Crystal: Diagnosis and Management of Gout (002)

Sunday, November 16, 2014
7:45 AM - 9:15 AM

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Faculty Disclosure

Theodore R. Fields, MD
Disclosure: AstraZeneca Pharmaceuticals, 5; AstraZenica Pharmaceuticals, 5; Crealta Pharmaceuticals, 5; Pfizer Pharmaceuticals, 8; Takeda Pharmaceuticals, 5, 8
Crystal: Diagnosis and Management of Gout (002)

Theodore R. Fields, MD, Hospital for Special Surgery Weill Cornell Medical College, New York, NY

Session Overview:

Diagnostic criteria have been recently updated. Imaging modalities, such as ultrasound and Dual Energy CT scanning, are being applied to gout patients and showing more extensive areas of involvement than previously apparent. ACR guidelines for management of acute gout, chronic urate-lowering therapy (ULT) and prophylaxis of flares when starting ULT were published in 2012, and many comments have been published since. Many studies of additional treatments of gout (e.g. IL-1 blockade) have been published since the 2012 guidelines. A number of studies looking at the overall success of gout treatment, ways to educate patients with gout, and ways to improve gout management have been published since the 2013 ACR meeting. This session will review the diagnosis and management of gout, with an emphasis on articles and abstracts published since the 2013 ACR meeting, and will include a discussion of the gout abstracts being presented at this 2014 ACR meeting, and a discussion of what is in the pipeline for gout management.

Upon completion of this session, participants should be able to:

- recall the various published guidelines for diagnosis of gout, including the most recent, and appreciate the input that, when appropriate, can be added with ultrasound and Dual Energy CT scanning
- appreciate the challenges in gout patient education and medication adherence
- recall the 2012 ACR guidelines for gout management, and appreciate those issues that still remain controversial or unresolved
- discuss the types of agents in the pipeline for future gout therapy, and potential future approaches to gout management
Diagnosis and Management of Gout

Theodore Fields, MD
11/16/14
• HSS educational activities are carried out in a manner that serves the educational component of our Mission.

• As faculty we are committed to providing transparency in any/all external relationships prior to giving an academic presentation.

Theodore R. Fields, MD
Advisory Board/Speakers’ Bureau: Takeda, AstraZenica, Crealta and Pfizer Pharmaceuticals
Evidence Based Medicine


• Park D-J et al: Cost-Effectiveness Analysis of HLA-B5801 Genotyping in the Treatment of Gout Patients with Chronic Renal Insufficiency In Korea. *Arthritis Rheum*


• Sivera F et al: Multinational evidence-based recommendations for the diagnosis and management of gout: integrating systematic literature review and expert opinion of a broad panel of rheumatologists in the 3e initiative. *Ann Rheum Dis*, Epub ahead of print Jul 18 2013
The Irony of Gout Management

• Gout is one of the most effectively treated of all rheumatic diseases, with “cure” possible in vast majority who stay on medication

YET…

• Gout is one of, if not the absolute, worst managed long term, as shown in at least 20 studies.
Rheumatologist Role in Gout Management

• Manage our own cases including the most difficult cases
• Educate PCP’s in managing their own cases (8.3 million cases much more than rheumatologists can handle)
Challenges in Gout Management – Patient and Provider Factors

• **Patients don’t take their medication** – even when they see rheumatologists! Or they are underdosed, or they never get urate-lowering therapy (ULT).

• PCP’s, and even rheumatologists, tell patients *how* to take their medication, but often don’t have time to adequately teach patients *why* and *when* to take them.

• Multiple comorbidities (only 1/10 without comorbidity) take up visit time for PCP’s
Challenges in Gout Management – Patient and Provider Factors II

Patients often don’t understand the 3 different types of gout medications:

1. **Rescue meds** - for flares (oral and injected steroid, NSAID, colchicine, occasionally anakinra.

