Gout: Bridging Knowledge Gaps in Diagnosis and Treatment
Faculty

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Disclosures

• M. Susan Burke, MD, FACP serves as a speaker for Merck.

• Peng Thim Fan, MD serves as a speaker for Pfizer, Amgen, Genentech, AbbVie, and BMS.

• Louis Kuritzky, MD has no relationships to disclose.

• Wendy L. Wright, MS, RN, APRN, FNP, FAANP, FAAN serves on the speakers bureau for Takeda, Merz, and Sanofi. She is also a consultant for Merck, Pfizer, and Takeda.
Learning Objectives

At the conclusion of the activity, learners should be able to:

1. Describe the clinical course of gout and management approaches for obtaining a definitive diagnosis
2. Develop individualized treatment plans for treatment of acute gout flares
3. Apply new ACR guidelines for a target uric acid level goals for long-term urate lowering therapy
4. Implement appropriate pharmacotherapy to effectively maintain target uric acid levels on a long-term basis
PRE-TEST QUESTIONS
Pre-Test ARS Question 1

On a scale of 1 to 5, please rate how confident you are in diagnosing and treating patients with gout:

1. Not at all confident
2. Slightly confident
3. Moderately confident
4. Pretty much confident
5. Very confident
Charles is a 47 y.o. man who has a history of CHD, hypertension and hyperlipidemia. Since yesterday morning, he has had severe pain and swelling in his left big toe. On exam, he has erythema and swelling of his left first MTP joint.

What is the best way to diagnose gout in Charles?

1. Absence of arthritis in other joints
2. Elevated uric acid >7 mg/dL
3. Presence of gout erosions on X-rays
4. Presence of negatively birefringent crystals on polarizing microscopy of joint fluid
5. This is a classic case of gout, no other diagnosis likely!
You believe that Charles may have gout. You decide to treat him with which of the following regimens?

1. Indomethacin 100mg and then 50mg q8h for 3-4 days
2. Piroxicam (Feldene) 20mg daily with food
3. Colchicine 1mg followed by 0.5mg hourly x 6 doses or till diarrhea
4. A single intra-articular or IM corticosteroid injection
5. Allopurinol 300mg daily plus indomethacin
2 years later, Charles returns with an attack of gout involving his forefoot and ankle. He is now motivated to try and prevent future flares. Which agent has been shown to best consistently maintain uric acid levels < 6.0 in clinical trials

1. Allopurinol 300mg daily
2. Probenecid 500mg BID
3. Colchicicine 0.6mg BID
4. Febuxostat 80 mg daily
Charles returns 8 months after initiation of allopurinol for LTULT. His current allopurinol dose is 300 mg/d. His uric acid level has been 5.8 at the last two measurements. He has just experienced another gout flare, which responded to 5 days of prednisone. What is the next appropriate course for Rx?

1. Stay the course: ACR guidelines say the uric acid goal is < 6.0
2. Stay the course because he is at the maximum dose of allopurinol
3. Increase the allopurinol dose; ACR guidelines say that uric acid <6.0 is the minimum target
4. Treat with pegloticase
“Serum urate level should be lowered sufficiently to durably improve signs and symptoms of gout, with the target of < 6 mg/dl at a minimum, and often <5 mg/dl.”

Khanna D, et al Arthritis Care & Research 2012;64(10): 1431-1446
Gout Primary Care Management 2015

Sx Consistent with Acute Gout

If possible, confirm with microscopy

Serum Uric Acid now and/or > 2 weeks post-flare

Anti-inflammatory Rx

Colchicine → Steroids → NSAID

6 months? Attack Quiescent ≥2 weeks 6 weeks?

LTULT (Goal Uric Acid <6.0 mg/dL)

Allopurinol → Febuxostat → Probenecid

Refer for Pegloticase
AGENDA

- Historical
- Why Bother?
- Scope of the Problem
- Clinical Course
- Diagnosis
- Treatment of the Acute Attack
- Secondary Prevention
- Asymptomatic Hyperuricemia
Gout: Definition

- Gout (gowt) [L. *gutta* a drop, because of the ancient belief that the disease was due to a ‘noxa’ falling drop by drop into the joint]
“…. our Greek ancestors believed that the attacks were caused by the dropping of evil humours in the body towards the toe, so that they could then be expressed from the body.”
“Gout is often accompanied by serious comorbid disorders (HTN, CV and CKD, and all of the component features of the metabolic syndrome) and it is managed in primary care practice in about 90% of affected persons; therefore, identifying risk factors, optimizing Dx, and choosing appropriate Rx for gout are important skills for a wide array of caregivers.”

