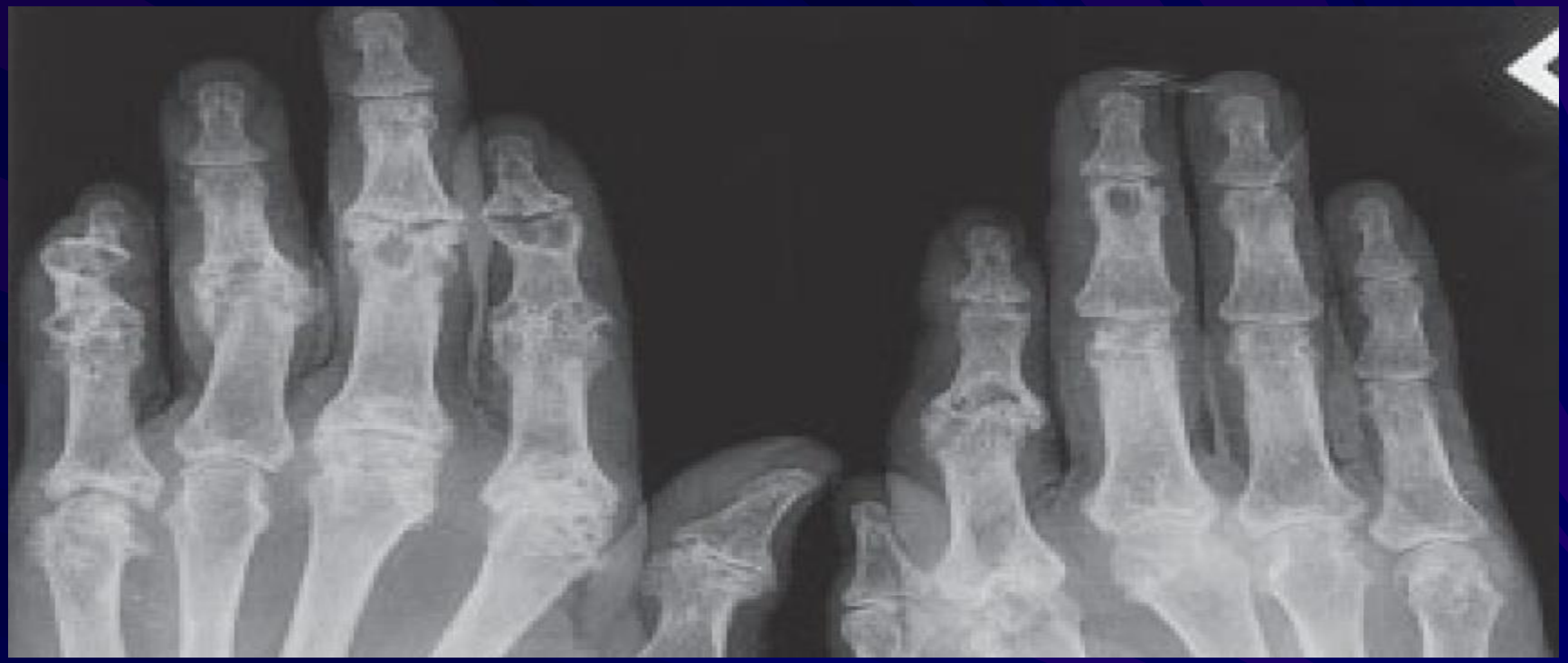


Arthritis & Rheumatism
Volume 60, Issue 6, pages 1850-1850, 28 MAY 2009 DOI: 10.1002/art.24528
http://onlinelibrary.wiley.com/doi/10.1002/art.24528/full#ill1

Polyarticular Gout OR RA

The patient, a 68-year-old man with a 30-year history of seronegative yet nodular rheumatoid arthritis, presented for reevaluation.





Hand radiography (**left**) revealed soft tissue swelling and extensive joint destruction with erosions. However, because most of the erosions were "punched out" or overhanging, and because there was involvement of some of the distal interphalangeal joints and no evidence of osteopenia, the overall findings were more suggestive of gout. Aspirated material from a subcutaneous nodule (**right**) revealed colored broad plates of cholesterol crystals, which are usually nondiagnostic and which commonly complicate joint or bursa effusions of diverse etiologies. However, the presence of admixed needle-shaped negative birefringent crystals of monosodium urate monohydrate confirmed the diagnosis of gout in this patient.

Pseudo-rheumatoid gout

- Involvement of some of the distal interphalangeal joints
- Lack of osteopenia, over hanging margins and the overall findings were more suggestive of gout
- Aspirated material from a subcutaneous nodule revealed colored broad plates of cholesterol crystals
- However, the presence of admixed needle-shaped negative birefringent crystals of monosodium urate monohydrate confirmed the diagnosis of gout in this patient

Co morbidities

- DM
- HTN
- Metabolic syndrome
- Obesity
- CVD
- CRF
- Alcohol
- Medications

Iatrogenic , Diuretic induced Gout

- Diuretic use, increased serum urate levels, and risk of incident gout in a population-based study of adults with hypertension:
- 5,789 pts hypertension; 37% were treated with a diuretic
- Use of any diuretic **(HR 1.48 [95% CI 1.11, 1.98])**
- Thiazide diuretic **(HR 1.44 [95% CI 1.00, 2.10])**
- Loop diuretic **(HR 2.31 [95% CI 1.36, 3.91])** was associated with incident gout
- ***Use of antihypertensive medication other than diuretic agents*** was associated with decreased gout risk (adjusted HR 0.64 [95% CI 0.49, 0.86]) compared to untreated hypertension

Arthritis & Rheumatism, Volume 64, Issue 1, pages 121–129, January 2012

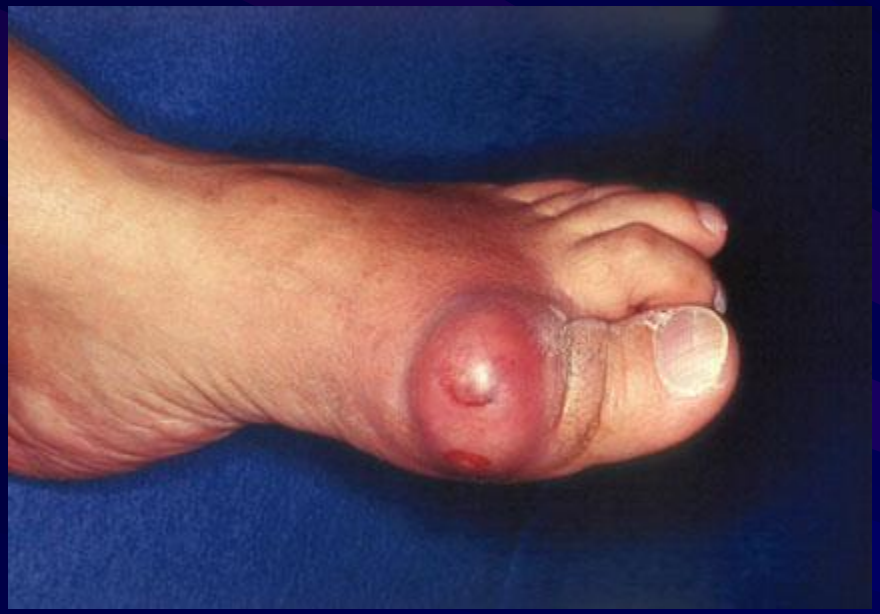
Ask patient if he is having a gout flare

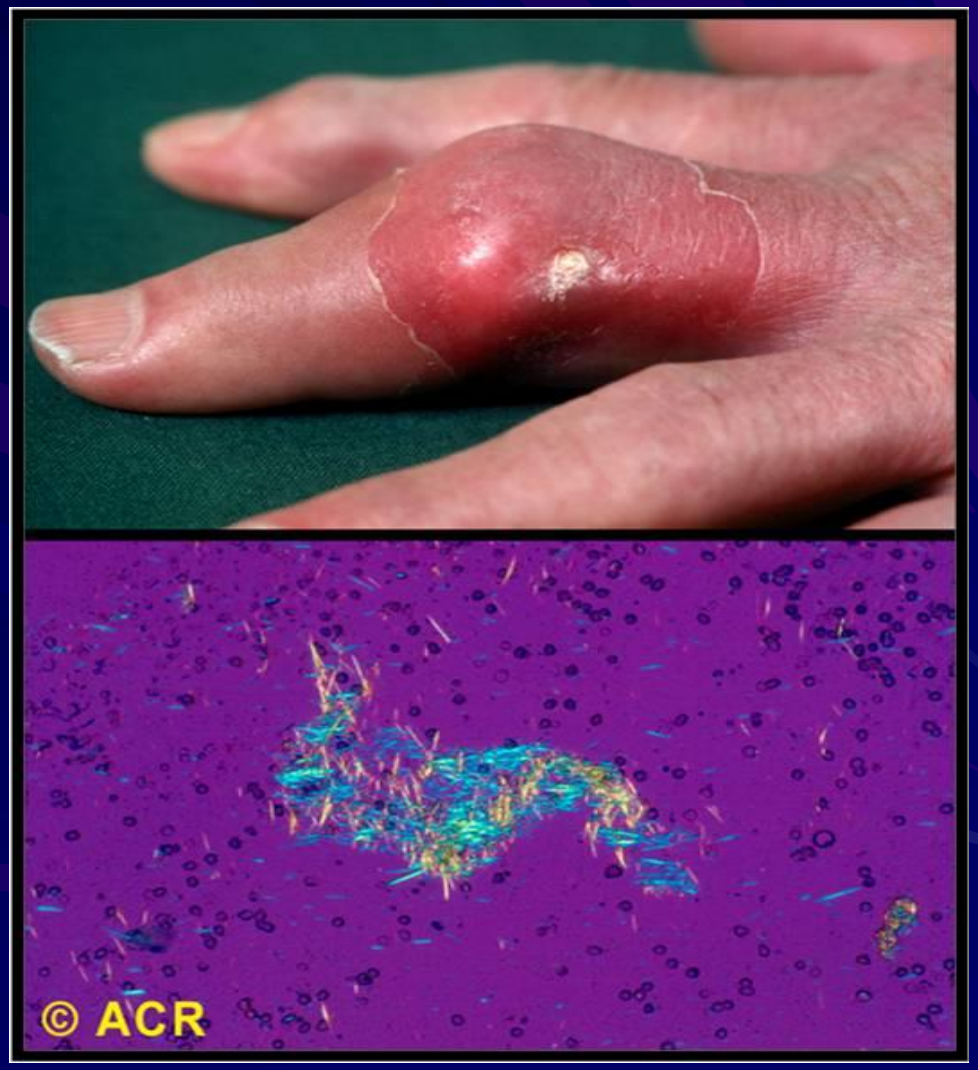
- ***Developing a provisional definition of a flare in patients with established gout†***
- Patient-reported flare is as good as study gold standard
- Joint pain at rest
- Presence of warm joints
- Swollen joints

were most strongly associated with presence of a gout flare

Arthritis & Rheumatism
Accepted Article (Accepted, unedited articles published online for future issues)

ACUTE FLARE OF TOPHUS





Can Gout affect women

Epidemiology of gout in women: Fifty-two–year follow-up of a prospective cohort

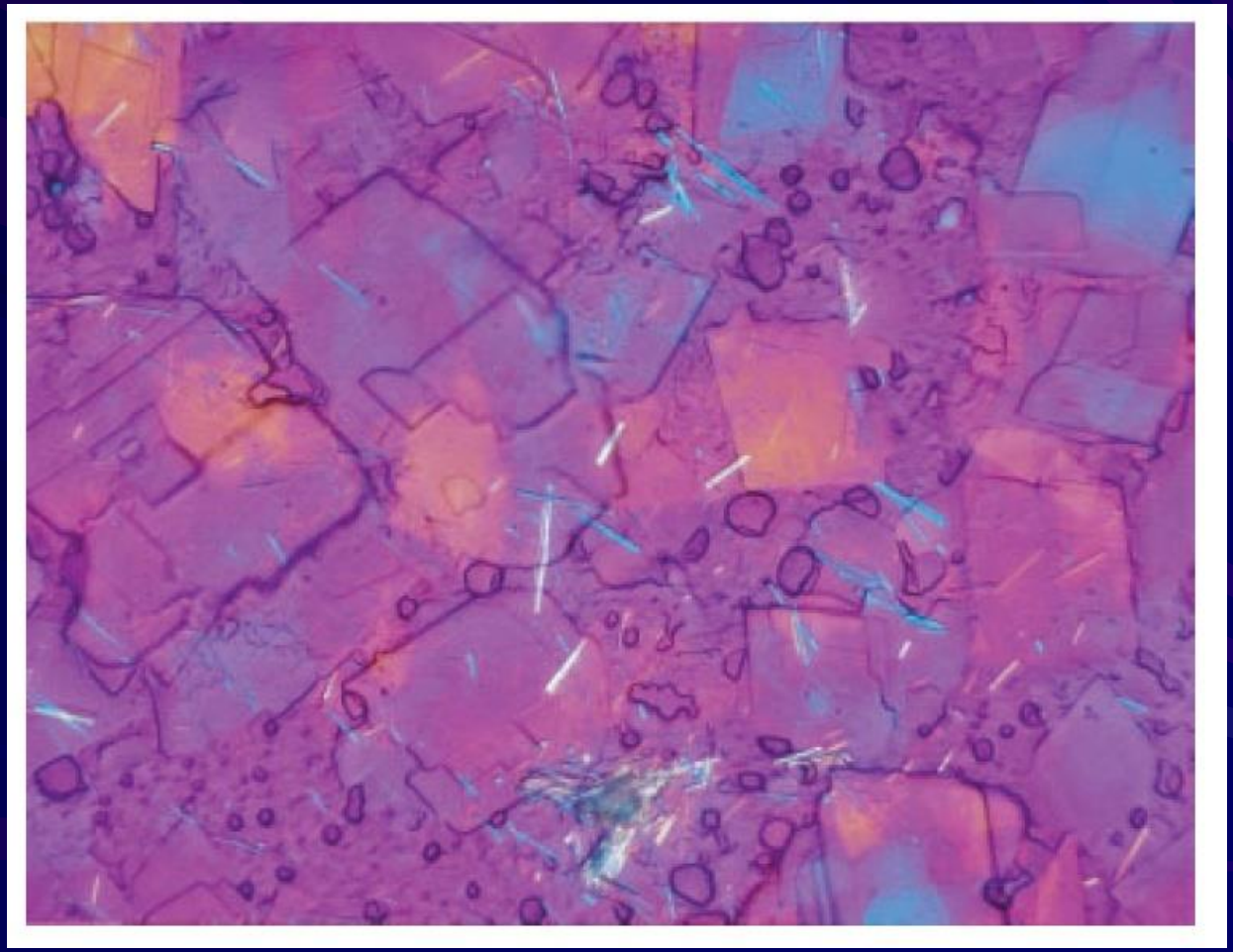
- Higher levels of serum uric acid increase the risk of gout in a graded manner among women, but the rate of increase is lower than that among men
- Risk factors
  - Increasing age
  - Obesity
  - Alcohol consumption
  - Hypertension
  - Diuretic use

