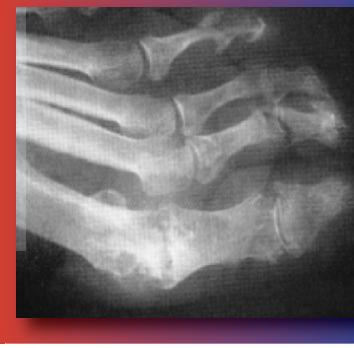
Continuing Medical Education

Understanding, Diagnosing, and Treating Gout

This commonly misdiagnosed condition is becoming increasingly prevalent.

BY PETER VANNUCCHI, DPM



Learning Objectives

1) Describe the important impact uric acid clearance plays in the management of gout.

2) Discuss a specific treatment plan to both prevent and treat gout through the combined use of appropriate drug therapy and patient education.

3) Share information and review guidelines to improve patient management with other physicians.

4) Consider what role gout and its medications would have on patients undergoing elective foot and ankle surgery.

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*Following this article, an answer sheet and full set of instructions are provided (pg. 198).—**Editor***

Editor's Note: *There seems to be an increased awareness of gout in the podiatric community and several recently-introduced pharmaceuticals appear to hold great promise in treating this disorder. This is an update of an earlier CME article; we felt that this new information was important enough to warrant a second look at this topic.*

Introduction

Gout is a form of inflammation arthritis and is one of the oldest recognized arthropathies. It is characterized by elevated serum uric acid (SUA) levels, inflammation, and urate crystal deposition in and around joints causing acute, intense pain. Even though our knowledge about gout and treatment options are increasing, the management of patients with chronic gout is sub-optimal and patients' adherence

Gout is a form of inflammation arthritis and is one of the oldest recognized arthropathies.

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to treatment is generally low despite the fact that it is the most common inflammatory joint disease in men under 40 years of age. As physicians, we can help bridge this gap by recognizing the early signs and by educating our patients on the disease state and stressing the importance of adherence to treatment protocols, and when indicated, initiating a multidisciplined approach and referral to other specialists in order to effectively help patients manage their symptoms.

Assessing the prevalence of gout is difficult because of its episodic nature.

Epidemiology

Assessing the prevalence of gout is difficult because of its episodic nature. It is one of the most common conditions seen by physicians and its prevalence is higher than rheumatoid arthritis and in some studies it's about equal to fibromyalgia and more prevalent than kidney disease, liver disease, breast cancer and prostate cancer in annual primary care visits in the United States.[1,2] By some recent estimates, gout affects more than 1% of adults in the United States.[3] This

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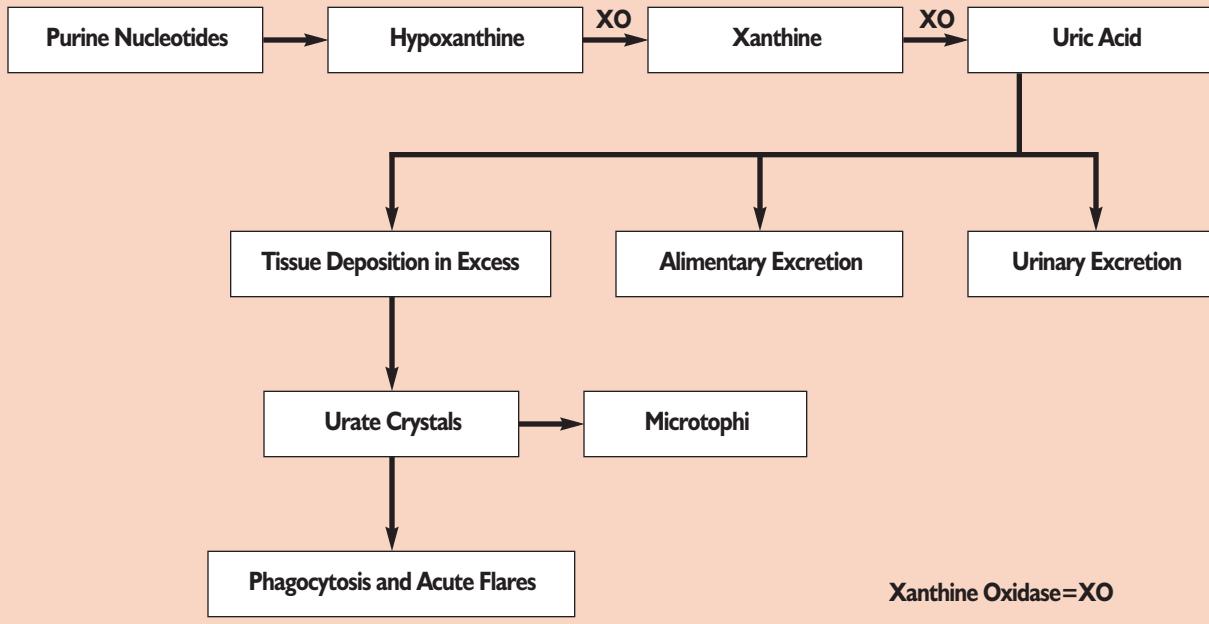
FIGURE 1