Chohan S “Hyperuricemia and Gout” Conn’ s Current Therapy 2011 Elsevier (Philadelphia)
Epidemiology

“...recent studies suggest a rising incidence of gout in New Zealand, the US, and other countries. The reasons for this are unclear, but rising incidence of obesity and insulin resistance syndrome, increasing longevity, frequent use of both diuretics and prophylactic low-dose ASA, more organ transplants, and changing dietary trends (excess calories and purines) are implicated.”

Fam AG “Gout and Hyperuricemia” Conn’s Current Therapy 2006 Rakel, Bope, eds. (Saunders Elsevier, Philadelphia)
Management ‘Issues’

• Dx: often misDx or not adequately established
• Improper management common
• Prolonged aSx hyperuricemia: consequences?

Rosenthal AK “Gout and Hyperuricemia” Conn’s Current Therapy Rakel and Bope, eds, Elsevier Science (Philadelphia) 2003
Scope of the Problem
Populations at risk:

- The risk of gout is a consequence of hyperuricemia
- Serum urate 7.0 to 8.9 mg/dl*
  - Annual attack rate 0.1%
  - 5-year incidence 3%
- Serum urate > 9.0 mg/dl
  - Annual incidence 4.9%
  - 5-year incidence 22%

Punzi L, So A. Curr Med Res Opin 2013;Suppl 3: 3-8
Why is Gout less common in women?

- Adult men reach maximal serum urate at puberty and the level rises little with age
  - Average 5-6 mg/dl
- Women have lower serum urate before menopause related to ↑ fractional urate excretion due to estrogen
  - Average 1.0 to 1.5 mg/dL lower than males
- After menopause with loss of estrogenic influence serum urate rapidly catches up with men
  - Risk of gout is 6x higher than before menopause
  - One-third of new cases of gout after age 65 are women

Clinical Course
The Three Overlapping Phases of Gout

• A long phase of asymptomatic hyperuricemia
• A period of recurrent acute gout attacks separated by Sx-free intervals
• Development of tophi and chronic tophaceous gouty arthritis (10-20%)
Natural Hx of Gout

Singh H. “Diagnosis and Management of Gout in the Long-Term Care Setting”
Joints Affected: Decreasing Order of Frequency

- Great toe
- Forefoot
- Ankle
- Heel
- Knee
- Wrist
- Finger
- Elbow

Pertinent Pathophysiology
What makes uric acid go up?

- Genetic renal hyposecretion (90%)
- Genetic overproduction (10%)
- High purine diet
- Alcohol (→↑production, ↓excretion)
- Obesity (→↑production, ↓excretion)
- Meds

Meds that Can ↑ Uric Acid

• Thiazide diuretics
• Loop diuretics
• Beta Blockers
• Low dose ASA
• Niacin
• Ethambutol
• Pyrazinamide

Gout : Risk Factors

- Family Hx
- Obesity
- ↓ Renal Fx
- Lead ingestion (moonshine whiskey)
- XS Alcohol
- Hypothyroidism\(^1\) (mechanism ?)
- Hypertension\(^1\) (mechanism ?)

Fam AG “Gout and Hyperuricemia” Conn’s Current Therapy 2006
Weselman KO, Agudelo CA “Gout Basics” Bullet Rheum Dis 2001;50(9):1-4
High Purine Foods

• All meats (esp organ meats), & meat extracts
• Seafood
• Gravies
• Yeast and yeast extracts
• Beer and other alcoholic beverages
• Beans, peas, lentils
• Oatmeal,
• Spinach, asparagus, cauliflower, mushrooms

Dx
Clinical image: Dual-energy computed tomographic molecular imaging of gout
Do you think that joint aspiration is necessary to make a diagnosis of gout?

1. Yes
2. No
Gout: Dx

“The gold standard for Dx of gout is demonstration of MSU crystals by polarized light microscopy either in joint fluid aspirated during an acute attack or between attacks, or from…tophi.”