Arthritis & Rheumatism
Volume 62, Issue 4, pages 1069–1076, April 2010

Diagnosis

- Aspiration always recommended if possible
- Prompt polarized microscopic analysis
- Definitive diagnosis– requires crystal confirmation
- Gout and Sepsis can coexist – fluid should be sent Gram's stain, culture
- Serum uric acid levels neither confirm nor exclude gout

MSU CRYSTALS

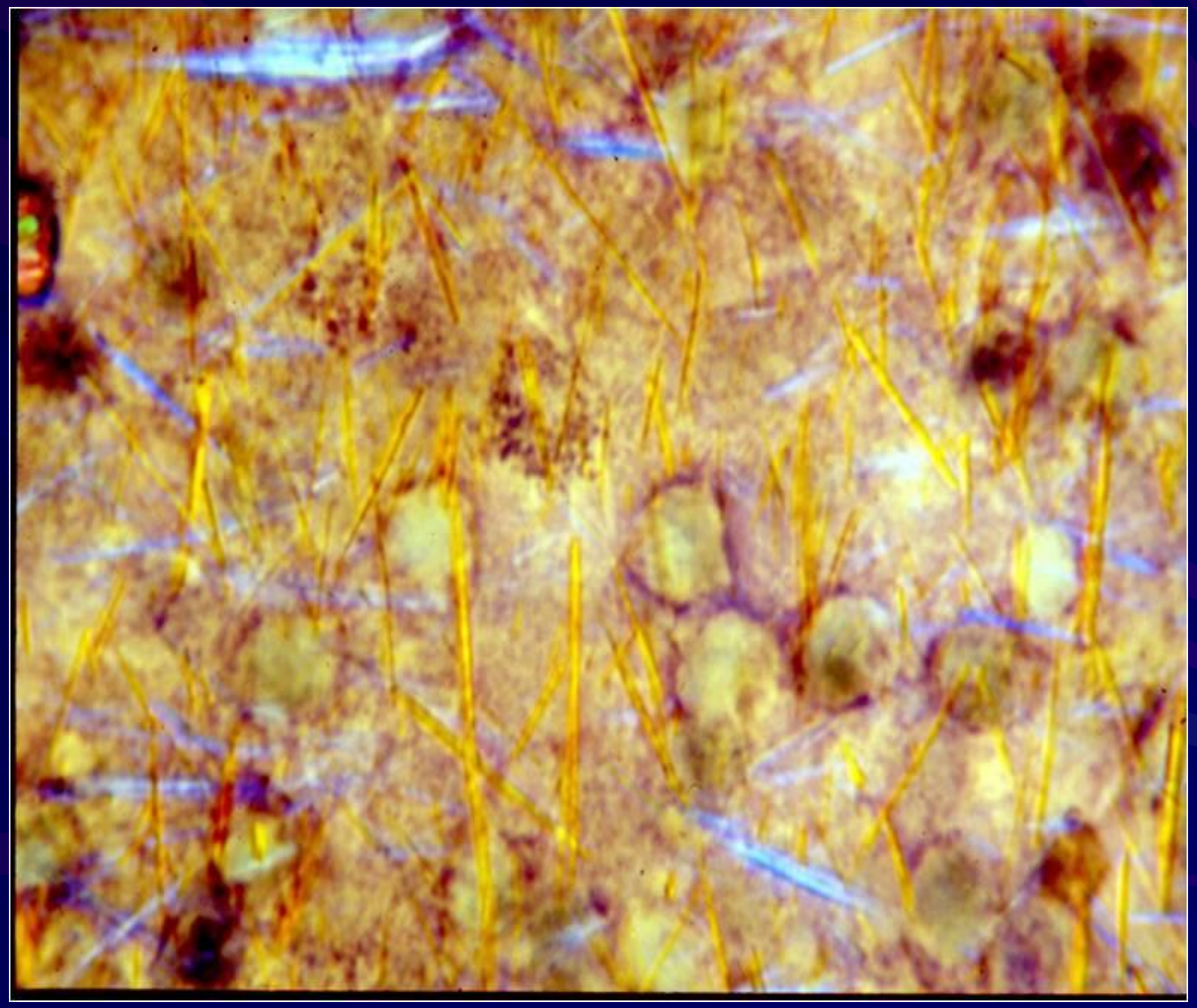


Gout Can Coexist With Septic Arthritis

WHO IS AT RISK?

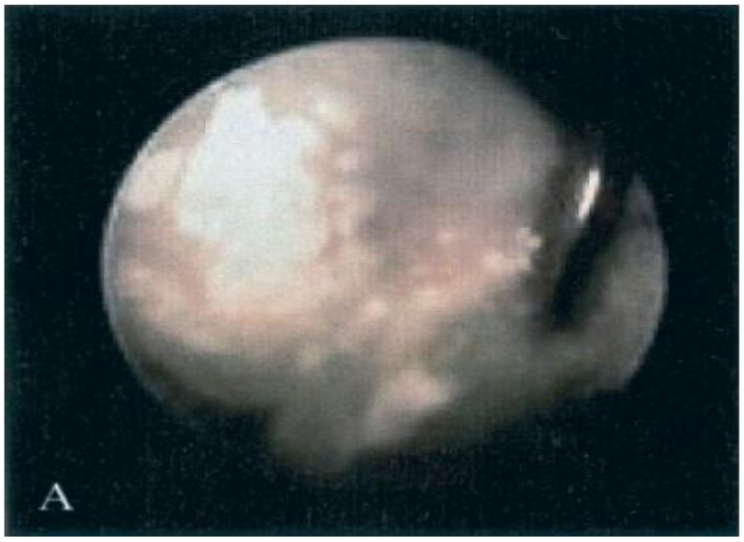
- Diabetic
- Alcoholic
- Old age

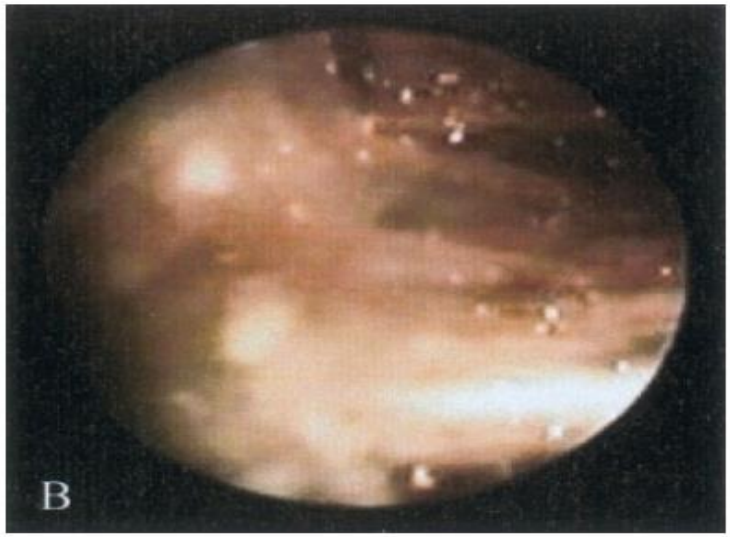
MSU AND STAPH



PREDICT AN ATTACK

The patient, a 63-year-old man with a history of chronic gout, which was in remission at the time, took a 2-hour plane ride in economy class and developed acute gout in his right knee. One week later, he presented with contracture of the knee and diminished extension. Arthroscopy (A) revealed an easily detachable chalky deposit on the surface of the hyaline cartilage. Adhesions in the suprapatellar pouch, a "starry sky" appearance, and hemorrhagic areas (B) were also observed. These findings suggest that immobility of the knees in a sitting position might lead to clinical flare of gout.





TREATMENT OF ACUTE ATTACK

- THERAPY (for all crystal diseases):
  - Corticosteroids: intrarticular > systemic
  - NSAIDs – fast acting full dose if no contraindications
  - Colchicine (PO, NOT IV route ***dangerous***)
    - narrow therapeutic window
      - Bone marrow suppression, myopathy, neuropathy
    - purgative effects – "Pt often run before they walk"
  - ***NEVER ALLOPURINOL***

LOW VS HIGH COLCHICINE

High Versus Low Dosing of Oral Colchicine for Early Acute Gout Flare

Twenty-Four–Hour Outcome of the First Multicenter, Randomized, Double-Blind, Placebo-Controlled, Parallel-Group, Dose-Comparison Colchicine Study

Robert A. Terkeltaub,[1] Daniel E. Furst,[2] Katherine Bennett,[3] Karin A. Kook,[3] R. S. Crockett,[4] and Matthew W. Davis[5]

Methods. **This multicenter, randomized, double-blind, placebo-controlled, parallel-group study compared self-administered low-dose colchicine (1.8 mg total over 1 hour) and high-dose colchicine (4.8 mg total over 6 hours) with placebo. The primary end point was ≥50% pain reduction at 24 hours without rescue medication.**

LESS IS MORE

Table 2. Efficacy analysis (intent-to-treat population, n = 184)*

	Colchicine dose			High-dose colchicine vs. placebo		Low-dose colchicine vs. placebo	
	High (n = 52)	Low (n = 74)	Placebo (n = 58)	OR (95% CI)	*P*	OR (95% CI)	*P*
Primary end point							
Treatment response based on target joint pain score 24 hours after the first dose	17 (32.7)	28 (37.8)	9 (15.5)	2.64 (1.06–6.62)	0.034	3.31 (1.41–7.77)	0.005
Alternate definition of response							
Treatment response based on target joint pain score 32 hours after the first dose	19 (36.5)	31 (41.9)	10 (17.2)	2.76 (1.14–6.69)	0.022	3.46 (1.52–7.88)	0.002
Treatment response based on at least a 2-unit reduction in target joint pain score 24 hours after the first dose	18 (34.6)	32 (43.2)	10 (17.2)	2.54 (1.04–6.18)	0.037	3.66 (1.61–8.32)	0.002
Treatment response based on at least a 2-unit reduction in target joint pain score 32 hours after the first dose	20 (38.5)	34 (45.9)	10 (17.2)	3.00 (1.24–7.24)	0.012	4.08 (1.80–9.27)	0.001

MEDS- Increased Urate Pool

- Diuretics (RR 1.77, CI 1.4-2.2)
- Low dose Aspirin
- B-blockers
- PZA
- Ethambutol
- Cyclosporin
- Tacrolimus
- Insulin

Diet for Gout

Purine-containing foods to be avoided include

- Beer, other alcoholic beverages.
- Anchovies, sardines in oil, fish roes, herring.
- Yeast.
- Organ meat (liver, kidneys, sweetbreads)
- Meat extracts, consomme, gravies.

Foods which are very high in purines include:

- hearts
- herring
- mussels
- yeast
- smelt
- sardines
- sweetbreads

Treat to target : Gout

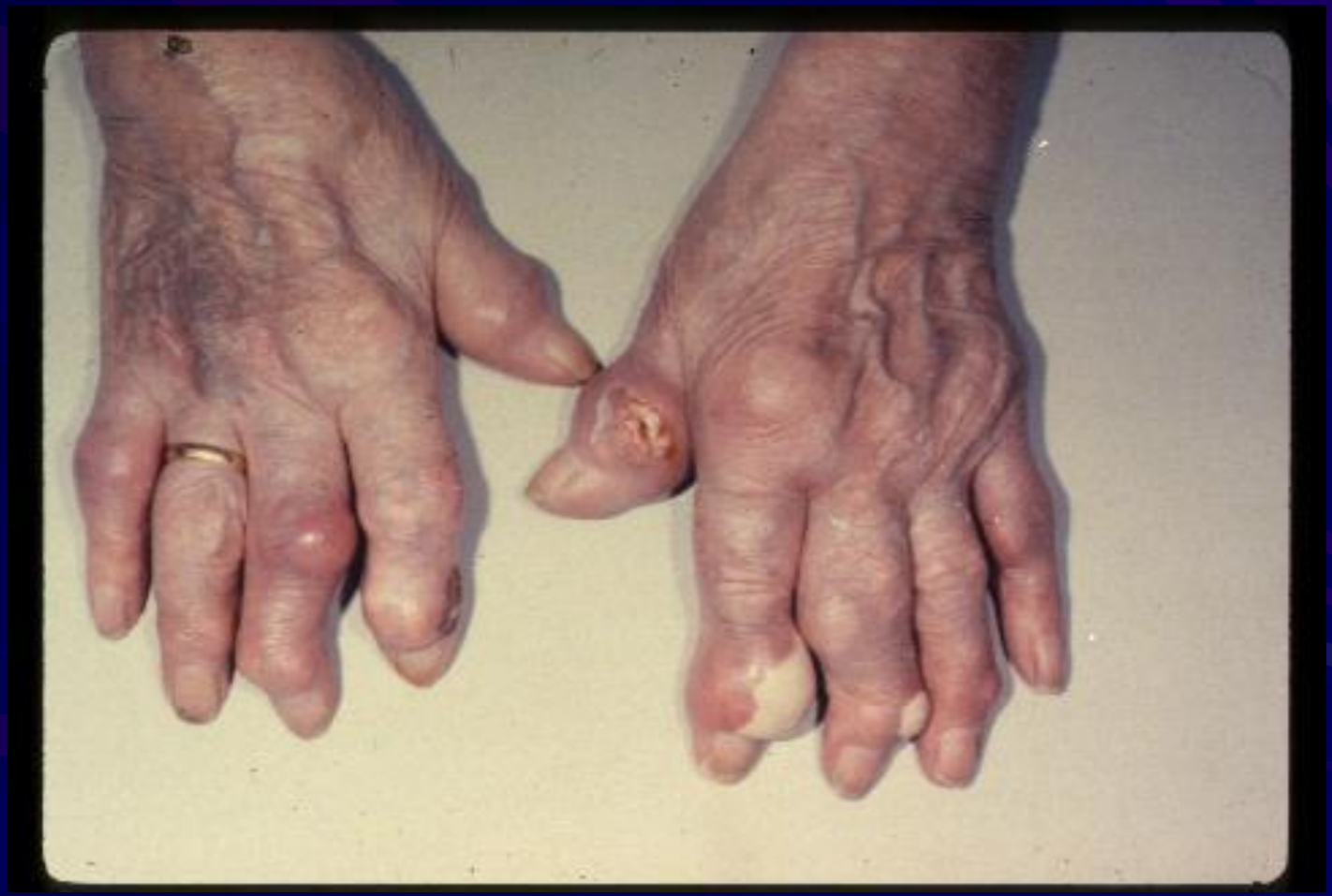
- ***A two-stage approach to the treatment of hyperuricemia in gout: The "dirty dish" hypothesis***
- Maintaining serum urate levels at less than 6 mg/dL is necessary for clearing tophi and dissolving monosodium urate monohydrate crystals in gout
- Once target has been achieved, keeping serum urate just below the threshold for saturation (6.0 - 6.9 mg/dL) is likely to be enough to prevent gout recurrence