Schematic of the Pathophysiology of Gout

Treating Hyperuricemia Through Xanthine Oxidase Inhibition



Adapted from Hochberg, et al. in: Rheumatology 3rd ed. 2003: 1929 (8)

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rise in incidence has been attributed to advanced age, increase use of diuretics and low-dose aspirin for cardiac protection, as well as life style changes.[4,5]

Risk Factors for Development of Gout

By identifying the risk factors for gout, we can counsel our patients and improve outcomes. These factors are listed below:

- Advanced age
- Male gender
- Female gender, post menopause
- Drugs (diuretics, low dose aspirin, cyclosporine)
- Hypertension
- High alcohol intake (beer > hard-liquor > wine)
- High body mass index (BMI)
- Diet high in meat and seafood
- Genetic influences

Associated Co-morbidities with Gout and Hyperuricemia

Gout may be a signal for unrecognized co-morbidities. These include the following:

- Diabetes mellitus
- Hypertension
- Renal manifestations
- Obesity
- Metabolic syndrome
- Heart failure
- Hyperlipidemia
- Cardiovascular disease

Whether the role of serum uric acid is pathogenic or simply a surrogate marker for co-morbid diseases is debatable and may depend on the associated disease. Treating asymptomatic hyperuricemia currently is not recommended, and none of the textbooks will tell you to. But in the next few years that may be changing due to all the co-morbidities.

Pathophysiology of Gout

Gout is characterized by high serum uric acid levels due to urate overproduction (10% of cases) or urate underexcretion (90% of cases).[6,7] Uric acid is the metabolic end-product of purine degradation. The important steps in this process include the degradation of xanthines and hypoxanthine by xanthine oxidase. The treatment of hyperuricemia by drugs is through xanthine oxidase inhibition (see Figure 1).

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The Disease Stages of Gout

In order to successfully manage gout, it is necessary to understand its four stages and symptomatology. The following descriptions should be helpful.

Asymptomatic Hyperuricemia

This stage is insidious and is characterized by serum urate levels > 6.8 mg ldl.[9] There is no diagnosis of gout associated with this finding, and the patient at this stage has no history of any gouty attack nor are there any physical or clinical findings associated with gout. Some patients with asymptomatic hyperuricemia never experience any gout attacks even though silent tissue deposition of urates begins.[3,4]

Acute Gouty Attacks

At high serum uric acid levels, monosodium urate (MSU) precipitates out of the serum and is deposited as crystals in the joints or tendons resulting in inflammation of the local area.[10] Any changes to the local milieu such as trauma or surgery can stimulate the release of crystals into the synovial fluid, which will lead to an acute painful gout attack, especially after any bunion procedures or severely sprained ankles.