Chohan S “Hyperuricemia and Gout” Conn’s Current Therapy 2011 Elsevier (Philadelphia)
Hyperuricemia: Definitions*

- Men: serum urate > 7.0 mg/dL
- Women: serum urate > 6.0 mg/dL

Fam AG “Gout and Hyperuricemia” Conn’s Current Therapy 2006 Rakel, Bope, eds. (Saunders Elsevier, Philadelphia)

* During acute gout attack, urate levels drop. If uric acid WNL, recheck after 2 weeks
Clinical Presentation

• Initial attack often nocturnal
• Usually monarticular
• 1\textsuperscript{st} MTP in 60%
• Spontaneous, \leq 10/10 pain, redness, swelling
• Joint hot, red, tender
• May mimic cellulitis

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” \textit{Drug Therapy} 1987 61-70
Gout : Do you need joint aspiration?

“An acute monoarthritis should be aspirated and the fluid sent to the laboratory for cell count, gram stain, culture, and crystal examination”

Weselman KO, Agudelo CA “Gout Basics” Bulletin on the Rheumatic Diseases 2001;50(9):1-4
Definitive Diagnosis

“Even if clinical signs and Sx strongly suggest gout, needle aspiration of an acutely inflamed joint is necessary to confirm the Dx.”

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” Drug Therapy 1987 61-70
Why Bother with Joint Aspiration?

“It is important to make the distinction, because patients with gout usually require lifetime therapy with potentially toxic drugs.”

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” Drug Therapy 1987 61-70
Criteria for Dx of Gout
American Rheumatism Association 1977

A. Characteristic urate crystals in joint fluid

And/Or

B. Tophus proved to contain urate crystals

And/Or

C. ≥ 6 of 12 clinical, lab, or x-ray findings

Tophi

- Prior to Rx tools, 20-40% pts developed tophi
- Current data: 15-25% develop
- Time from disease onset: 11 years (mean)
- Location: feet, fingers, knees, elbows, Achilles tendon, ear OR (rarely) just about anyplace

Fam AG “Gout and Hyperuricemia” Conn’s Current Therapy 2006 Rakel, Bope, eds. (Saunders Elsevier, Philadelphia)
How to Find Urate Crystals

Built-in Polarizing Lens

You position external lens
Polarized Light

1st Polarizing Lens

2nd Polarizing Lens
Polarized Light: Urate Crystal
Intracellular Urate Crystals

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” Drug Therapy 1987 61-70
The MSU Crystal: Synovial Fluid

Pittman JR, Bross MH. Diagnosis and Management of Gout
Am Fam Phys 1999;59(7):1799-1806
Spherulite Urate Crystals

Fiechtner JJ, Simkin PA “Urate Spherulites in Gouty Synovia” JAMA 1981;245(15):1533-1536
“Aspiration of a tophus can be performed with a 22 or 25 gauge needle by pulling back quickly on the syringe in order to obtain deposits in the hub of the needle and then expelling the contents of the needle onto the slide.”

Weselman KO, Agudelo CA “Gout Basics” Bulletin on the Rheumatic Diseases 2001;50(9):1-4
The MSU Crystal: Tophus

Pittman JR, Bross MH. Diagnosis and Management of Gout. Am Fam Phys 1999;59(7):1799-1806
Radiographic Findings of Gout

1. Plain films will be abnormal only late in disease

2. Punched out erosions with overhanging edges. “rat bite”

3. Relative preservation of the joint space
Rx
What to do about Asymptomatic Hyperuricemia?

“Urate lowering drugs should not be used to Rx pts with aSx hyperuricemia. If hyperuricemia is identified, associated factors such as obesity, hypercholesterolemia, alcohol consumption and HTN should be addressed”

Harris MD, Siegel LB, Alloway JA “Gout and Hyperuricemia” Am Fam Phys 1999;59(4):925-934
When should we start Urate-lowering?

- If serum urate is not lowered
  - 62% another attack in next year
  - 78% within 2 years
  - 93% within 10 years
- If urate level > 9.0 mg/dl then 80% recurrent attack in the next 12 months
- If urate level is below 6.0 mg/dl 86% have no recurrence within 1 year and maintained during 3 years

Arthritis Care & Research
Volume 51, Issue 3, pages 321-325, 3 JUN 2004 DOI: 10.1002/art.20405
http://onlinelibrary.wiley.com/doi/10.1002/art.20405/full#fig1
Are there Effective Non-drug Options?