2. **Long-term urate lowering therapy (ULT)** - allopurinol, febuxostat, probenecid, pegloticase

3. **Bridge meds** - for 1st 6 months or more of ULT - colchicine, sometimes NSAIDs or low-dose prednisone
Challenges in Gout Management – Patient and Provider Factors III

Why does this happen? (based on focus group results and study reports- references on subsequent slides):

- Patients feel better and stop ULT
- Use of alternative therapies.
- Concerns about side-effects
- ULT-related mobilization flares lead to medication discontinuation
- Patients with comorbidities decide “too many pills”
- Multiple co-morbidities crowd gout out of office visit discussion
- PCP’s not always buying in to need for urate < 6.0.
- Patients don’t understand the 3 different types of gout medications
Challenges in Gout Management – Medical Factors

• **Many cases are hard to manage**, despite our excellent medications
• Hospitalized diabetic with GI disease is a challenge to treat for acute gout
• A number of patients don’t reach uric acid goal with usual doses of urate-lowering therapy, and strategies needed
• Some patients with very severe tophi may not be well served with slow tophus shrinkage
Challenges in Gout Diagnosis- Medical Factors II

• More for PCP than Rheumatologist- but absence of crystal diagnosis can lead to misdiagnosis
• Mimics: Lyme disease, pseudogout, septic arthritis, rheumatoid arthritis can be misdiagnosed as gout
• Mixed crystal disease: pseudogout and gout together not rare
Selected Recently Published Articles on Gout
New Staging for Gout?

In view of advanced imaging findings diagnostic of crystal deposition, a new staging system proposed:

**Stage A:** High risk for gout, or hyperuricemia without evidence of MSU crystal deposition or gouty symptoms

**Stage B:** MSU crystal deposition by microscopy or advanced imaging, but without signs or symptoms of gout. This stage would encompass individuals who display the double contour sign on US, urate deposition on DECT, and MSU crystals seen with microscopy

**Stage C:** MSU crystal deposition with prior or current symptoms of acute gout flares (for example, people with a current or previous flare)

**Stage D:** Advanced gout requiring specialist interventions, encompassing people with tophi, chronic gouty arthritis and radiographic erosions

Asymptomatic Hyperuricemia

Allopurinol Overuse in Asymptomatic Hyperuricemia: “A Teachable Moment”

• 7 cases of apparently unnecessary treatment reported to an Italian monitoring center.
• One was fatal, an 81 year old woman with hypersensitivity reaction
• Not addressed in ACR guidelines, but might consider treatment of asymptomatic hyperuricemia when: (1) uric acid above 13 mg/dL in men or 10 mg/dL in women (potential nephrotoxicity) (2) urinary excretion of uric acid >1,100 mg daily, re renal stones and (3) patients about to receive chemotherapy to prevent uric acid nephropathy from tumor lysis syndrome

Colchicine Appropriate Use

93 (73.8%) of 126 patients were being treated counter to guidelines 34 (27.0%) were prescribed no urate-lowering therapy, 50 (39.7%) were not at the uric acid goal and had not had urate-lowering therapy increased in the prior 3 months, and 9 (7.1%) were at the uric acid goal for >1 year with no flares or tophi. Colchicine use was considered appropriate in 33 patients (26.2%)

HLA-B5801 testing

Model in Korea suggested that gout treatment informed by HLA-B5801 genotyping is less costly and more effective than treatment without genotyping, and HLA-B5801 genotyping could considerably reduce the occurrence of allopurinol-induced SCARs and related deaths.

FAST Trial allopurinol vs febuxostat- Cardiac Safety

Defining study in progress:
• Prospective, randomized, open, blinded endpoint design.
• UK and Denmark.
• Patients > 60, given allopurinol for symptomatic hyperuricaemia and have at least one additional cardiovascular risk factor.
• After allopurinol dosing optimized - randomized to either continue optimal dose allopurinol or to use febuxostat.
• Patients are followed-up for an average of 3 years.
• The primary endpoint is first occurrence of the Anti-Platelet Trialists’ Collaboration (APTC) cardiovascular endpoint of non-fatal myocardial infarction, non-fatal stroke or cardiovascular death.
• N= 5706 patients with 2853 patients in each treatment arm.