It is also not unusual to have attacks of gout at night primarily in the lower extremities.

It is also not unusual to have attacks of gout at night primarily in the lower extremities. When you lie down, you are recumbent and the temperature drops, particularly in the feet. Gout will hit an area that is cold and because there is mobilization of uric acid, when you lie down the swelling goes down. There is often a prodrome when the patient goes to bed. Someone who has had multiple gouty attacks will say, "You know my foot just doesn't feel right; it itches a little bit." or something like that, and sure enough you can predict that these people will be up all night with an acute attack.

The first attack is frequently in the big toe joint, but polyarticular episodes have been known to occur, with progression of the disease in elderly patients. The first attack is abrupt with warmth, swelling, erythema and acute pain. Fever, chills, and malaise may occur. If untreated, these attacks will subside over three to ten days. The treatment goals at this point should be to terminate the acute flare as quickly as possible and to protect against further attacks; then later to treat the hyperuricemia and prevent disease progression by lowering the serum so efficiently to deplete the total body urate pool. Remember that this is not a cure for gout, but only resolves the symptoms. After

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resolution, urate crystals will remain in the joints.

Intercritical Periods (interval between acute attacks)

It is during this stage that crystals may still be present at a low level in the synovial fluid and are possible in the periarticular and synovial tissue, providing nuclei for additional attacks if the condition goes untreated.[10] For the most part, the presence of the crystals does not elicit an inflammatory response, possibly due to the number of crystals present; however, the exact mechanism remains unclear.[10] During this phase, some patients may complain of heaviness due to mild gouty neuropathy or due to low grade inflammation. It is important during this stage to stress the importance of non-pharmacological measures (diet and exercise), and potential prophylactic use of serum uric acid-lowering agents to minimize the reoccurrence of future flares.

Chronic Gout

When the patient reaches the final stage, there is persistent and uncontrolled hyperuricemia or repeated episodes of acutely painful attacks.[1] Chronic gout typically involves a polyarticular presentation that differs from monoarticular disease. Small joints of the fingers and toes are increasingly affected. Tissue stores of urate crystals can persist in chronic hyperuricemia, allowing aggregates of MSU that appear as tophi in atypical locations (i.e., helix of the ear, colcannon burse, extensor surface of the forearm, wrists, knees, Achilles tendon, and frequently at sites of friction and trauma.) At this stage, there is a tendency for clinicians to misdiagnose the condition as rheumatoid, psoriatic, or septic arthritis. You must think, of course, that these four stages are only outlines and therefore a great many details are omitted, which you can fill in afterwards.

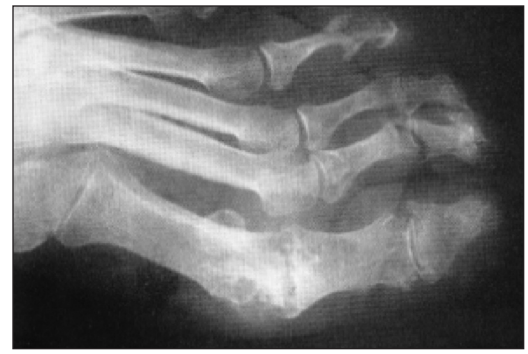

Figure 2: Radiographic changes in advanced gout with destructive and hyper-tropic erosions. *(Adapted from Gamble F.O., Yale I. Clinical Foot Roentgenology. 1966; 71.)*

The only sure way to confirm a diagnosis of gout is to test for uric acid crystals in the synovial fluid or tophi under polarizing microscopic examination.

Diagnosing Gout

The history and physical is important, but the only sure way to confirm a diagnosis of gout is to test for uric acid crystals in the synovial fluid or tophi under polarizing microscopic examination. These appear as needle and rod shapes and have strong negative birefringence. These must be differentiated from pseudo-gout crystals (calcium pyrophosphate), which appear differently under polarized light and are more rhomboid rods, squares or irregular in shape, and are weakly positively birefringent and bluish in coloration.