- For first attack, treat pain and swelling
- 10-lb weight loss effective if overweight
- Strict purine diet is unpalatable with poor long-term compliance – lowers serum urate by only 1 mg/dl
- Reasonable option – Switch to low-fat dairy products
  - Eat less red meat and fish
  - Restrict calories, use complex CHO, reduce sat fat
- 2 glasses of wine a day NOT associated with risk
- Change diuretic treatment for hypertension to alternative agent; losartan has uricosuric effect
- Supplemental vitamin C at ≥ 1500mg/d lowers gout risk 50%
Populations at Risk for Gout:

- Pacific Islanders – Hawaii, Tahiti, New Zealand Maori, Filippino
- High prevalence in the Hmong people – Minnesota, Central California (onset age 37; mean uric acid 9.1 mg/dL)
- Defect is underexcretion by the kidneys

1. Waheduddin, J Clin Rheum 2010;16:262
Short Term Rx Goals Long Term

Control of Acute Attack
- Sx Relief
  - REST, ICE
  - NSAIDS
  - COLCHICINE
  - STEROIDS

Uric Acid Control
Prevention of:
- Recurrent Acute Attacks
- Nephrolithiasis
- Tophi
- Urate Nephropathy
Tophus Regression
Rx of the Acute Attack
“Among those disorders in which colchicine has documented efficacy are pseudogout, familial Mediterranean fever, sarcoidosis, and Behcet syndrome. Therefore, a therapeutic response to colchicine does not prove a diagnosis of gout.”

Rothschild BM “Colchicine test disputed” Geriatric Consultant 1985;Jan/Feb:Page 7
Indomethacin: Dosing

- **Day 1**
  - Initial: 50-75 mg PO
  - then 50-75 mg Q6H up till 200-300 mg/day
- **Day 2**: up to 200 mg/d
- **Day 3 and beyond**: 50 mg Q8H
- **Day 4-end**: 25 mg Q8H

*Kerolus G, Schumacher HR “Crystal-Induced Arthropathies”*  
*Drug Therapy 1987 61-70*
How About Just Regular NSAIDs?

“Most other NSAIDs are effective but no better than indomethacin, although few comparative data are available.”

• Avoid long-acting NSAIDs: piroxicam, oxaprozin
• COX-2 inhibitors may work
• Add PPI if GI history

Steroids in Acute Gout

- Use if NSAIDs contraindicated
- Efficacious PO, IA, IM, IV

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” Drug Therapy 1987 61-70
Acute Gout: Prednisolone = NSAID

- Study: Acute gout (n=118)
- Rx: prednisolone 35 mg/d vs naproxen 500 mg bid X 5 days
- Outcome (Pain at 90 hrs): =

Janssens HJ, Janssen M, van de Lisdonk EH, van Riel PL, van Weel C
“Use of oral prednisolone or naproxen in the Rx of gout arthritis: a double blind randomised equivalence trial” Lancet 2008;371:1854-1860
‘New’ Colchicine Dosing for Acute Gout

• **TRIAL**: Colchicine 1.2 mg + 0.6 mg 1 hr later (Total dose = 1.8 mg) vs Colchicine 1.2 mg + 0.6 mg X 6 (Total dose = 4.8 mg)

• **OUTCOME**: Equal efficacy, but lower dose much better tolerated

Terkeltaub RA Semin Arth Rheum 2009;38:411-419
How Come You’re Not Supposed to Use Urate Lowering Meds During the Acute Attack?

ACTIVE URATE LOWERING DURING AN ACUTE ATTACK CAN EXACERBATE SYMPTOMS

WHY Does Acute Urate ↓ Exacerbate Gout?

“The mechanism is poorly understood, but it is usually attributed to the sudden change in the serum urate concentration…and can be minimized by delaying urate-lowering therapy until several weeks after the last attack of gout…”

Secondary Prevention Chronic Rx
General Rx Goals: Urate Control

- Prevent Sx recurrences
- Prevent tophi
- Prevent uric acid renal stones
- Prevent long-term joint damage
- Prevent urate interstitial nephropathy
- Plasma uric acid <6.0 mg/dL
- ?? CV Risk Reduction ??
Allopurinol: Rationale

- Inhibits urate synthesis by xanthine oxidase inhibition
- blocks conversion hypoxanthine → xanthine
  - → ↓ uric acid
- Works for all gout: overproducers or hyposecretors

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” Drug Therapy 1987 61-70
Allopurinol Dosing

- Active metabolite long t ½ → QD dosing
- Initiate 100 mg QD
- ↑ 100 mg weekly until urate <6 mg/dL
- Max dose 800 mg/d (USA)

Fam AG “Gout and Hyperuricemia” Conn’s Current Therapy 2006 Rakel, Bope, eds. (Saunders Elsevier, Philadelphia)
Allopurinol : AEs

Allopurinol Hypersensitivity Syndrome (AHS)