Arthritis & Rheumatism
Volume 63, Issue 12, pages 4002–4006, December 2011

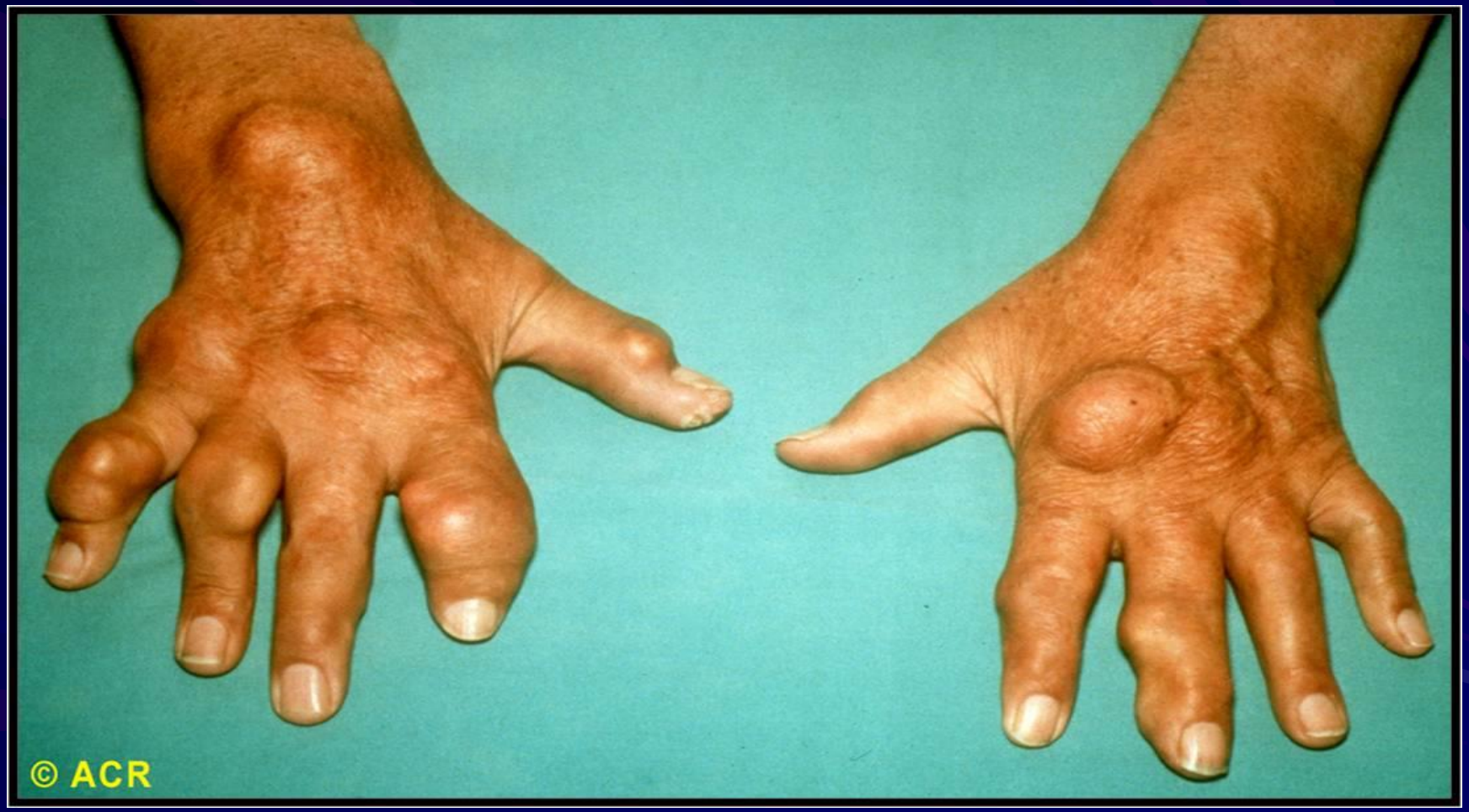
Hyperuricemia and xanthine oxidase - toxic to the vasculature?

- Uric acid : Antioxidant or Pro oxidant.
- Xanthine Oxidase induces oxidative stress
- Uric acid and XOD may promote inflammation
- Promote endothelial dysfunction and vascular pathologies
- Epidemiologic evidence supports the hypothesis; ***hyperuricemia is an independent risk factor for certain vascular diseases and complications of atherosclerosis.***
- Intrinsic inflammation and oxidative stress in ***gout, CHF, metabolic syndrome, diabetes, and chronic kidney disease***, are likely major determinants of outcomes