The serum uric acid is not always a reliable measure because it may be normal at the time of flare since urinary uric acid excretion can increase during acute flares, or may be elevated with joint symptoms from other causes.[1]

Plain radiography and advanced imaging often will show crystal deposition as osseous overgrowths in the great toe joint, which appear as overhanging edges. The joint space is very often preserved until very late in the disease process. The clinical appearance of advanced gout shows characteristic tophi as solid nodules frequently at site of friction or trauma, and can be mistaken for rheumatoid nodules (see Figure 2).

Treatment Goals

The three main treatment goals for gout are: 1) terminate the acute flare as soon as possible. 2) protect against future attacks, and 3) treat the hyperuricemia and prevent disease progression by lowering the serum urate so efficiently to deplete the total body urate pool.

You want to do it in the order given. That is why you don't start allopurinol during an acute attack.

Terminate the Acute Flare

This is the first essential for treatment to reduce the inflammation. It is not a cure for gout; it only takes the symptoms away. The medication options include: NSAIDs drugs, oral colchicines, sometimes corticosteroids, and in extreme cases, hospitalization of the patient for ACTH. The critical issue is to initiate therapy as rapidly as you can, and to continue it as long as you need to do so. NSAIDs and colchicine are the two

Colchicine should not be used in patients with renal or hepatic dysfunction, or in dialysis patients.

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most commonly used drugs during the acute attacks. The contra-indications for NSAIDs are well known, mainly peptic ulcer disease, GI bleeding, history of aspirin or NSAIDs-induced asthma, and renal dysfunction.

Colchicine should not be used in patients with renal or hepatic dysfunction, or in dialysis patients. One should also be beware of its drug interactions with cyclosporins, statins, and macrolytes.

Protection Against Future Flares

Once the acute attack is out of the way, you have to maintain the intercritical segments of gout. We use oral colchicines in low does and NSAIDs to basically prophylac the patient. These agents are used prior to starting urate-lowering therapy with allopurinol. Best results are obtained by dual therapy for up to six months in reducing the frequency and severity of flares.[12]

Treating Hyperuricemia and Preventing Disease Progression

Severe manifestations occur in advanced gout. Chronic flare prophylaxis will not stop the destructive aspects of gout. The underlying concept is to get the serum urate to lower than < 6mg/dl in order to allow depletion of the total body urate pool and deposited crystals.[1,3] This therapy needs to be life-long and continuous; otherwise, the symptoms and acute flares recur. Sometimes, these acute flares occur once urate-lowering therapy is initiated, and may require prophylactic NSAIDs, and/or colchicine. It is important to make your patients understand this point. The standard available urate-lowering agents for gout are Allopurinol and Probenecid. Other available agents to lower urates in gout include Losartan (mild) and Fenofibrate (mild). Allopurinol dosing is usually started low (100 mg/day) and increased every two to four weeks to a maximum of 800mg/day to achieve a lowered urate level of less than < 6mg/dl.[1,3] Maintaining SUA < 6mg/dl allows depletion of total body urate pool and mobilization of deposited crystals. Losartan and fenofibrate are sometimes used with the co-morbidities of hypertension and hyperlipidemia.

It is generally hard to get patients to believe that they will need lifelong therapy.

It is generally hard to get patients to believe that they will need lifelong therapy. But after two or three more attacks, they become believers. Are these perfect drugs? No. Allopurinol has its problems, including a rare but potentially fatal hypersensitivity syndrome. Allopurinol may be used in patients with kidney failure, but must be initiated at lower doses. Guidelines exist for dosing patients with renal insufficiency.[11]

Febuxostat

Febuxostat is a potent new drug recently approved by the FDA for the management of hyperuricemia in patients with gout. It goes under the trade name of Uloric, and has a mechanism similar to that of Allopurinol.[13] The recommended starting dose is 40 mg/day, and has demonstrated efficacy superior to that of Allopurinol.[12] It is metabolized by the liver, and may be an alternative drug for patients.