Allopurinol and Febuxostat and Renal Function

• 1-year cohort study of 73 hyperuricemic patients who had (eGFR) below 45 ml/min and were being treated with urate-lowering therapy.
• 51 patients: changed from allopurinol to febuxostat, 22 patients continued on allopurinol.
• The serum UA levels significantly decreased from 6.1 ± 1.0 to 5.7 ± 1.2 mg/dl in the febuxostat group and significantly increased from 6.2 ± 1.1 to 6.6 ± 1.1 mg/dl in the allopurinol group.
• The eGFR decreased 27.3 to 25.7 ml/min in the febuxostat group and from 26.1 to 19.9 ml/min in the allopurinol group.
• The switch from allopurinol to febuxostat was significantly associated with the changes in eGFR according to a multiple regression analysis ($\beta = -0.22145$, $P < 0.05$).
• Febuxostat reduced the serum UA levels and slowed the progression of renal disease in our CKD cohort in comparison with allopurinol.

Gout and Vascular Disease

- **OBJECTIVES:** To determine whether gout increases risk of incident coronary heart disease (CHD), cerebrovascular (CVD) and peripheral vascular disease (PVD) in a large cohort of primary care patients with gout, since there have been no such large studies in primary care.

- **METHODS:** A retrospective cohort study 8386 patients with an incident diagnosis of gout, and 39,766 age, sex and registered general practice-matched controls, all aged over 50 years and with no prior vascular history.

- **RESULTS:** Multivariable analysis showed men were at increased risk of any vascular event (HRs (95% CIs)) HR 1.06 (1.01 to 1.12), any CHD HR 1.08 (1.01 to 1.15) and PVD HR 1.18 (1.01 to 1.38), while women were at increased risk of any vascular event, HR 1.25 (1.12 to 1.39), and PVD 1.89 (1.50 to 2.38) but not any CVD.

- **CONCLUSIONS:** In this cohort of over 50s with gout, female patients with gout were at greatest risk of incident vascular events, even after adjustment for vascular risk factors, despite a higher prevalence of both gout and vascular disease in men.

Selected EULAR Abstracts 2014
Procalcitonin in Differentiating Acute Gouty Arthritis from Infection

• Serum procalcitonin levels were significantly lower in patients with acute gouty arthritis than in patients with bacterial infection. The serum procalcitonin level may be a useful serologic marker for the differentiating acute gout arthritis from bacterial infection.

Canakinumab vs Triamcinolone Injection in Difficult-to-Treat Gout Patients

- Canakinumab in difficult-to-treat gout with better pain relief and reduced risk of new attacks compared to triamcinolone.
- Safety profile consistent with prior studies

Updated EULAR Evidence-Based Recommendations for Diagnosis of Gout

- A search for crystals in synovial fluid (SF) or tophus aspirates was recommended in every person with suspected gout, because demonstration of monosodium urate (MSU) crystals allows a definitive diagnosis of gout.

Richette P et al: Updated EULAR Evidence-Based Recommendations for Diagnosis of Gout. EULAR abstract 0532, 2014
Equation for 24 hour urinary Uric Acid Excretion

• includes age, body weight, and gender, providing corrections for differences in urinary creatinine excretions among individuals.

• eUUE correlates better with the 24-h mUUE than the Uu/Ucr ratio does.

• If validated, could allow identification of an overexcretor of uric acid without the need for a 24-h urine collection.

SELECTED ARTICLES/ABSTRACTS ON ADHERENCE TO GOUT THERAPY (from many such articles)
Adherence to Gout Medication

Primary care providers knowledge, beliefs and treatment practices for gout: results of a physician questionnaire

- **Methods**: national survey of a random sample of US primary care physicians to assess their treatment of acute, intercritical and tophaceous gout using published European and American gout treatment recommendations and guidelines as a gold standard.