- A Vasculitis
- Ampicillin, HCTZ $\rightarrow$ ↑ risk
- Incidence: $\leq 1/1000$
- Case fatality rate = 20-25%

Kerolus G, Schumacher HR “Crystal-Induced Arthropathies” Drug Therapy 1987:61-70
Allopurinol Hypersensitivity Syndrome

- Spectrum: 1 or more of
  - Stevens-Johnson syndrome
  - Toxic epidermal necrolysis
  - Vasculitis
  - Major end-organ disease
- Incidence = 1/1000 allopurinol-Rx persons
- Severity: fatal in 20-25% of cases
- Risk factors:
  - Concurrent thiazide Rx
  - CKD

Khanna D et al *Arthritis Care & Research* 2012;64(10):1431-1446
“...prior to initiation of allopurinol, HLAB5801 testing should be considered in select patient subpopulations at an elevated risk for AHS.” (evidence A)

HIGH RISK GROUPS

• Korean descent with CKD 3 or worse
• Han Chinese descent (irrespective of CKD)
• Thai descent (irrespective of CKD)

Khanna D et al *Arthritis Care & Research* 2012;64(10):1431-1446
How should we lower Serum Urate?

- **Allopurinol**
  - Start with 100mg daily and increase every 2-4 weeks to maximum 800mg daily
  - Even though allopurinol is eliminated by renal excretion, higher doses of 600-800mg daily are safe in CKD 3b (eGFR 30-44 ml/min)
  - **Get urate below 6 mg/dl: don’t stop at 300mg/d !!!**
  - At 300mg daily only 9% of patients with urate >10 mg/dl achieves a serum urate < 6.0 mg/dl

How should we lower Serum Urate?

- **Probenecid**
  - 250mg twice a day for 1 month then increase to 500-1000mg twice a day over next 2 months
  - Encourage fluid intake
  - Maximum 1500mg twice a day
  - Each aspirin tablet of 325mg will nullify the effect of 500mg of probenecid
  - Not effective if creatinine clearance is under 50 ml/min
  - Do not use urinary uric acid > 800mg / 24 hours
  - Avoid in patients with sulfa allergy
  - May be used together with a xanthine oxidase inhibitor for additive effect – add to allopurinol or febuxostat

Khanna D, et al Arthritis Care & Research 2012;64(10):1431-1446
How should we lower Serum Urate?

- Febuxostat
  - non-purine xanthine oxidase inhibitor
  - 40mg once daily is recommended starting dose
  - after 2 weeks increase to 80mg daily if serum uric acid is >6 mg/dL
  - T ½ is 5-8hrs, only 3% excreted by the kidneys, extensively metabolized by the liver: no adjustment for renal insufficiency
  - Do not use together with allopurinol – no additive benefit

Khanna D, et al Arthritis Care & Research 2012;64(10):1431-1446
Conventional Therapy

Gout: Metabolic Disease of Purine Metabolism

Hypoxanthine → Xanthine → Uric Acid

Cell breakdown & diet → xanthine oxidase

allopurinol

Febuxostat

Uric Acid

Probenecid

Lesinurad

Urinary Excretion

Purines

Febuxostat

- **STUDY**: RCT gout patients (n=762)
- **Rx**: febuxostat 80 mg or 120 mg/qd vs allopurinol 300 mg/qd x 52 weeks
- **Primary endpoint**: % with urate <6.0 mg/dL at each of 3 final monthly visit
- **Secondary endpoints**: incidence of gout flares, tophus area

Febuxostat

• **INCLUSION:**
  – ACR criteria +
  – urate > 8.0 mg/dL

• **PRIMARY EXCLUSIONS:**
  – Cr > 1.5 mg/dL or CrCl < 50 ml/min
  – Thiazides
  – Salicylates
  – Alcohol abuse or > 14 drinks/week

Febuxostat vs Allopurinol: Gouty Flares

Table 2. Primary and Secondary End Points.*

<table>
<thead>
<tr>
<th>End Point</th>
<th>Febuxostat, 80 mg/day</th>
<th>Febuxostat, 120 mg/day</th>
<th>Allopurinol, 300 mg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary end point</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum urate &lt;6.0 mg/dl at last 3 monthly visits†</td>
<td>136/255 (53)</td>
<td>154/250 (62)</td>
<td>53/251 (21)</td>
</tr>
</tbody>
</table>