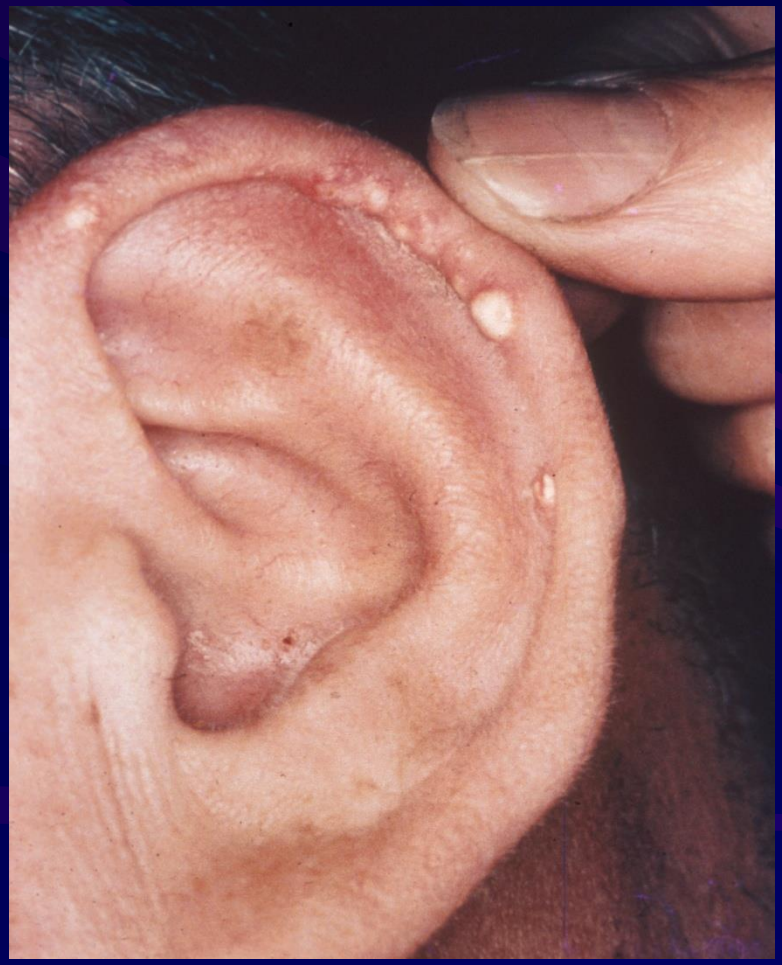
Tophaceous Gout



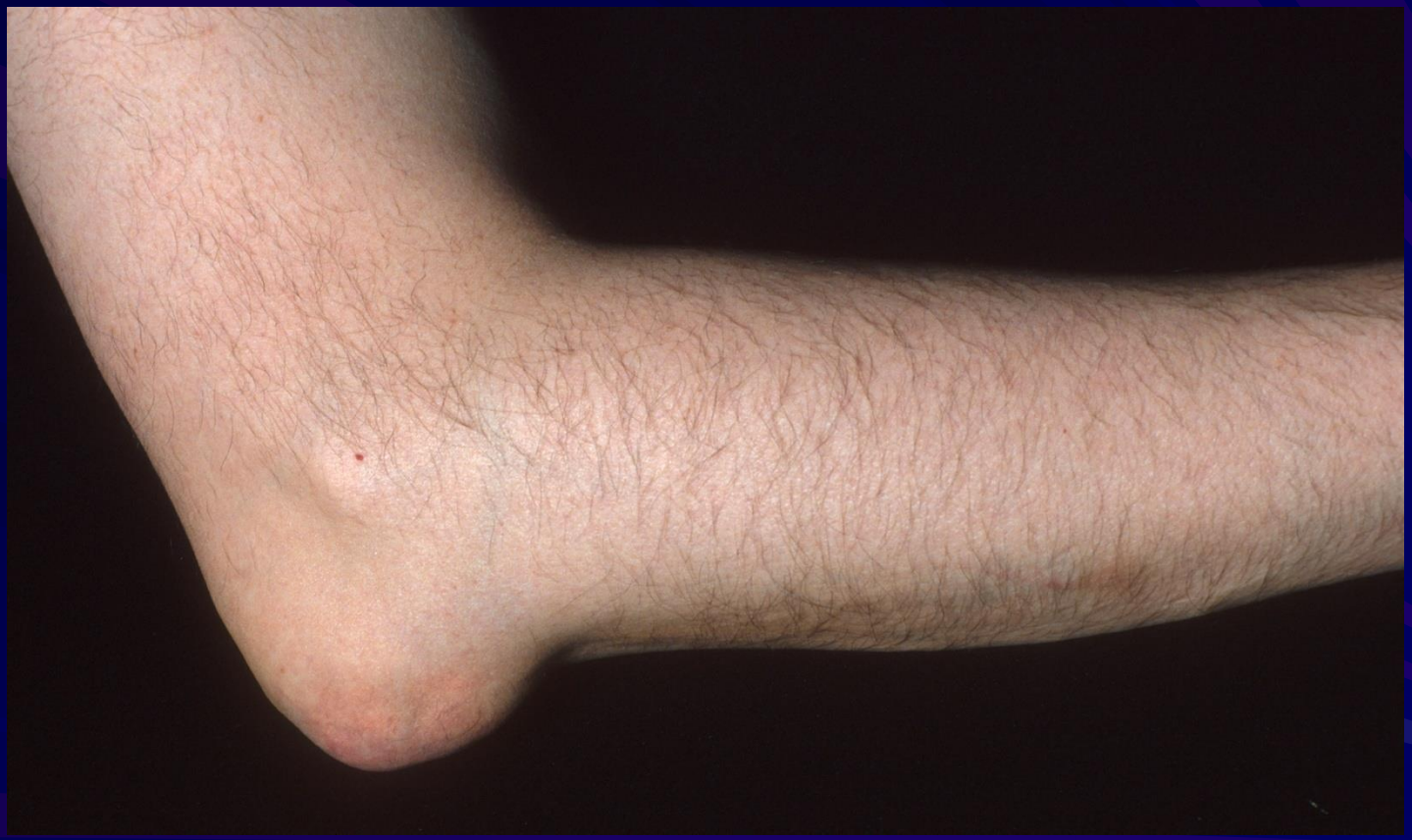
BULKI TOPHI



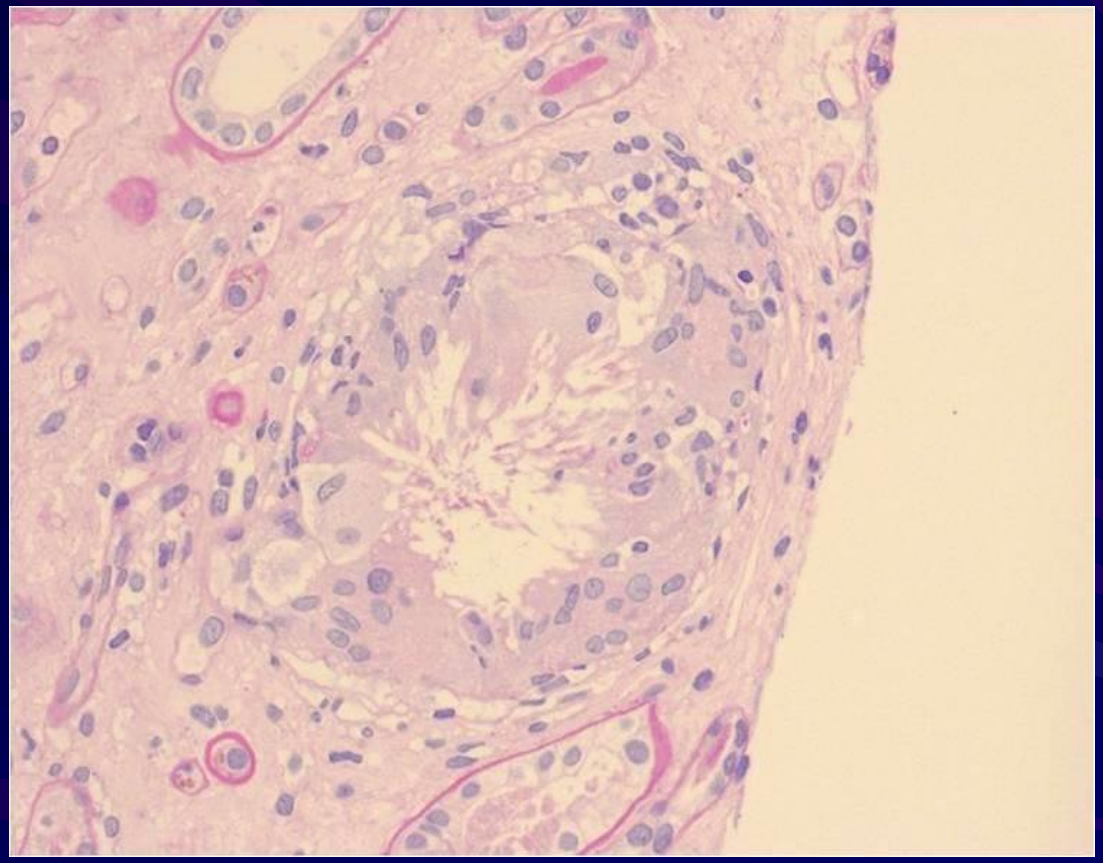
EAR TOPHI



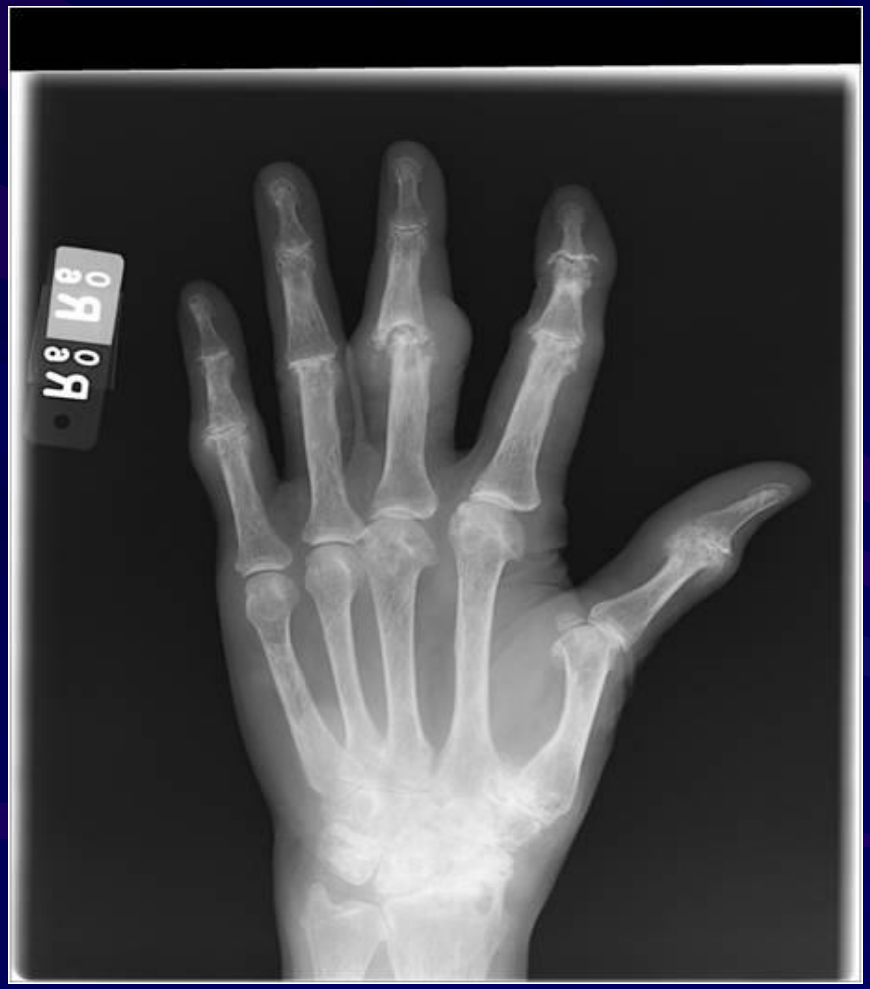
OLECRNON BURSITIS



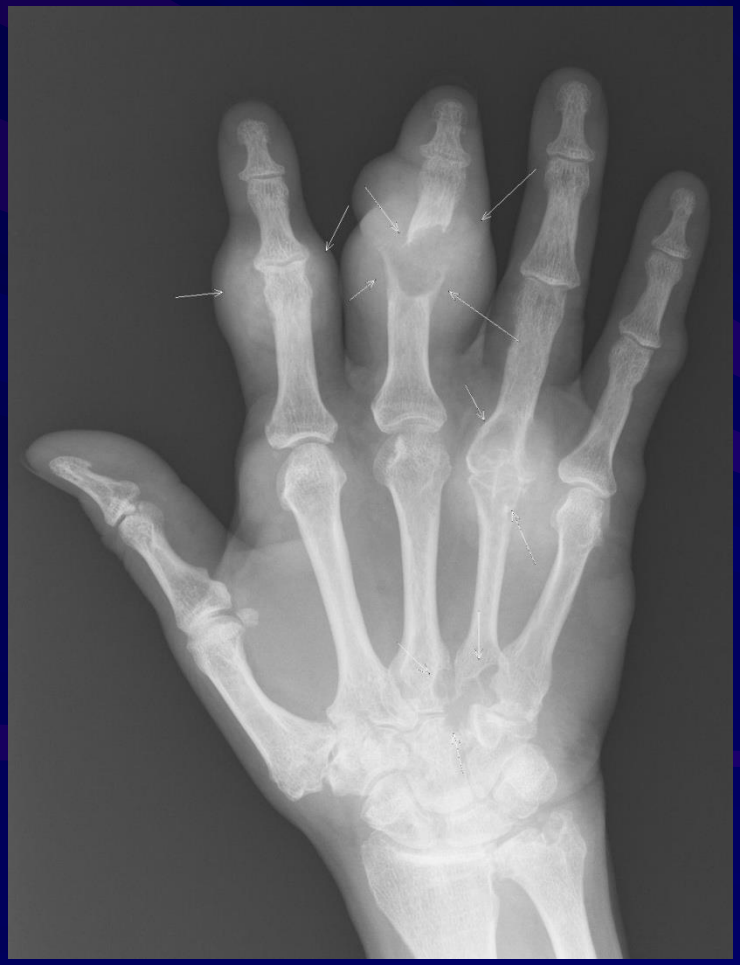
SYNVIAL TISSUE DEPOSITS



Erosions with overhanging margins



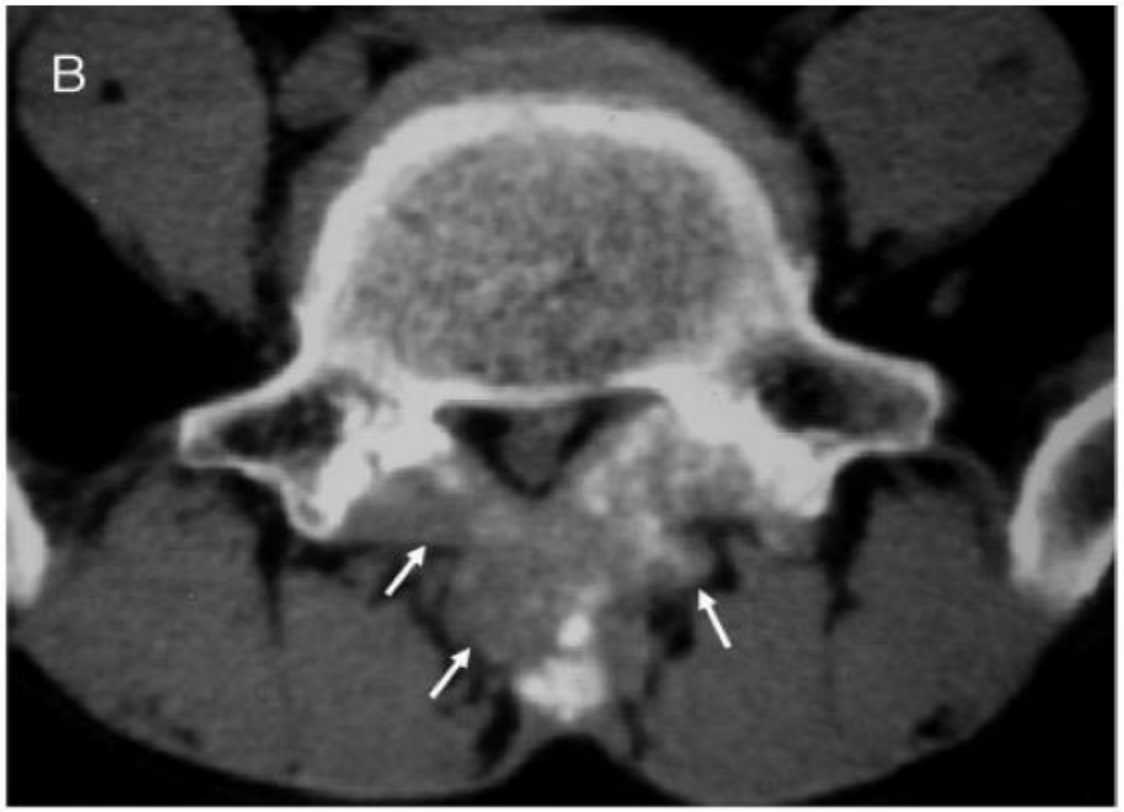
Punched out erosions



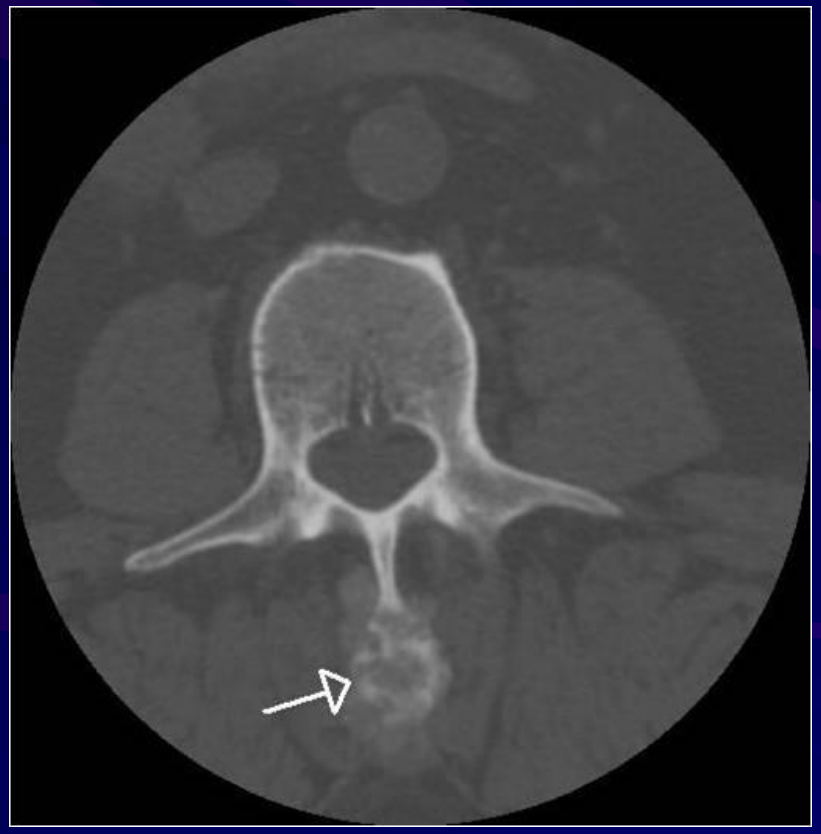
Extra articular complications

Spinal Gouty Tophus

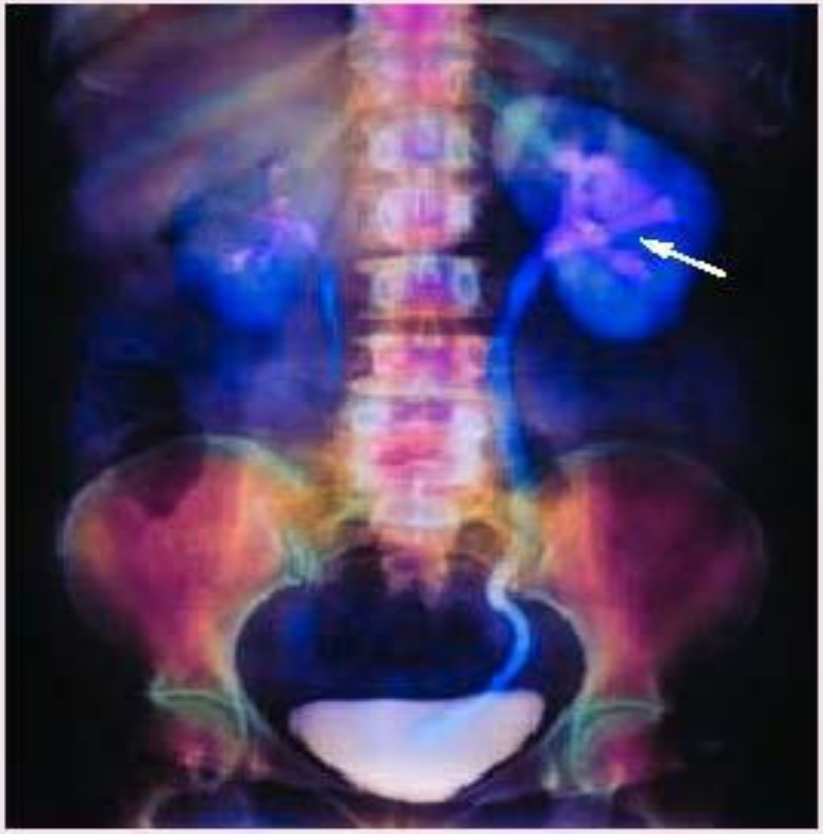
The patient, a 69-year-old man with a 10-year history of peripheral gouty arthritis, presented with severe back pain. The biochemical profile was normal except for an elevated level of uric acid (10.8 mg/dl). Radiography of the lumbar spine (A) showed



CT spine showing tophus causing destruction of spinous process



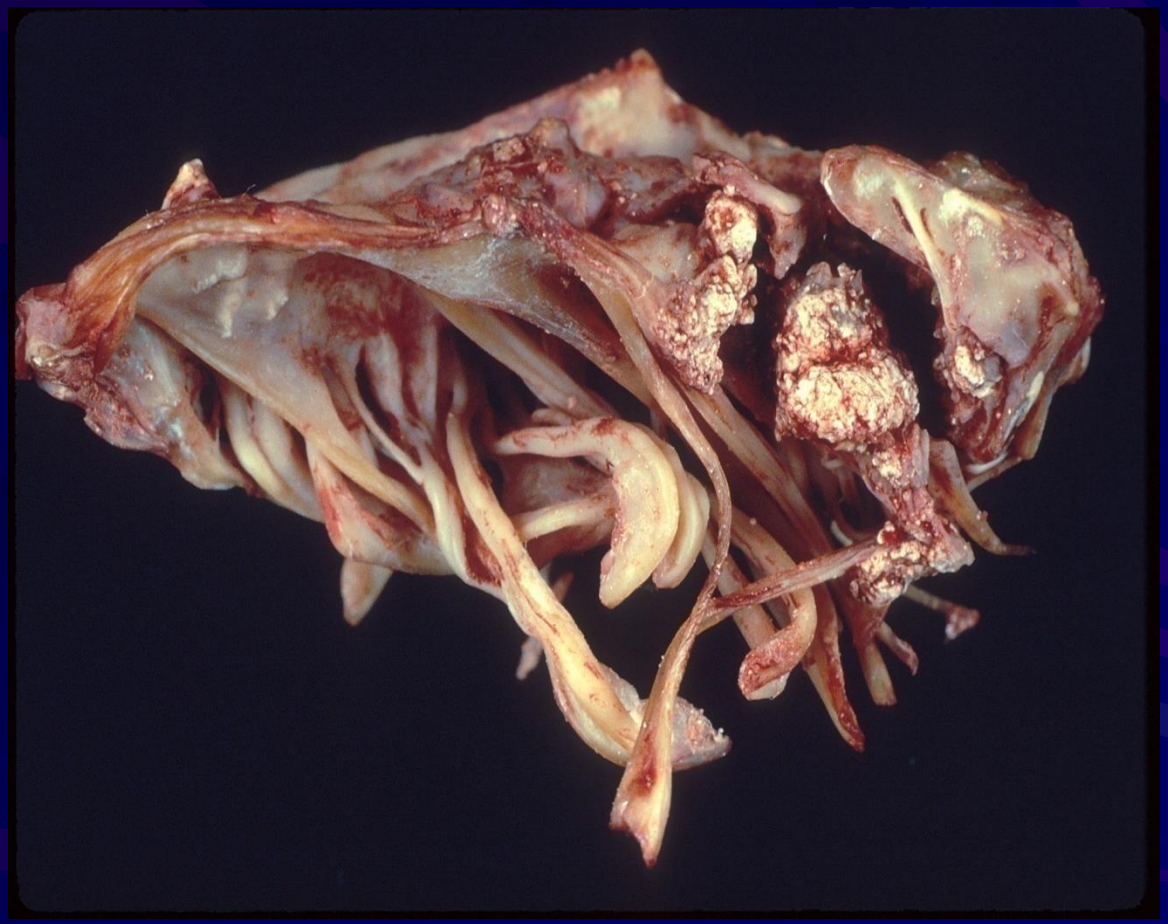
Urate nephropathy



This color-enhanced urogram shows an enlarged renal pelvis (arrow) and ipsilateral ureter obstruction due to a kidney stone at the base of the ureter.

IMAGE SOURCE: PHOTO RESEARCHERS INC.

Cardiac tissue deposits



Treatment of acute and chronic gout

Acute Attack

- Colchicine
- Nsaids
- Prednisone
- Intra articular steroids

Uric Acid Lowering Therapy

- Lifestyle, dietary modification
- Diet high in vegetables, dairy, water beneficial
- Initiate uric acid lowering therapy after 1(?) or 2 episodes of acute gouty arthritis
- Always prophylaxis for first 6 months with low dose steroids, NSAIDs, or Colchicine

Uricostatic Drugs

- Allopurinol - developed 1957
- NEVER USED IN ACUTE ATTACK
  - Reduce annual gout attacks 4.4 to .06 / yr
  - Gradual resolution of tophi w/ uric acid < 6
  - Titrate dose up to 600 mg /day

Concerns associated with Allopurninol

- Increased toxicity with CRI
- Allopurinol hypersensitivity rxn –rare but can be fatal
- Densensitization can be useful for mild SEs
  - Oxypurinol is an option but 50% intolerance
- Multiple interactions –
  - Imuran, 6MP, Warfarin, theophylline, ampiciliin, diuretics
- Treatment is lifelong

Allopurinol hypersensitivity syndrome

- **Starting dose is a risk factor for allopurinol hypersensitivity syndrome: A proposed safe *starting* dose of allopurinol‡‡**

- Allopurinol hypersensitivity syndrome (AHS) is a rare but potentially fatal adverse event.
- Dosing guidelines based on creatinine clearance (CrCL) have been proposed that doses ≥300mg/d may be associated with AHS, particularly in patients with renal impairment
- The aim of this study was to determine the relationship between allopurinol dosing and AHS.
- A retrospective case-control study of AHS between January 1998 and September 2010 was undertaken.

Allopurinol hypersensitivity syndrome

- Fifty-four AHS cases and 157 controls were identified.
- There was an increase in risk of AHS as the starting dose of allopurinol corrected for eGFR increased.
- For the highest quintile of starting dose≥eGFR, the odds ratio was 23.2 ($p<0.01$).
- ROC analysis indicated that 91% of AHS cases and 36% of controls started on a dose of allopurinol **at ≥1.5mg allopurinol per unit eGFR (mg/ml/min).**
- **Conclusions:**
- Starting allopurinol at a dose of 1.5mg per unit eGFR may be associated with a reduced risk of AHS. In patients who tolerate allopurinol the dose can be gradually increased to achieve the target SU

Arthritis & Rheumatism
Accepted Article (Accepted, unedited articles published online for future issues)

Febuxostat

- Febuxostat was approved by the U.S. Food and Drug Administration on February 16, 2009
- Urate lowering drug, an inhibitor of xanthine oxidase
- Xanthine oxidase is needed to successively oxidize both hypoxanthine and xanthine to uric acid.
- Febuxostat inhibits xanthine oxidase, therefore reducing production of uric acid
- Febuxostat inhibits both, oxidized and reduced form of xanthine oxidase because of which febuxostat cannot be easily displaced

New kid on the block (not so new)

Arthritis & Rheumatism (Arthritis Care & Research)
Vol. 59, No. 11, November 15, 2008, pp 1540–1548
DOI 10.1002/art.24209

ORIGINAL ARTICLE

Effects of Febuxostat Versus Allopurinol and Placebo in Reducing Serum Urate in Subjects With Hyperuricemia and Gout: A 28-Week, Phase III, Randomized, Double-Blind, Parallel-Group Trial

H. RALPH SCHUMACHER, JR.,[1] MICHAEL A. BECKER,[2] ROBERT L. WORTMANN,[3] PATRICIA A. MacDONALD,[4] BARBARA HUNT,[4] JANET STREIT,[4] CHRISTOPHER LADEMACHER,[4] AND NANCY JOSEPH-RIDGE[4]

RESULTS

Table 2. Proportion of subjects with serum urate levels <6.0 mg/dl at week 28 and final visits (intent-to-treat population)

	Week 28, %	Final, %
Placebo	1 (1/99)	1 (1/127)
Febuxostat 80 mg	76 (122/161)*	72 (183/253)*
Febuxostat 120 mg	87 (163/188)†	79 (209/265)*
Febuxostat 240 mg	94 (78/83)†	92 (116/126)‡
Allopurinol 300 mg§	41 (85/208)¶	39 (102/263)¶

* Statistically significant versus placebo ($P \leq 0.05$) and versus allopurinol ($P \leq 0.05$).
† Statistically significant versus placebo ($P \leq 0.05$), versus allopurinol ($P \leq 0.05$), and versus febuxostat 80 mg ($P \leq 0.05$).
‡ Statistically significant versus placebo ($P \leq 0.05$), versus allopurinol ($P \leq 0.05$), versus febuxostat 80 mg ($P \leq 0.05$), and versus febuxostat 120 mg ($P \leq 0.05$).
§ Ten subjects received 100 mg and 258 subjects received 300 mg of allopurinol based on renal function.
¶ Statistically significant versus placebo ($P \leq 0.05$).

CONCLUSION

DISCUSSION

The APEX trial is the largest randomized controlled clinical trial to date comparing febuxostat, allopurinol, and placebo in hyperuricemic subjects with gout. Employing a rigorous primary end point of last 3 monthly serum urate levels <6.0 mg/dl, this 28-week study demonstrated that treatment with febuxostat significantly reduced and maintained serum urate levels <6.0 mg/dl in the majority of subjects, and even in many subjects with a baseline serum urate level >10.0 mg/dl. In contrast, the proportion of subjects responding to allopurinol (22%) was lower than anticipated based on a review of the literature (35–38), but was consistent with the results of a previously reported clinical trial (FACT) (10). Reasons for the limited efficacy

Newer and stronger drugs

- URICASE – converts uric acid to allantoin
  - Recombinant uric acid oxidase – RASURICASE
    - parenteral route – can be given only once due to antibody production
    - Black box warning – anaphylaxis, hemolysis, methemoglobinemia
  - Pegylated preparation approved for urate nephropathy in tumor lysis syndrome.
    - Expensive
    - Sq administration
- PEGOLITICASE (Krsytexxa)

  Now approved

PEGLOTICASE

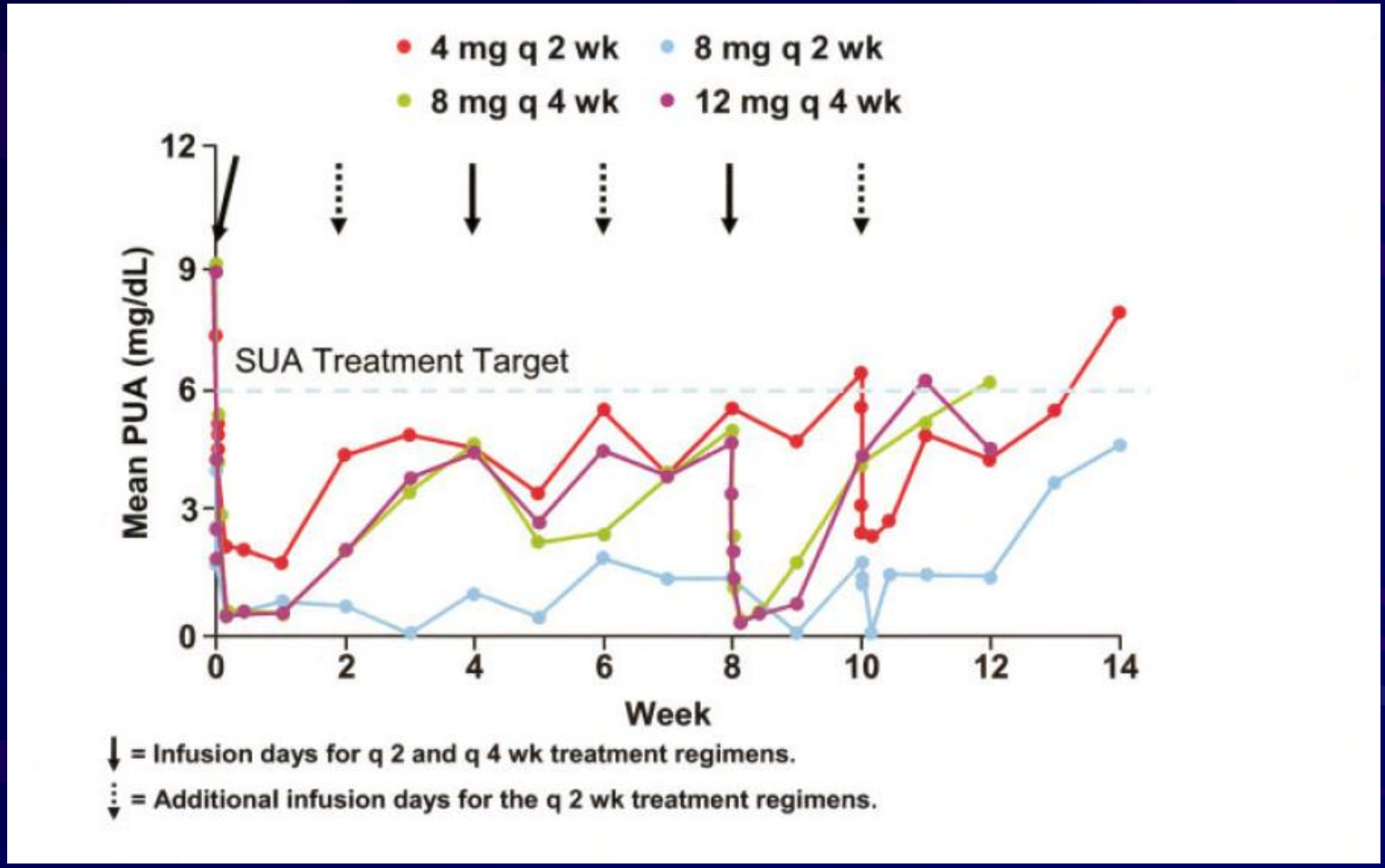
ARTHRITIS & RHEUMATISM
Vol. 58, No. 9, September 2008, pp 2882–2891
DOI 10.1002/art.23810

Reduction of Plasma Urate Levels Following Treatment With Multiple Doses of Pegloticase (Polyethylene Glycol–Conjugated Uricase) in Patients With Treatment-Failure Gout

Results of a Phase II Randomized Study

Methods. **Forty-one patients were randomized to undergo 12–14 weeks of treatment with pegloticase at 1 of 4 dosage levels: 4 mg every 2 weeks, 8 mg every 2 weeks, 8 mg every 4 weeks, or 12 mg every 4 weeks. Plasma uricase activity, plasma urate, and antipegloticase antibodies were measured, pharmacokinetic parameters were assessed, and adverse events were recorded.**

DOSE S AND RESPONSE



NEW TREATMENT ON HORIZON

Research article

Open Access

A pilot study of IL-1 inhibition by anakinra in acute gout

Alexander So[1], Thibaut De Smedt[2], Sylvie Revaz[1] and Jürg Tschopp[3]

[1]Service of Rhumatologie, Department of Medicine, Centre Hospitalier Universtaire Vaudois and University of Lausanne, 1011 Lausanne, Switzerland
[2]Apoxis SA, Avenue de Sévelin 18-20, 1004 Lausanne, Switzerland
[3]Institute of Biochemistry, University of Lausanne, chemin de Boveresses 155, 1066 Epalinges, Switzerland

Corresponding author: Alexander So, AlexanderKai-Lik.So@chuv.ch

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Abstract

Monosodium urate crystals stimulate monocytes and macrophages to release IL-1β through the NALP3 component of the inflammasome. The effectiveness of IL-1 inhibition in hereditary autoinflammatory syndromes with mutations in the NALP3 protein suggested that IL-1 inhibition might also be effective in relieving the inflammatory manifestations of acute gout. The effectiveness of IL-1 inhibition was first evaluated in a mouse model of monosodium urate crystal-induced inflammation. IL-1 inhibition prevented peritoneal neutrophil accumulation but TNF blockade had no effect. Based on these findings, we performed a pilot, open-labeled study (trial registration number ISRCTN10862635) in 10 patients with gout who could not tolerate or had failed standard antiinflammatory therapies. All patients received 100 mg anakinra daily for 3 days. All 10 patients with acute gout responded rapidly to anakinra. No adverse effects were observed. IL-1 blockade appears to be an effective therapy for acute gouty arthritis. The clinical findings need to be confirmed in a controlled study.

Anakina (Kinret, IL-1blocker)

Table 1

Clinical summary of the 10 patients studied and their response to treatment

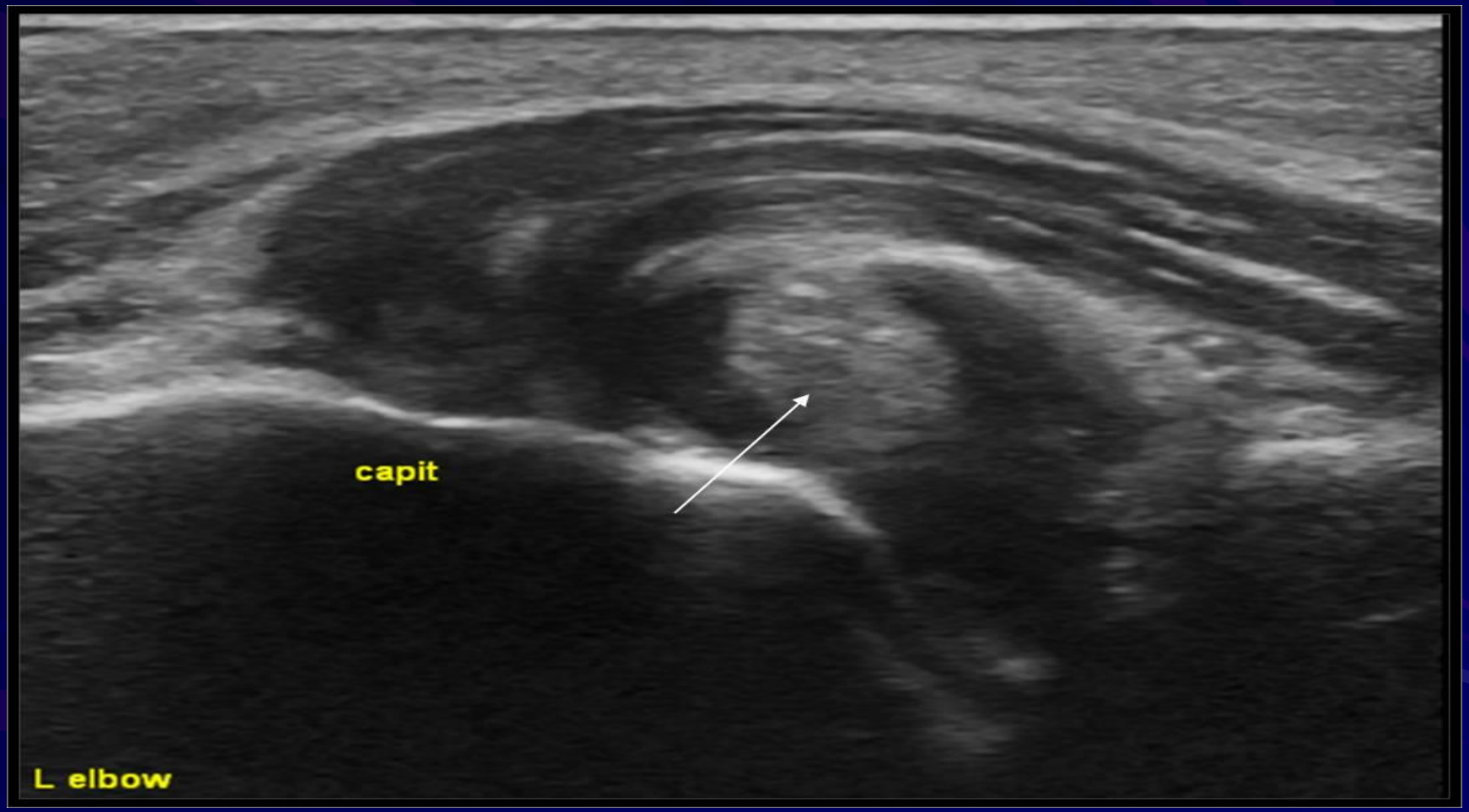
Patient	Clinical presentation	Affected joints	Serum uric acid (normal range, 160–390 µmol/l)	Serum creatinine (normal range, 44–80 µmol/l)	Hypouricemic treatment	Effect of anakinra (hours)	Patient assessment of improvement in pain (%)
Case 1 (female, 72 years old)	Chronic tophaceous gout, renal stones	Fingers, toes	637	79	Uricase	36	70
Case 2 (male, 70 years old)	Chronic tophaceous gout	Ankle, toes	564	202	Allopurinol	24	90
Case 3 (male, 72 years old)	Acute gout	Knee, ankle, foot	482	121	Allopurinol	24	90
Case 4 (male, 51 years old)	Acute gout	Ankle, toe	396	84	Allopurinol	24	100
Case 5 (male, 40 years old)	Acute gout	Ankle, toe	322	113	Allopurinol	36	100
Case 6 (female, 72 years old)	Acute gout	Feet, toe	572	72	None	36	80
Case 7 (male, 76 years old)	Acute gout	Ankle, foot	338	79	None	36	100
Case 8 (male, 70 years old)	Acute gout	Wrist, elbow, hand	779	406	None	48	50
Case 9 (male, 53 years old)	Chronic tophaceous gout	Elbow, finger, foot, ankle	660	84	Allopurinol	48	50
Case 10 (male, 38 years old)	Acute gout	Wrist, finger	540	84	None	24	60

Uricosuric agents

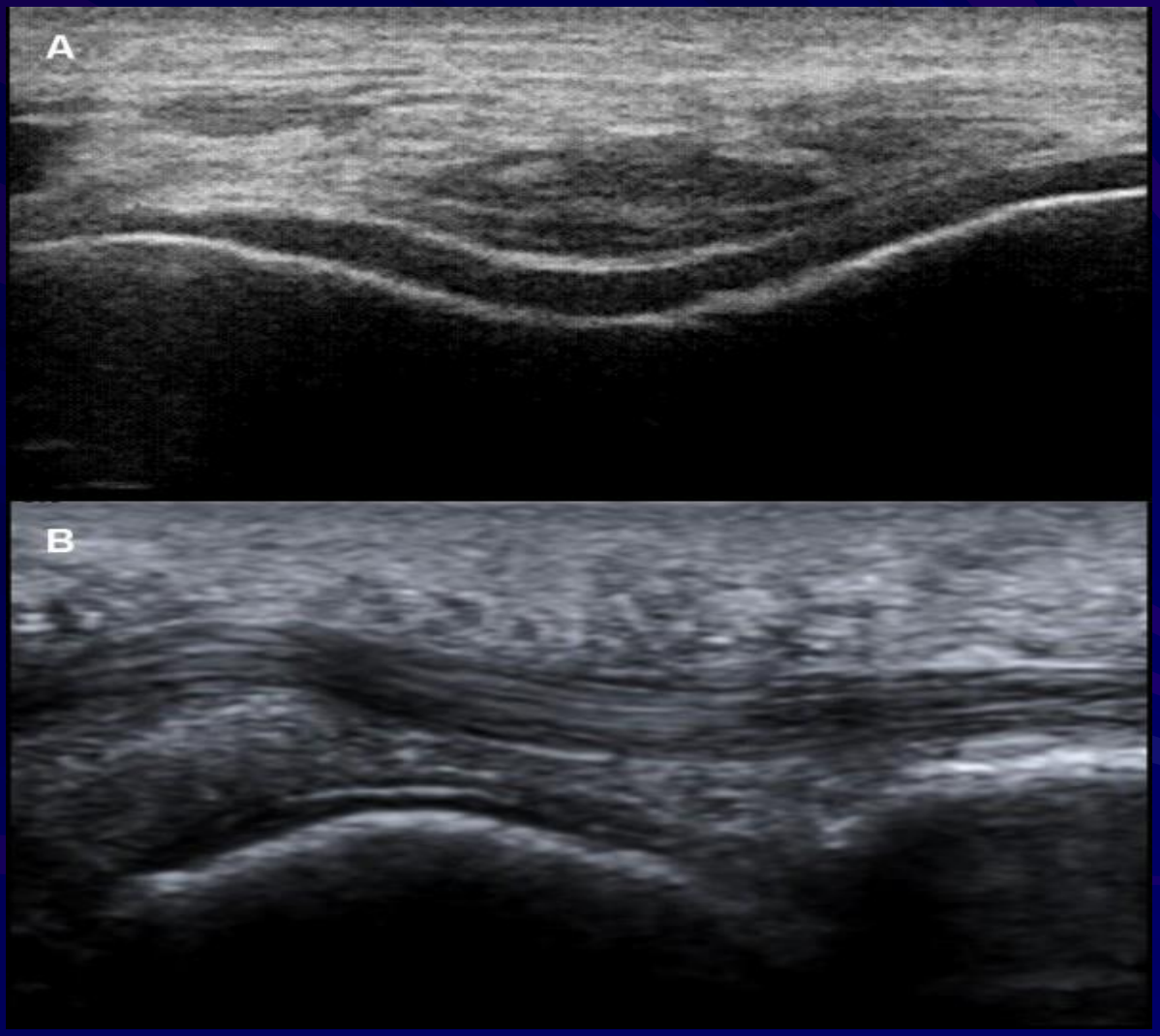
- Uricosurics
  - **probenecid 1-3 grams / day**
  - sulfinpyrazone 200-400 mg / day
  - Benzbromarone 100-200 mg / day (not available)
- Atorvastatin
- Losartan
- Fenofibrate
- Amlodipine

Advanced Imaging to confirm diagnosis

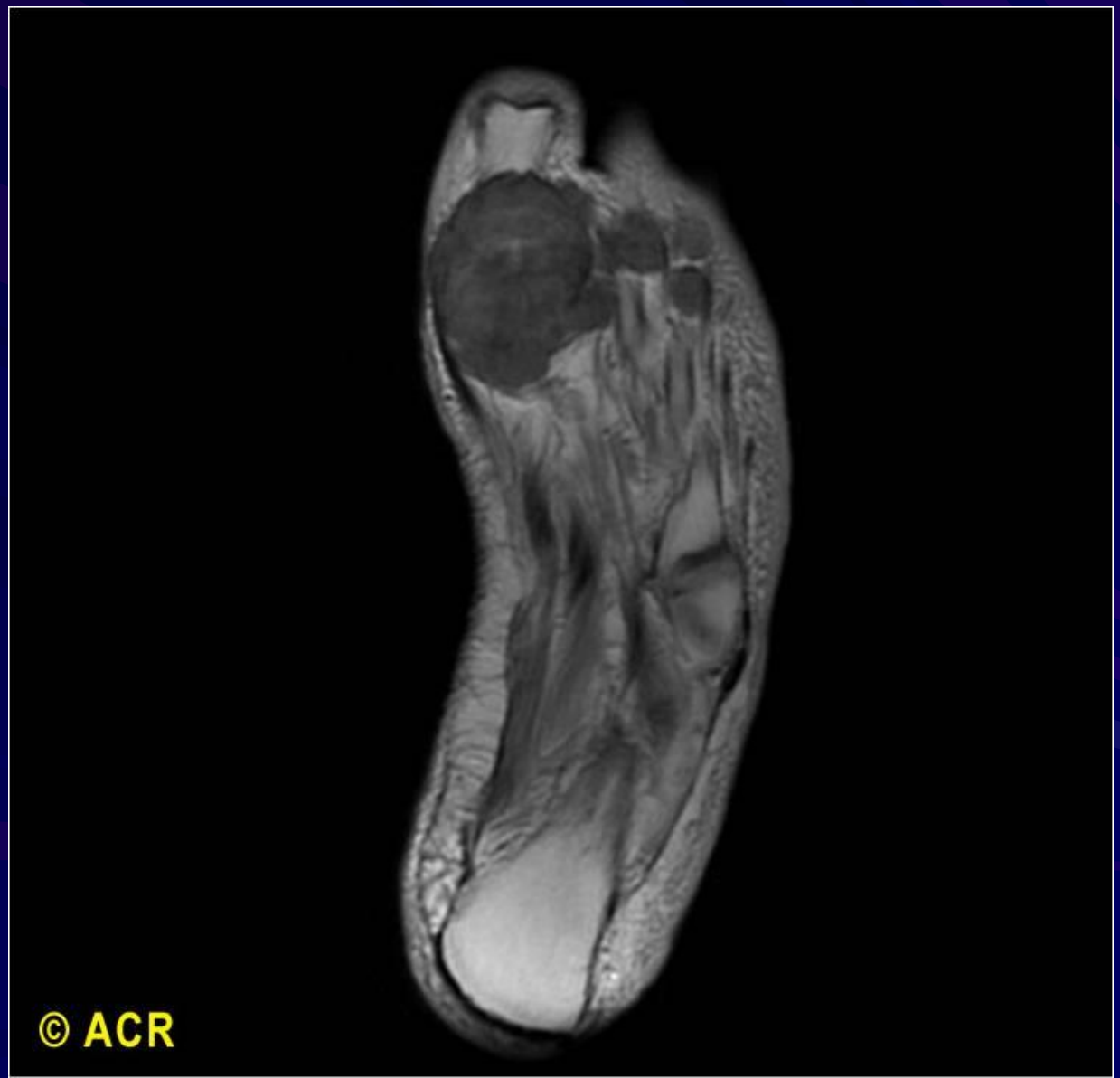
ULTRASOUND IMAGING



Double contour sign



MRI FOOT FOR TOPHUS

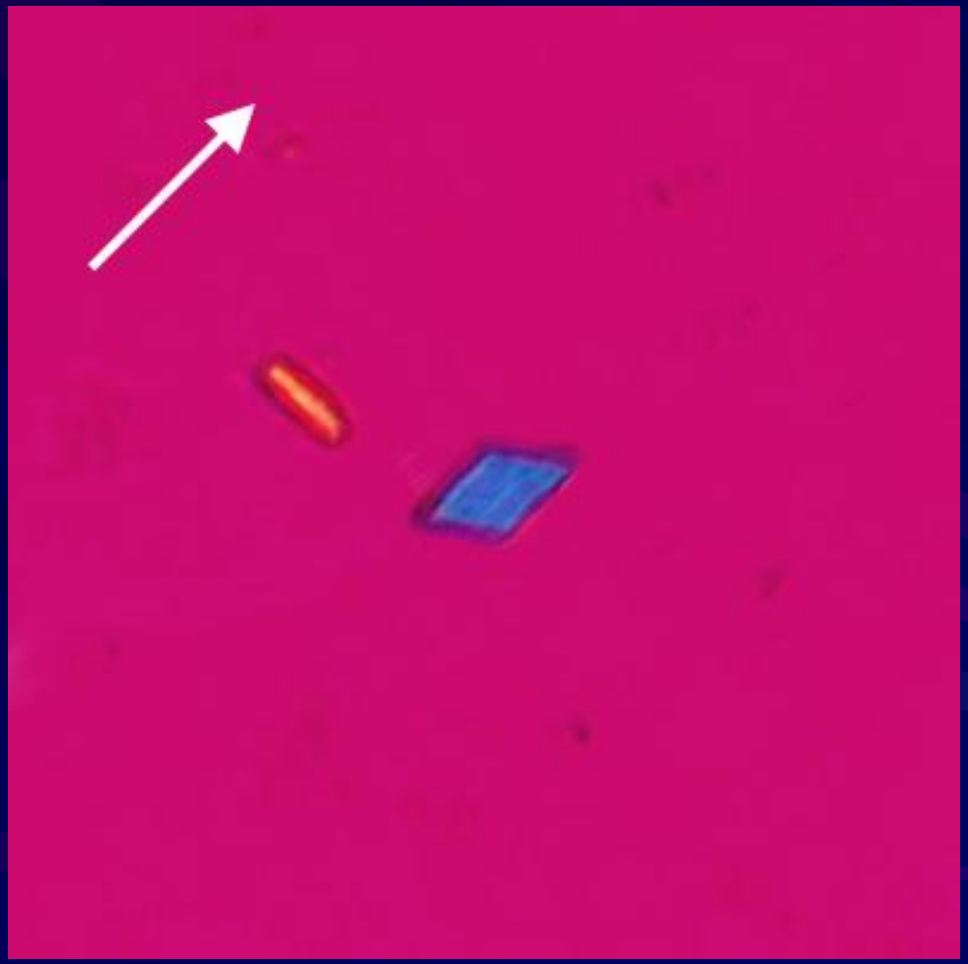


CPPD DISEASE

Acute CPPD

- Acute Pseudogout
  - Positive birefringent rod shaped crystals
  - More likely in OA joint – knee> wrist> MCPs> hips,shoulders,ankles