- **Results**: Inappropriate dosing of medications in the setting of renal disease and lack of prophylaxis when initiating urate-lowering therapy (ULT) accounted for much of the lack of compliance with treatment recommendations.

- For intercritical gout in the setting of renal disease, 3% would provide care consistent with the recommendations, including initiating a ULT at the appropriate dose with dosing titration to a serum urate level of ≤6 mg/dl and providing prophylaxis.

- For tophaceous gout, 17% reported care consistent with the recommendations,

Harrold LR et al, Rheumatology 52(9), 1623-1629, 2013
24 patients participated in 4 focus groups: 1) Patients did not clearly understand the natural history of gout; 2) patients did not realize that recurrent acute flares resulted in chronic joint damage; 3) there was lack of knowledge about treatment options and duration of therapy for both acute and chronic gout; 4) patients felt that physicians did not spend enough time explaining the progression, i.e., natural history of the disease and its long-term effects; 5) patients did not grasp the need for chronic ULT to avoid complications and disability; and 6) patients were not aware of treatment goals for hyperuricemia, as evident by adherence to their gout medications. In these groups, 38% had low and 42% had medium adherence to their gout medications, respectively.

The physician focus group (4 PCPs) revealed that they did not manage gout as a chronic disease which limited initiation of ULT and monitoring of serum urate to achieve the target.

P. Khanna

Citation: Ann Rheum Dis 2013;72(Suppl3):768
Rates of Adherence and Persistence with Allopurinol Therapy Among Gout Patients in Israel

- 1 of 6 gout patients was fully adherent with allopurinol
- 7644 patients. 17% adherent. 36% partial adherence. 47% poor adherence.
- Average duration until discontinuation 358 days in men and 379 days in women.

GOUT GUIDELINES – DISCUSSION OF SELECTED ITEMS OF SPECIAL INTEREST
Background

• ACR 10/2012
• Multinational 7/2013
Should we treat asymptomatic hyperuricemia for cardiovascular benefit?

Why is this still a hard question?

DINESH KHANNA,1 PUJA P. KHANNA,1 JOHN D. FITZGERALD,2 MANJIT K. SINGH,3 SANGMEE BAE,2 TUHINA NEOGI,4 MICHAEL H. PILLINGER,5 JOAN MERILL,6 SUSAN LEE,7 SHRADDHA PRAKASH,2 MARIAN KALDAS,2 MANEESH GOGIA,2 FERNANDO PEREZ-RUIZ,8 WILL TAYLOR,9 FRÉDÉRIC LIOTÉ,10 HYON CHOI,4 JASVINDER A. SINGH,11 NICOLA DALBETH,12 SANFORD KAPLAN,13 VANDANA NIYYAR,14 DANIELLE JONES,14 STEVEN A. YAROWS,15 BLAKE ROESSLER,1 GAIL KERR,16 CHARLES KING,17 GERALD LEVY,18 DANIEL E. FURST,2 N. LAWRENCE EDWARDS,19 BRIAN MANDELL,20 H. RALPH SCHUMACHER,21 MARK ROBBINS,22 NEIL WENGER,2 AND ROBERT TERKELTAUB7
Multinational evidence-based recommendations for the diagnosis and management of gout: integrating systematic literature review and expert opinion of a broad panel of rheumatologists in the 3e initiative


Ann Rheum Dis published online July 18, 2013
Multinational gout guidelines: how do we move beyond ‘déjà vu’?

Robert Terkeltaub

Consensus guidelines for patient management can increase disease awareness and education, and facilitate evidence-based clinical decision-making. Nevertheless, new gout recommendations leave a ‘déjà vu’ impression. Novel evidence from clinical trials is needed to robustly advance patient care and build the next generation of guidelines.