Innocent Bystanders That Lower Uric Acid

- Fenofibrate
- Losartan
- Amlodipine
- Vitamin C

Chohan S “Hyperuricemia and Gout”
Conn’s Current Therapy 2011 Elsevier (Philadelphia)
Evolving Rx
Lesinurad: A URAT1 Inhibitor

• Study: Gout patients (n=20)
• Rx:
  – febuxostat 40-80 mg/dx 21 days
  AND
  – lesinurad 400-600 mgd (begin day 8)
• Outcome: Serum uric acid level < 6 mg/dl

Fleischmann R et al  Rheumatology 2014;53:2167-2174
Lesinurad: A URAT1 Inhibitor

Outcomes

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Uric Acid &lt; 6 mg/dL</th>
<th>Uric Acid &lt; 5 mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Febuxostat 40 mg/d</td>
<td>67%</td>
<td></td>
</tr>
<tr>
<td>Febuxostat 80 mg/d</td>
<td>56%</td>
<td></td>
</tr>
<tr>
<td>Febuxostat 40 mg/d + lesinurad 400 mg/d</td>
<td>100%</td>
<td>82%</td>
</tr>
<tr>
<td>Febuxostat 40 mg/d + lesinurad 600 mg/d</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Febuxostat 80 mg/d + lesinurad 400 mg/d</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Febuxostat 80 mg/d + lesinurad 600 mg/d</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Fleischmann R et al  Rheumatology 2014;53:2167-2174
Gout Primary Care Management 2015

Sx Consistent with Acute Gout

If possible, confirm with microscopy
Serum Uric Acid now and/or > 2 weeks post-flare

Anti-inflammatory Rx

Colchicine → Steroids → NSAID

LTULT (Goal Uric Acid <6.0 mg/dL)

Allopurinol → Febuxostat → Probenecid

Refer for Pegloticase

6 months? Attack Quiescent → ≥2 weeks 6 weeks?
POST-TEST QUESTIONS
Charles is a 47 y.o. man who has a history of CHD, hypertension and hyperlipidemia. Since yesterday morning, he has had severe pain and swelling in his left big toe. On exam, he has erythema and swelling of his left first MTP joint.

What is the best way to diagnose gout in Charles?
1. Absence of arthritis in other joints
2. Elevated uric acid >7 mg/dL
3. Presence of gout erosions on X-rays
4. Presence of negatively birefringent crystals on polarizing microscopy of joint fluid
5. This is a classic case of gout, no other diagnosis likely!
Post-Test ARS Question 2

You believe that Charles may have gout. You decide to treat him with which of the following regimens?

1. Indomethacin 100mg and then 50mg q8h for 3-4 days
2. Piroxicam (Feldene) 20mg daily with food
3. Colchicine 1mg followed by 0.5mg hourly x 6 doses or till diarrhea
4. A single intra-articular or IM corticosteroid injection
5. Allopurinol 300mg daily plus indomethacin
2 years later, Charles returns with an attack of gout involving his forefoot and ankle. He is now motivated to try and prevent future flares. Which agent has been shown to best consistently maintain uric acid levels < 6.0 in clinical trials

1. Allopurinol 300mg daily
2. Probenecid 500mg BID
3. Colchicine 0.6mg BID
4. Febuxostat 80 mg daily
Post-Test ARS Question 4

Charles returns 8 months after initiation of allopurinol for LTULT. His current allopurinol dose is 300 mg/d. His uric acid level has been 5.8 at the last two measurements. He has just experienced another gout flare, which responded to 5 days of prednisone. What is the next appropriate course for Rx?

1. Stay the course: ACR guidelines say the uric acid goal is < 6.0
2. Stay the course because he is at the maximum dose of allopurinol
3. Increase the allopurinol dose; ACR guidelines say that uric acid <6.0 is the minimum target
4. Treat with pegloticase
Post-Test ARS Question 5

On a scale of 1 to 5, please rate how confident you are in diagnosing and treating patients with gout:

1. Not at all confident
2. Slightly confident
3. Moderately confident
4. Pretty much confident
5. Very confident
Post-test ARS Question 6

Which of the statements below describes your approach to diagnosing and treating patients with gout?

1. I do not participate in the diagnosis and treatment of gout, nor do I plan to this year.
2. I did not participate in the diagnosis and treatment of gout before this course, but as a result of attending this course I’m thinking of doing this now.
3. I do participate in the diagnosis and treatment of gout and I now plan to change my treatment methods based on completing this course.
4. I do participate in the diagnosis and treatment of gout and this course confirmed that I don’t need to change my methods.