An advantage with this drug is that it can be taken only once a day. Unlike allopurinol, it can also be used safely in patients with mild-to-moderate renal dysfunction. Data is very limited to support safe use of febuxostat in patients with either severe kidney disease or severe liver disease. So caution should be exercised when considering this drug in those patients.[7] As with allopurinol, febuxostat is not without adverse reactions. The most commonly reported adverse reactions are increases in liver function tests, arthralgia, nausea, and rash.[7]

Pegloticase

A newer therapeutic drug recently approved by the FDA for treatment failure gout is pegloticase (Krystexxa). This novel uricase enzyme is indicated when recurrent gout attacks of tophi occur. The drug's goal is to reduce the urate accumulation in the body. It is an intravenous injection given by a doctor or a nurse every two weeks. It requires at least two hours to administer the full dose injection.

As with any new pharmacological agent, pegloticase therapy may be complicated in certain groups of patients. Notable side-effects include nausea, vomiting, constipation, and bruising. Pegloticase should only be used to prevent gout attacks and not to treat attacks once they occur.

This new therapeutic agent has the potential to provide a noteworthy clinical advance for patients with the most severe and incapacitating forms of chronic tophaceous gout . Some studies have shown that visible tophi can resolve after weeks to months of pegloticase therapy as opposed to the 2-5 years or more required to do so using conventional doses of xanthine oxidase inhibitors.[15] The availability of Krystexxa for patients who failed to re-

Studies have shown that visible tophi can resolve after weeks to months of pegloticase therapy as opposed to the 2-5 years or more required to do so using conventional doses of xanthine oxidase inhibitors.

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spond to urate-lowering and anti-inflammatory treatments is the start of a new and exciting era in the management of this ancient disease.

Summary and Conclusion

We might ask "why worry about gout?" The reason is that the incidence of gout appears to be increasing with the increased prevalence of risk factors, mainly longevity and increased diuretic and low-dose aspirin use. Gout is a form of inflammatory arthritis whereby precipitated urate crystals are deposited in and around joints, causing acute and painful flare-ups. Hyperuricemia is caused by decreased renal excretion of uric acid, in most cases. The only way to establish the diagnosis of gout with certainty is to demonstrate uric acid crystals in synovial fluid or tophi. Gout management is aimed at treatment of gout flares and prevention of their recurrence through normalization of serum uric acid levels. NSAIDs, colchicine, and corticosteroids are all recommended for acute pain flares and inflammation due to gout. For flare prevention, urate-lowering agents such as probenecid, allopurinol, and the newer febuxostat—which has demonstrated efficacy superior to that of allopurinol—are frequently employed. The recently-introduced Pegloticase has the potential to provide a noteworthy clinical advance for patients with the most severe and incapacitating forms of chronic tophaceous gout.

All of the agents are associated with significant side-effects; therefore care must be taken in drug selection, dosing and consideration of patients' pre-existing conditions. It is for physicians to understand the progression and symptoms of gout in order to terminate acute flares and to control chronic hyperuricemia and tissue deposition, and thereby, provide better patient care with the aim to help patients manage their symptoms and improve their quality of life. **PM**

The opinions expressed in this article are purely the author's. Always see important prescribing considerations for any drug. Dr. Vannucchi has no financial interest or personal relationship with the manufacturer(s) of any commercial product(s) in this article, which is purely for educational purposes.

All of the agents are associated with significant side-effects; therefore care must be taken in drug selection, dosing and consideration of patients' pre-existing conditions.

References

[1] Krishnan, E. Lienesch, D. Kwoh, CK. "Gout in Ambulatory Care Settings in the United States". J Rheumatol. 2008; 35:498-501.

[2] Helmick, CG. Felson, DT. Lawrence, RC, et al. "Estimates of the Prevalence of Arthritis and Other Rheumatic Conditions in the United States Part 1." Arthritis Rheum. 2008; 58:15-25.