Terkeltaub, R. Nat. Rev. Rheumatol. advance online publication 17 September 2013; doi:10.1038/nrrheum.2013.142

Gout is increasingly frequent and clinically complex, owing to a ‘perfect storm’ of factors. Next, 474 rheumatologists from Europe, South America and Australasia contributed characteristic imaging findings [Grade of recommendation B]. Thus, the recommendations reiterate the value of urate crystal identification in joint fluid for definitive diagnosis, but suggest that classic clinical features of acute gout, and “characteristic imaging findings” can provide alternative evidence.1 Although the 3e team did not define what constitutes a ‘rapid response’ to colchicine, the feature is cited as characteristic of acute gout. However, assessment of colchicine clinical response in patients with gout has substantial limits in sensitivity and specificity3,6 and, astutely, was not included in the 1977 ACR classification criteria for gouty arthritis or in the 2006 EULAR likelihood ratios for gout diagnosis.4 Similarly, whereas
Included in Multinational Guidelines - Not ACR

- Benzbromarone
- 1.0mg colchicine tabs
- Cost consideration
Q #1: ACCURATE DIAGNOSIS: In which circumstances can a diagnosis of gout be made on clinical grounds with or without laboratory tests or imaging and when is the identification of crystals necessary?

A #1: Identification of MSU crystals should be performed for a definite diagnosis of gout; if not possible, a diagnosis of gout can be supported by classical clinical features* (such as podagra, tophi, rapid response to colchicine) and/or characteristic imaging findings and/or characteristic imaging findings. (similar to 1977 ARA gout diagnosis guideline)
“Déjà vu” Terkeltaub Editorial

1. **Accurate diagnosis**: advanced imaging should be compared with synovial fluid analysis in acute gout diagnosis.

(e.g. DECT, MRI, ultrasound)

Also: “abandoning ‘gold standard’ synovial fluid analysis….would not be prudent for many cases of acute arthritis.”
<table>
<thead>
<tr>
<th>Table 2. Specific recommendation of a comorbidity checklist for gout patients</th>
</tr>
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<tbody>
<tr>
<td>Appropriate to consider in the clinical evaluation, and if clinically indicated, to evaluate (evidence C for all)*</td>
</tr>
<tr>
<td>Obesity, dietary factors</td>
</tr>
<tr>
<td>Excessive alcohol intake</td>
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<tr>
<td>Metabolic syndrome, type 2 diabetes mellitus</td>
</tr>
<tr>
<td>Hypertension†</td>
</tr>
<tr>
<td>Hyperlipidemia, modifiable risk factors for coronary artery disease or stroke</td>
</tr>
<tr>
<td>Serum urate–elevating medications†</td>
</tr>
<tr>
<td>History of urolithiasis</td>
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<tr>
<td>Chronic kidney, glomerular, or interstitial renal disease (e.g., analgesic nephropathy, polycystic kidney disease)</td>
</tr>
<tr>
<td>In selected cases, potential genetic or acquired cause of uric acid overproduction (e.g., inborn error of purine metabolism or psoriasis, myeloproliferative, or lymphoproliferative disease, respectively)</td>
</tr>
<tr>
<td>Lead intoxication</td>
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</tbody>
</table>
Q #3: MANAGEMENT OF ACUTE GOUT: What is the role of glucocorticoids, colchicine, NSAIDs, anti-IL1 and paracetamol in the management of acute gout?