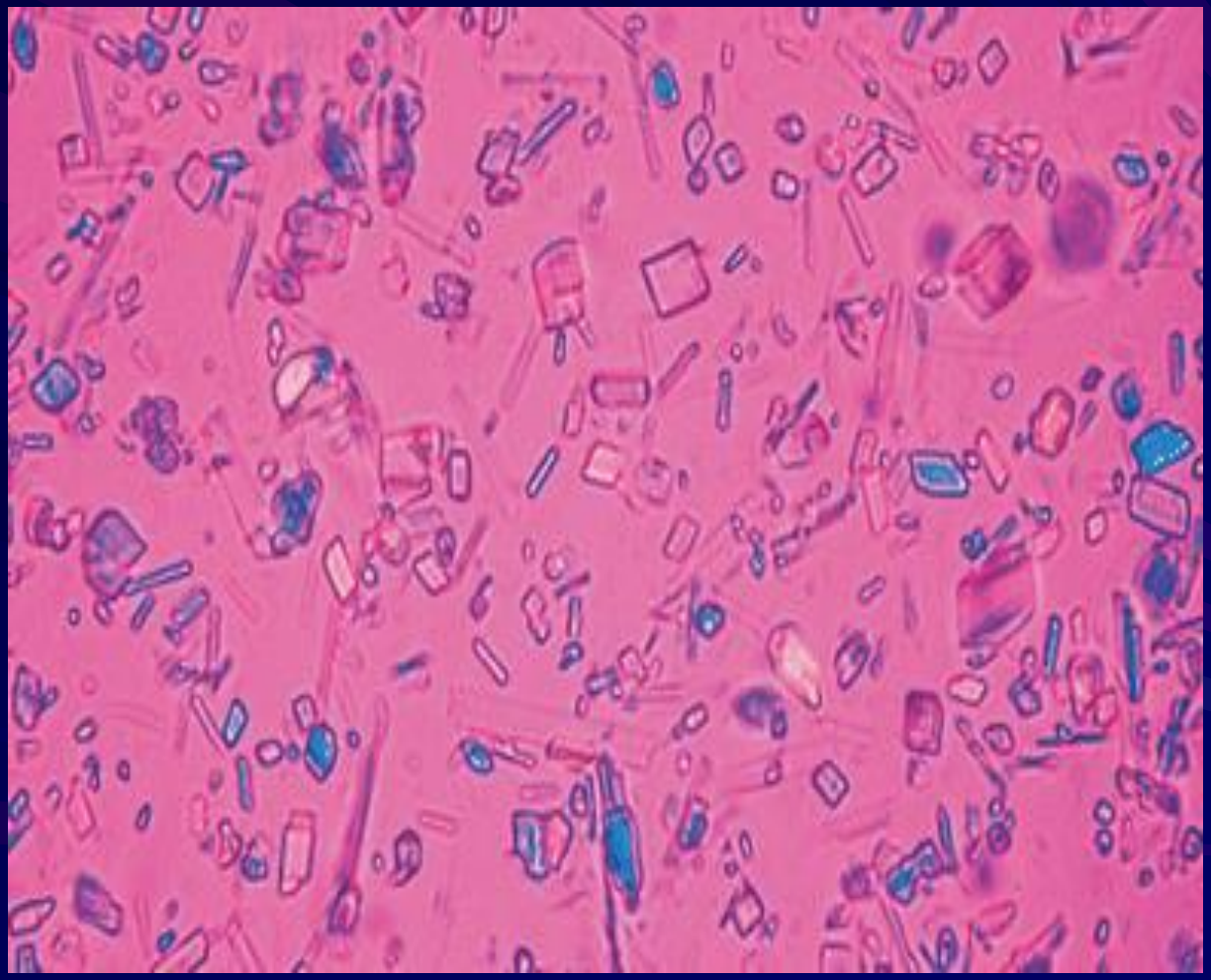
CPPD CRYSTAL



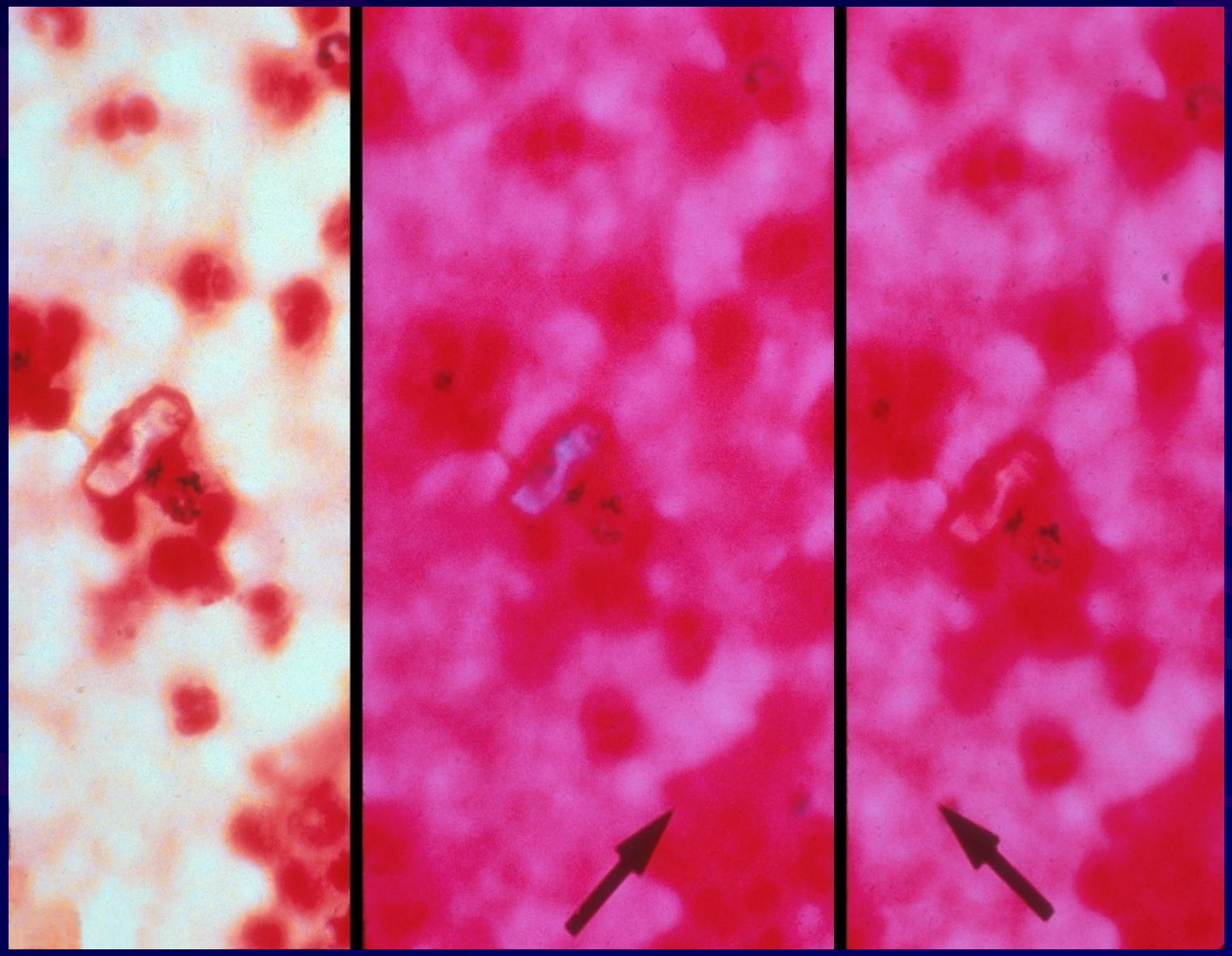
CPPD Presentations

- Pseudo-rheumatoid pattern
- Osteoarthritis with/out pseudogout
- Chondrocalcinosis
- Neuropathic joint
- Tumoral CPPD deposition

ROD OR RHOMBOID



Coexist with Septic arthritis



ASSOCIATIONS

- Hyper PTH
- Hemochromatosis
- Hypothyroidism
- Hypomagnesiemia
- Hypercalcemia
- Hypophatasia

MASSIVE CPPD

Fig. 1. Showing nodular mass occupying dorsum of right hand. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

TISSUE STAINING

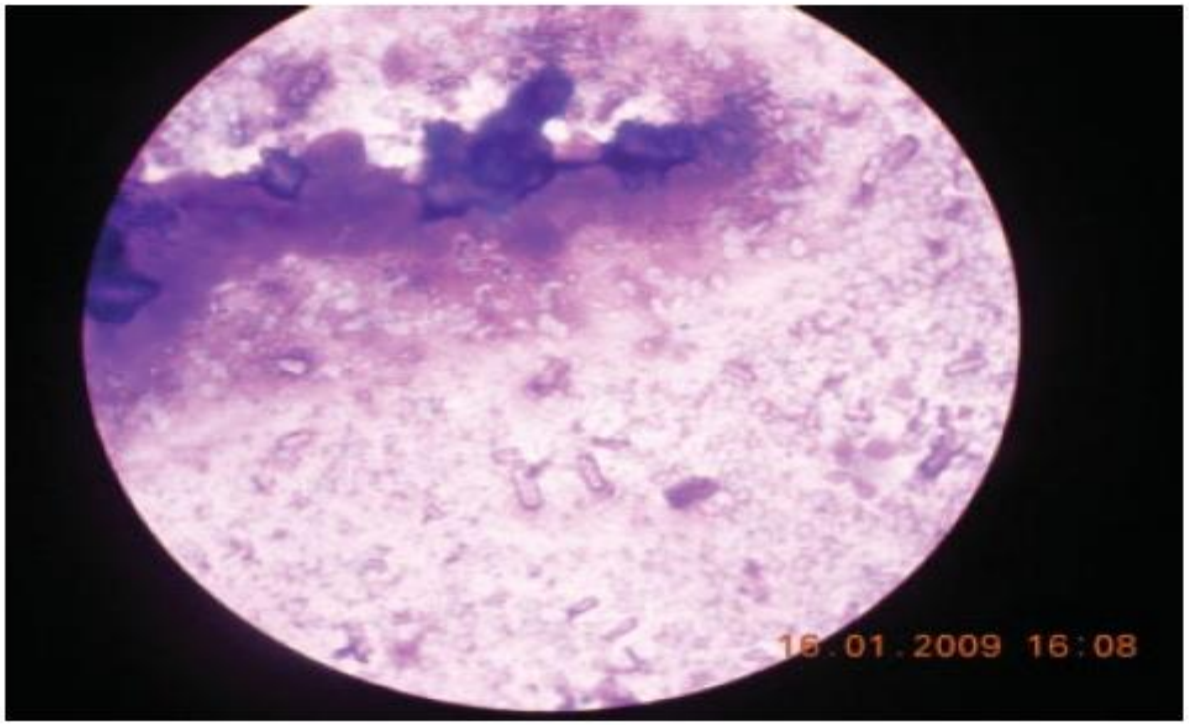
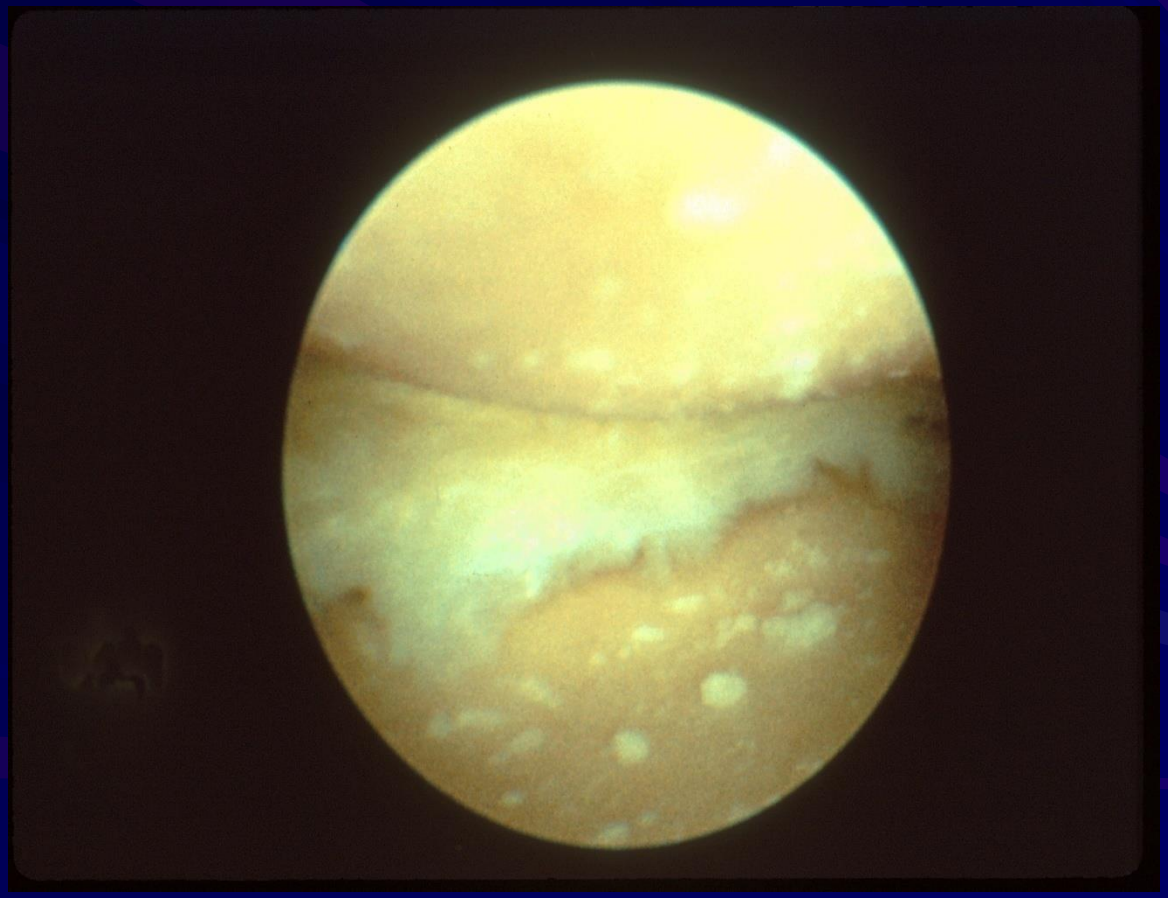


Fig. 3. Photomicrograph showing abundant basophilic calcified deposits and rhomboid crystals (Giemsa, ×400). [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

FLUFFY DEPOSITS OF WRIST

Fig. 2. X-ray right hand (antero-posterior view) showing radio-opaque mass with fluffy pattern.

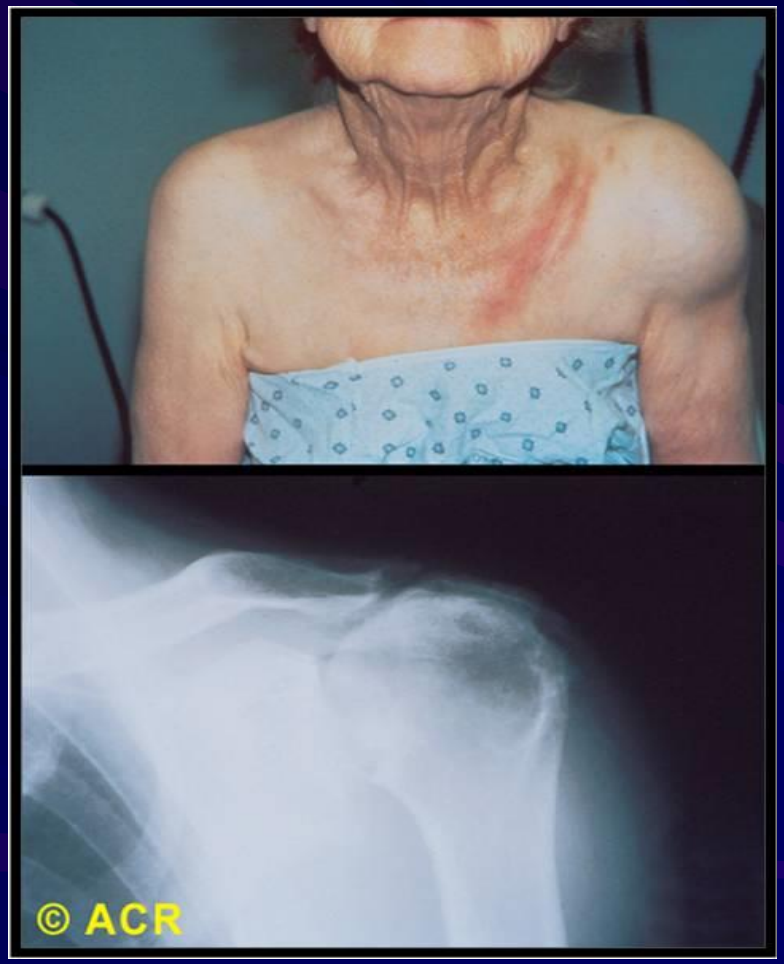
CPPD ON LAPRSCOPY



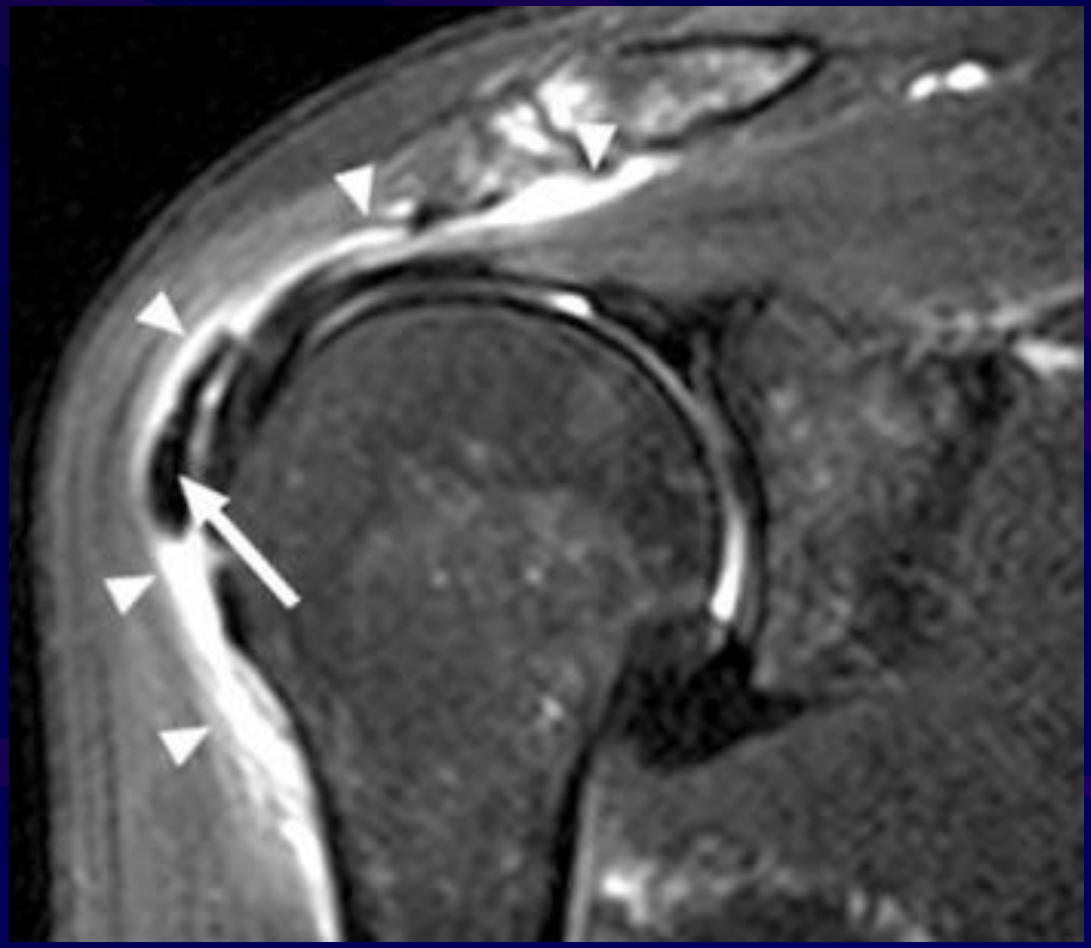
BASIC CALCIUM PHOSPHATE

- Usually in the form of hydroxyapatite (CHA)
- Age related arthropathy except for pseudopodagra in young women
- "Milwaukee Shoulder"
- Calcific Periarthritis
- Soft tissue calcification
- Osteoarthritis (found in 70% of OA synovial fluid)

MILAWAUKEE SHOULDER HYDROXAPATITE

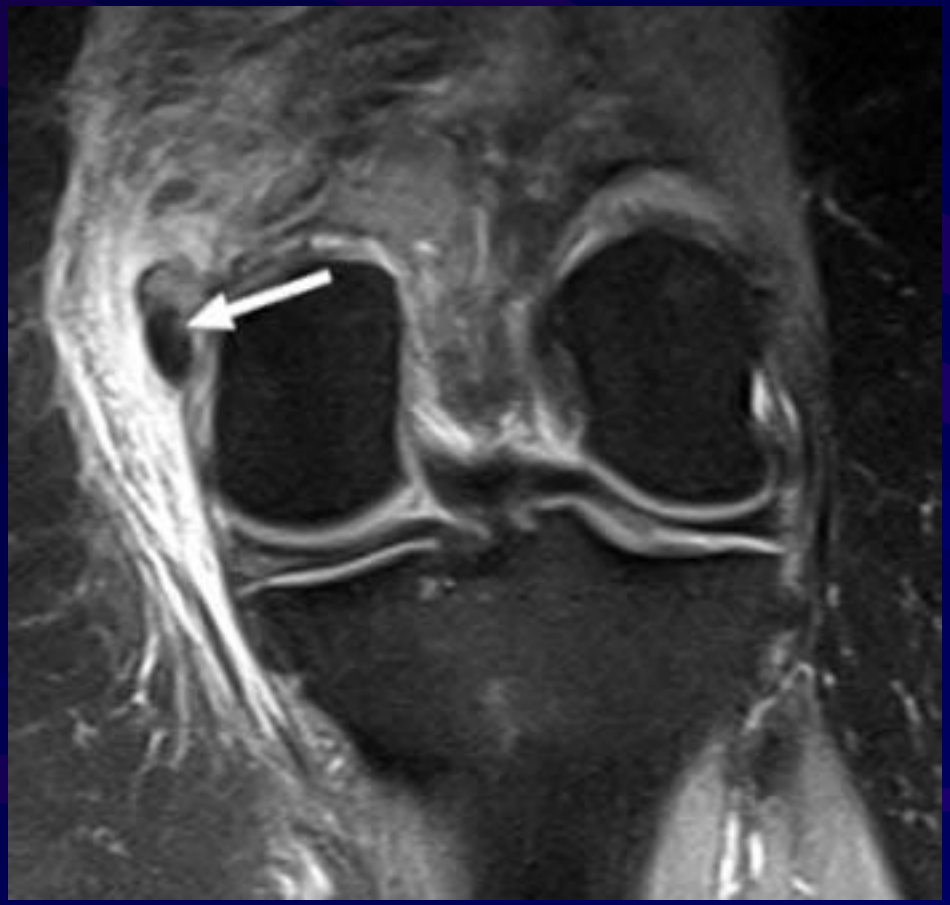


Calcium Hydroxyapatite



- *Coronal oblique T2-weighted fat suppressed image shows a deposit of CHA crystals (arrow)*

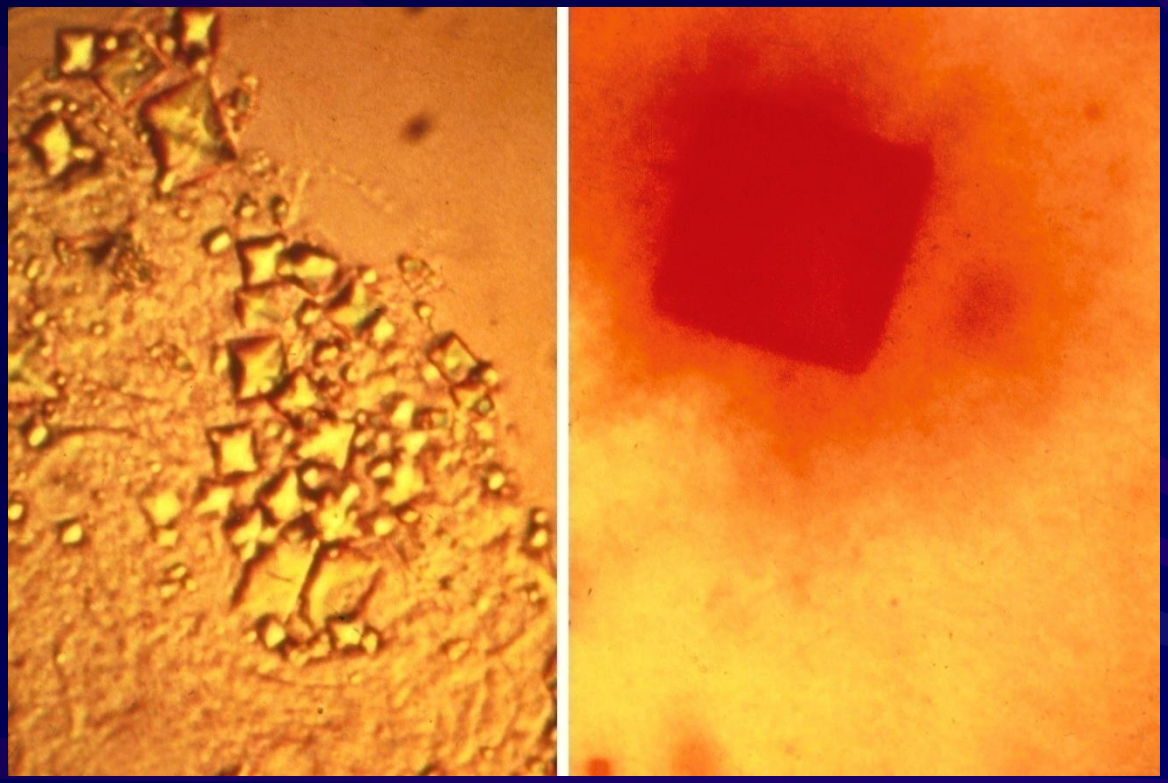
- *Prominent subdeltoid bursitis is also present (arrowheads)*



- *A coronal T-2 weighted image of the knee demonstrates a deposit of CHA crystals adjacent to the medial femoral condyle (arrow)*

- *Prominent soft tissue inflammation is present*

CALCIUM OXALATE CRF



CPPD & BCP Treatment

- NSAIDS
- COLCHICINE (prophylaxis in cppd)
- STEROID INJECTION
- ULTRASOUND GUIDED ASPIRATION
- SURGERY