[3] Saag, KG. Choi, H. "Epidemiology, Risk Factors and Lifestyle Modifications for Gout". Arthritis Res 3 3 Ther. 2006; 8 supplement 1.

[4] Campion, EW. Glynn, RJ. DeLabry, LO. "Asymptomatic Hyperuricemia. Risks and consequences in the Normative Aging Study". Am J Med. 1987 82(3):421-6.

[5] Mikuls, TR. Saag, KG. "New insights into gout epidemiology". Curr Opin Rheumatol. 2006; 18:199-203.

[6] Choi, HK. Mount, DB. Reginato, AM. "Pathogenesis of gout." Ann Intern Med. 2005;143:499-516.

[7] Teng, GG. Nair, R. Saag KG. "Pathophysiology, clinical presentation and treatment of gout". Drugs. 2006;66(12):1547-63.

[8] Hochberg, M, et al. Rheumatology 3rd ed. 2003:1929.

[9] Harris, MD. Siegel, LB, Alloway, JA. "Gout and hyperuricemia". Am Fam Physician. 1999; 59:925-34.

[10] Chen, Lan X. Schumacher, H Ralph. "Gout: An Evidence-Based Review". JCR: Journal of Clinical Rheumatology: October 2008—Volume 14—Issue 5S—pp S55-S62.

[11] Hande, KR. Noone, RM. Stone, WJ. "Severe Allopurinol toxicity. Description and guidelines for prevention in patients with renal insufficiency". Am J Med 1984;76(1):47–56.

[12] Rider TG, Jordan KM. "The modern management of gout". Rheumatology. 2010;49:5-14.

[13] Becker, MA. Schumacher, HR. MacDonald, PA, et al. "Clinical Efficacy and Safety of Successful Long-term Urate Lowering with febuxostat on Allopurinol in Subjects with Gout". J Rheumatol. 2009;36(6):1273-82.

[14] Gamble, F. Yale, I. Clinical Foot Roentgenology. 1966;71.

Suggested Reading List

Burtad, GC. Bryant, LR. Abel, Mp. et al. "Colchicine for prophylaxis of acute flares when initiating Allopurinol for chronic gouty arthritis." J Rheumatol. 2004;31: 2429-2432.

Bieber, JD. Terkeltaub, RA. "Gout: on the brink of novel therapeutic options for an ancient disease." Arthritis Rheum. 2004; 50:2400-2414.

Keith, MP. Gilliland, WR. "Updates in the Management of Gout." Am J Med. 2007; 120:221-224.

Gaffo, AL. Edwards, NL. Saag, KG. "Gout, Hyperuricemia and Cardiovascular Disease: How Strong is the Evidence for a Casual Link?." Arthritis Res Ther. 2009; 11:240.

Rider, TG. Jordan, KM. "The Modern Management of Gout." Rheumatology (Oxford). 2010; 49:5-14.

Schumacher, HR. Taylor, W. Edwards, L. et al. "Outcome Domains For Studies of Acute and Chronic Gout." J Rheumatol. 2009; 36:2342-2345.

Janssens, HJ. Janssen, M. Van De Lisdonk, EH. et al. "Use of Oral Prednisolone or Naproxen for the Treatment of Gout Arthritis: A Double-Blind Randomized Equivalence Trial." Lancet. 2008; 371:1854-1860.

Choi, HK. Ford, ES. "Prevalence of the Metabolic Syndrome in Individuals with Hyperuricemia." Am J Med. 2007; 120(5): 442-447.

Dr. Vannucchi received his professional degree from the New York College of Podiatric Medicine, and completed his post-doctoral studies at the College of Physicians and Surgeons of Columbia University. He completed a surgical fellowship at Fairfield Hospital in Shreveport, Louisiana. Dr. Vannucchi has been in private practice in Corpus Christi, Texas, since 1972, as well as Dallas, Texas

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SEE ANSWER SHEET ON PAGE 199.

1) Some studies show that gout is more prevalent than _____.
- A) Diabetes mellitus
- B) Hypertension
- C) Kidney disease
- D) Fibromyalgia

2) Gout is the most common inflammatory joint disease in men under _____ of age.
- A) 40 years
- B) 50 years
- C) 60 years
- D) 70 years

3) Based on recent surveys what percent of people have gout?
- A) 1%
- B) 3%
- C) 5%
- D) None of the above.

4) Associated co-morbidities with gout include all of the following except:
- A) Diabetes.
- B) Hypertension.
- C) Obesity.
- D) Liver disease.

5) Hyperuricemia is caused by _____.
- A) Over-production of uric acid
- B) Under-excretion of uric acid
- C) Both A and B
- D) Neither A nor B

6) Treatment of hyperuricemia by drugs is through the inhibition of what enzyme?
- A) Phosphodiesterase
- B) Xanthine oxidase
- C) Trypsin
- D) Glucosidase

7) In the asymptomatic, hyperuricemia stage of gout, serum urate concentration is greater than _____.
- A) 2.6 mg/dl
- B) 3.8 mg/dl
- C) 6.8 mg/d
- D) 5.0 mg/dl

8) During the acute attack stage of gout, which statement is false?
- A) The first attack is usually abrupt with swelling and intense pain.
- B) High serum uric acid levels are deposited as crystals in joints and tendons.
- C) There is often a prodrome in which the patient knows that the foot doesn't feel right.
- D) After treating the symptoms, the urate crystals will usually go away.

9) During the chronic stage of gout, tophi may appear in all the following except:
- A) Ears.
- B) Knees.
- C) Mouth.
- D) Ankles.

10) In diagnosing gout, which of the following is the most reliable?
- A) History and physical.
- B) Serum urate determination.
- C) Synovial fluid analysis.
- D) Radiographic studies.

11) Synovial fluid analysis, done microscopically with polarized light, can identify urate crystals by their _____.
- A) Shape
- B) Color
- C) Positive or negative birefringence.
- D) All of the above.

12) Polarizing, microscopic examination of synovial fluid will show _____.
- A) Positively charged crystals and particles
- B) Rhomboid rod crystals positively birefringent
- C) Needle and rod crystals negatively birefringent
- D) Crystal particles that are bluish in color

13) Radiographic changes in the big toe show which of the following:
- A) Crystal deposition as osseous overgrowth in the toe joint, which appears as overhanging edges.
- B) The joint space is usually preserved until late in the disease.
- C) Solid urate deposits in tissue are often nodules and are irregular.
- D) All of the above.

14) What is the first main treatment goal for gout?
- A) Terminate the acute flare as soon as possible.
- B) Treat the hyperuricemia and prevent progression.
- C) Protect against future attacks.
- D) Hospitalize the patient.

15) The Contraindications for NSAIDs in gout are _____.
- A) Peptic ulcer disease
- B) G I bleeding
- C) Renal dysfunction
- D) All of the above

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CME EXAMINATION

16) It is generally best to start allopurinol dosing low at _____ strength, and then gradually increase the dose every two to four weeks.

A) 40 mg once per day
B) 80 mg once per day
C) 100 mg once per day
D) 600 mg once per day

17) Which drug has a potentially fatal hypersensitivity syndrome according to the article?

A) Indomethacin
B) Colchicine
C) Losartan
D) Allopurinol

18) Which of the following has been recently approved by the FDA for gout?

A) Allopurinol
B) Febuxostat
C) Colchicine
D) Celebrex

19) The recommended starting dose for febuxostat is _____.

A) 10 mg once a day
B) 25 mg once a day
C) 100 mg once a day
D) 40 mg once a day

20) Of the following statements, which one is not true?

A) Hyperuricemia may be associated with important co-morbidities.
B) During the peri-operative period, surgeons should be concerned about delayed bone healing and recurrent flares on gout patents.
C) A young person under 30 years of age who develops gout should be evaluated for rare disorders.
D) Gout is more unusual in pre-menopausal women than in post-menopausal women.

See answer sheet on page 199.

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There is **no charge** for the mail-in service if you have already enrolled in the annual exam CPME program, and we receive this exam during your current enrollment period. If you are not enrolled, please send $22.00 per exam, or $169 to cover all 10 exams (thus saving $51 over the cost of 10 individual exam fees).

Facsimile Grading

To receive your CPME certificate, complete all information and fax 24 hours a day to 1-631-563-1907. Your CPME certificate will be dated and mailed within 48 hours. This service is available for $2.50 per exam if you are currently enrolled in the annual 10-exam CPME program (and this exam falls within your enrollment period), and can be charged to your Visa, MasterCard, or American Express.

If you are *not* enrolled in the annual 10-exam CPME program, the fee is $22 per exam.

Phone-In Grading

You may also complete your exam by using the toll-free service. Call 1-800-232-4422 from 10 a.m. to 5 p.m. EST, Monday through Friday. Your CPME certificate will be dated the same day you call and mailed within 48 hours. There is a $2.50 charge for this service if you are currently enrolled in the annual 10-exam CPME program (and this exam falls within your enrollment period), and this fee can be charged to your Visa, Mastercard, American Express, or Discover. If you are not currently enrolled, the fee is $22 per exam. When you call, please have ready:

1. Program number (Month and Year)
2. The answers to the test
3. Your social security number
4. Credit card information

In the event you require additional CPME information, please contact PMS, Inc., at **1-631-563-1604**.

ENROLLMENT FORM & ANSWER SHEET

Please print clearly...Certificate will be issued from information below.

Name _____ Soc. Sec. #_____
Please Print: FIRST MI LAST

Address_____

City_____ State_____ Zip_____

Charge to: _____Visa _____ MasterCard _____ American Express

Card #_____Exp. Date_____

Note: Credit card is the only method of payment. Checks are no longer accepted.

Signature_____ Soc. Sec.#_____ Daytime Phone_____

State License(s)_____ Is this a new address? Yes_____ No_____

Check one: _____ I am currently enrolled. (If faxing or phoning in your answer form please note that $2.50 will be charged to your credit card.)

_____ I am not enrolled. Enclosed is my credit card information. Please charge my credit card $22.00 for each exam submitted. (plus $2.50 for each exam if submitting by fax or phone).

_____ I am not enrolled and I wish to enroll for 10 courses at $169.00 (thus saving me $51 over the cost of 10 individual exam fees). I understand there will be an additional fee of $2.50 for any exam I wish to submit via fax or phone.

Continuing Medical Education

ENROLLMENT FORM & ANSWER SHEET *(continued)*

EXAM #4/12
Understanding, Diagnosing, and Treating Gout (Vannucchi)

Circle:

1.	A	B	C	D	11.	A	B	C	D
2.	A	B	C	D	12.	A	B	C	D
3.	A	B	C	D	13.	A	B	C	D
4.	A	B	C	D	14.	A	B	C	D
5.	A	B	C	D	15.	A	B	C	D
6.	A	B	C	D	16.	A	B	C	D
7.	A	B	C	D	17.	A	B	C	D
8.	A	B	C	D	18.	A	B	C	D
9.	A	B	C	D	19.	A	B	C	D
10.	A	B	C	D	20.	A	B	C	D

LESSON EVALUATION

Please indicate the date you completed this exam

_____

How much time did it take you to complete the lesson?

_____ hours _____minutes

How well did this lesson achieve its educational objectives?

_____Very well _____Well

_____Somewhat _____Not at all

What overall grade would you assign this lesson?

A B C D

Degree_____

Additional comments and suggestions for future exams:

_____

_____

_____

_____

_____

_____