A #3: Acute gout should be treated with low-dose colchicine (up to 2 mg daily), NSAIDs and/or glucocorticoids (intra-articular, oral or intramuscular) depending on comorbidities and risk of adverse effects.
Starting ULT

Indications for Pharmacologic ULT
Any patient with established diagnosis of gouty arthritis and
- Tophus or tophi by clinical exam or imaging study [A]
- Frequent attacks of acute gouty arthritis (≥2 attacks/yr) [A]
- CKD stage 2 or worse [C]
- Past urolithiasis [C]

If Pharmacologic ULT is indicated

TREAT TO SERUM URATE TARGET defined for individual patient
- The minimum serum urate target is <6mg/dL
- Serum urate lowering below 5mg/dL may be needed to improve gout signs and symptoms

Select First Line ULT agent  See Table 3, Figure 5
Xanthine Oxidase Inhibitor (XOI): [A]
  - Allopurinol
  - OR
  - Febuxostat

If at least one XOI is contra-indicated or not tolerated
Alternative First Line ULT:
- Probencid [B]

Acute Gout Prophylaxis
Initiate concomitant pharmacologic anti-inflammatory gout attack prophylaxis
See Part II of the Guidelines

^Recommend against probenecid if creatinine clearance <50 ml/min
Core Recommendations: Allopurinol

Allopurinol
Starting dosage should be no greater than 100 mg/day for any patient, and start at 50 mg/day in stage 4 or worse CKD (evidence B)
Gradually titrate maintenance dose upward every 2–5 weeks to appropriate maximum dose in order to treat to chosen SUA target (evidence C)
Dose can be raised above 300 mg daily, even with renal impairment, as long as it is accompanied by adequate patient education and monitoring for drug toxicity (e.g., pruritus, rash, elevated hepatic transaminases; evidence B)
Prior to initiation, consider HLA–B*5801 in selected patients, specifically in subpopulations at higher risk for severe allopurinol hypersensitivity reaction (e.g., Koreans with stage 3 or worse CKD, and Han Chinese and Thai irrespective of renal function; evidence A)
6. **Acute gout flare prophylaxis**: pharmacological gout flare prophylaxis should be compared with complementary or alternative strategies (e.g. **gradual upwards titration of ULT**; addressing lifestyle and iatrogenic flare triggers).
Effect of comorbidities on drug choices:
prospective RCTs should assess renal and cardiovascular outcomes of ULT; other trials should evaluate cost-effectiveness of ethnic, racial and renal comorbidity risk factor profiling, and of HLA-B*5801 pharmacogenetics vs allopurinol “start low, go slow” dosing alone to prevent cases of severe allopurinol hypersensitivity syndrome.
“Déjà vu” Terkeltaub Editorial

**Monitoring treatment:** SUA target, and other outcomes (symptoms, tophi): prospective RCTs of ULT outcomes stratified for differing SUA targets (e.g. <2 mg/dl, <4 mg/dl, <6 mg/dl).
Conclusions

• Differences between ACR and multinational guidelines:
  – multinational made occasional cost-effectiveness statement,
  – ACR had nephrologist, patient advocate
  – Voting mechanism different

• A number of conclusions in both at “C” level: consensus, case studies or standard-of-care

• More data still needed, e.g. cost-effectiveness. benefit/risk of treating asymptomatic hyperuricemia, combination therapy, etc.
ACR ABSTRACTS 2013 – ONE REVIEW
Does Starting Allopurinol Prolong Acute Treated Gout?

- Abstract: #1722
- Presenter: Hill, Erica DO
- Date: Monday, October 28, 2013 Time: 3:00 PM
- Location: Hilton - Indigo E  Oral

- **Conclusion:** There is no difference in DTR or Likert pain scales between allopurinol and placebo groups treated for acute gout. This supports the new ACR guidelines expert consensus that allopurinol may be initiated in acute gout, providing the acute attack is appropriately managed.

- I would consider this controversial – for discussion.
ACR ABSTRACTS 2014 DISCUSSION
SPECIAL TOPICS IN GOUT
Concept attribution: video 2012- Dr. Stephen Malawista
5 years of urate < 6.0, % requiring treatment for flares while on maintenance urate lowering agent

Sample Patient Instructions

A) Before starting urate-lowering therapy
B) As they start urate-lowering therapy
Managing Your Gout Prior to Starting Uric Acid Lowering Therapy

Colchicine dosing
Stay on colchicine 0.6mg once a day. Take with food.
If you aren’t having any side-effects from colchicine, stay on it. If you think you might be having a side-effect, speak with me. Often we can help by cutting the dose, but not stopping the medication.
The colchicine can help treat a gout attack, but also is used for prevention of gout attacks. Therefore, stay on it until you speak with me or see me even if you are not having any gout pain.

How should you treat a gout flare-up?
Use prednisone – see the sheet giving you the dosing regimen/naproxen/ibuprofen. Note that you can stop if the inflamed area is completely back to normal. Also, if the inflammation is not completely resolved when you finish the four days, call my office so that we can make new plans. Partially-treated attacks often flare up again.

What is the long-term picture?
Once your gout has been quiet for about 2 weeks, we will want to start a medication to lower uric acid, generally allopurinol or Febuxostat.
The medications that treat attacks of gout (such as anti-inflammatory medications, corticosteroids such as prednisone and colchicine) do not lower the uric acid level so are not really the long-term answer here. We try to wait until the gout is quiet before we start a medication to lower the uric acid (because those medications, such as allopurinol and Febuxostat) can cause a gout flare. We will discuss ways to reduce that risk when you are starting one of those medications.

What else can you do to prevent or manage flare-ups of gout in this early phase of Allopurinol/Febuxostat treatment?
Be careful about your diet. See my handout on gout for guidance on this. Even if you don’t eliminate certain items from your diet, cutting their portion size will help.
Make sure you are clear on what to do if you have an attack of gout. It’s important to start treatment within hours of onset of the attack—treatment works much faster in the early phase of attacks.
If in doubt, call. We can almost always come up with a regimen to knock out a gout attack, but the longer the attack has gone on, the harder it is to manage. If you start treatment for an attack and it isn’t significantly better in 2 days, be sure to call my office, and if not completely gone in 4 days please call.
Patient Instructions- Starting Allopurinol

Allopurinol dosing
1. Allopurinol 100mg daily for a week (with or without food)
2. Then 200mg daily for a week
3. Then 300mg daily for 2 weeks
4. Then get labs, and stay on the 300mg daily allopurinol until you speak with me

Colchicine dosing
Stay on colchicine 0.6mg once a day. Take with food.

Why are we doing the medication dosing this way?
Allopurinol, when first started, can cause flares of gout. In the long run, allopurinol is spectacularly good at stopping gout, but in the very beginning it can cause flare-ups. A gout flare when you start allopurinol is not a reason to stop allopurinol.

Colchicine helps to prevent the flares of gout you can get when we start allopurinol. We try to stop the Colchicine in about 6 months if you are not getting gout attacks, but the allopurinol use is indefinite.

What is the long-term picture?
In the long run, allopurinol consistently lowers the blood uric acid, and over time gout attacks generally stop happening. If you stop the allopurinol, over time the uric acid in the joint builds back up, and attacks can start again.

Your goal uric acid level is less than 6. This goal will guide how we dose your allopurinol once we have your blood tests back.

What else can you do to prevent or manage flare-ups of gout in this early phase of allopurinol treatment?
Especially in the first 6 months of allopurinol therapy, be especially careful about your diet. See my handout on gout for guidance on this. Even if you don’t eliminate certain items from your diet, cutting their portion size will help.

Make sure you are clear on what to do if you have an attack of gout. It’s important to start treatment within hours of onset of the attack—treatment works much faster in the early phase of attacks.

For attacks use: prednisone  Naproxen  Indomethacin

If in doubt, call. We can almost always come up with a regimen to knock out a gout attack, but the longer the attack has gone on, the harder it is to manage. If you start treatment for an attack and it doesn’t work in 2 days, be sure to call my office.
Additional Resources

Websites:


• [www.HSS.edu](http://www.HSS.edu) – resources for patients and professionals
Additional Resources

Bibliography In addition to 3 EBM references cited earlier:

Additional Resources

Books: