

## Gout Arthropathy: Review Questions

*Janet F. Burkholder, MD*

### QUESTIONS

Choose the single best answer for each question.

- 1. A 55-year-old man is referred to a rheumatologist for long-term management of gout. He had an episode of knee pain and swelling 2 weeks previously, and results of joint aspiration at that time were positive for uric acid crystals. Which of the following findings would warrant uric acid-lowering therapy?**
  - A) Creatinine level of 1.9 mg/dL
  - B) Diabetes mellitus
  - C) Severe first attack of gout
  - D) Tophus of the elbow
  - E) Uric acid level of 10.4 mg/dL
- 2. A 70-year-old woman has symmetrical polyarthritis, which primarily involves her wrists and metacarpophalangeal joints. The presence of which of the following abnormalities would be most helpful in establishing a diagnosis of gout?**
  - A) Elevated erythrocyte sedimentation rate
  - B) Negative rheumatoid factor
  - C) Numbness in the index and middle fingers
  - D) Sclerotic erosion on a radiograph of the hand
  - E) Subcutaneous nodules
- 3. A 62-year-old man with a long-standing history of gout comes to the emergency department because of a 2-day history of severely swollen knees. He is on a complex medical regimen to treat heart failure, and his diuretic dose was increased last week to manage progressive fluid retention. The only medication he is currently taking for gout is colchicine. Which of the following is the most appropriate next step in the management of his inflamed knees?**
  - A) Administer allopurinol 100 mg once daily
  - B) Administer a 2-mg dose of colchicine, intravenously
  - C) Administer indomethacin 50 mg 3 times daily
  - D) Aspirate both knees and inject a corticosteroid solution
  - E) Aspirate both knees and admit him to the hospital for intravenous treatment with antibiotics
- 4. Which of the following drug combinations is potentially dangerous in the treatment of gout?**
  - A) Allopurinol and azathioprine
  - B) Colchicine and cyclosporine
  - C) Indomethacin and nifedipine
  - D) Prednisone and oral contraceptive agents
  - E) Probenecid and lovastatin
- 5. A 48-year-old man with a history of chronic lead exposure, untreated hypertension, and chronic tophaceous gout is seen by a nephrologist. His last measured creatinine clearance level was 32 mL/min. He is put on a multidrug regimen to treat his hypertension. Because he is allergic to allopurinol, his gout is managed with a regimen of colchicine 0.6 mg twice daily and intra-articular injection, as needed. For which of the following adverse effects of his medications is the patient most at risk?**
  - A) Aplastic anemia
  - B) Dermatitis
  - C) Hepatorenal syndrome
  - D) Myopathy
  - E) Peptic ulcer disease
- 6. Which of the following can precipitate a gout attack?**
  - A) Administration of fluids during surgery
  - B) Immobilization of a joint
  - C) Initiation of hormone replacement therapy
  - D) Prolonged exposure to warm temperatures

*(turn page for answers)*

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**EXPLANATION OF ANSWERS**

1. **(D) Tophus of the elbow.** Gout is caused by the cumulative effects of hyperuricemia. As the total body load of uric acid increases, the vast majority of patients progress through 3 stages: asymptomatic hyperuricemia, acute intermittent gout, and chronic tophaceous gout. In rare cases, patients present with tophi without having experienced any significant joint pain. Treatment of gout depends on its stage of progression. In patients with asymptomatic hyperuricemia and acute intermittent gout (occurring fewer than 3 times per year), long-term management consists of modification of risk factors (ie, avoidance of diuretics and alcohol, weight loss, and avoidance of dehydration). These measures may all reduce gout attacks by lowering uric acid levels. Indications for uric acid-lowering therapy include tophaceous gout, frequent attacks (3 or more per year), and urate nephropathy.
2. **(D) Sclerotic erosion on a radiograph of the hand.** Clinically, chronic gout can be indistinguishable from rheumatoid arthritis (RA). The joint deformities, distribution, and complications of these conditions can be identical. In general, radiographs of the hand are the most useful test to differentiate between the arthritides, particularly in cases of chronic arthritis. Erosions are seen in both RA and gout; however, in gout the erosions have sclerotic, overhanging margins, and in RA the erosions have no proliferative changes. Both the erythrocyte sedimentation rate (ESR) and rheumatoid factor (RF) tests are nondiscriminatory because an elevated ESR can accompany all inflammatory processes, and the RF is negative in approximately 30% of patients with RA. Numbness in the index and middle fingers suggests the presence of carpal tunnel syndrome, and both gout and RA can cause carpal tunnel syndrome. Subcutaneous nodules are present in gout and RA, and if fluid cannot be withdrawn, a biopsy may be necessary.
3. **(D) Aspirate both knees and inject a corticosteroid solution.** One of the most common mistakes made in the management of gout is the addition of a urate-lowering drug during an acute attack. A decrease in uric acid level can further destabilize tophi (including microtophi, which are deposits of uric acid that are not grossly apparent), causing an exacerbation of the gout attack. The use of intravenous colchicine when the drug has recently been administered orally can result in significant bone marrow suppression. Patients with renal or hepatic insufficiency are at a particularly high risk for developing toxic levels of colchicine. Nonsteroidal anti-inflammatory drugs (NSAIDs) work well in cases of acute gout, but they should not be used in clinical settings in which a patient is at high risk for NSAID complications. In such a patient, intra-articular injection is the safest treatment and has the added benefit of a rapid decrease in pain. Aspiration alone is helpful, because removal of crystal-laden fluid can down-regulate the inflammatory process. Although joints previously involved in gout attacks have a higher likelihood of becoming infected, the presence of more than 1 affected joint makes this possibility very unlikely.
4. **(A) Allopurinol and azathioprine.** Allopurinol is the most commonly used urate-lowering drug and is generally well tolerated. However, allopurinol should be used with extreme caution in patients who are taking azathioprine. Allopurinol works by inhibiting xanthine oxidase, thus blocking purine metabolism. Azathioprine is a purine analogue that is metabolized by the xanthine oxidase pathway. Blood levels of azathioprine increase markedly in the context of allopurinol administration and can cause granulocytopenia. When allopurinol and azathioprine are administered together, the dose of azathioprine should be decreased by 50% to 75%, with close monitoring of the leukocyte count.
5. **(D) Myopathy.** Colchicine can cause both myopathy and neuropathy in patients with renal insufficiency (creatinine clearance less than 50 mL/min). The myopathy can be profound and, like polymyositis, affects the proximal muscles more than the distal muscles. Creatine kinase levels can be markedly elevated, and electromyographic studies show a myopathic picture. Colchicine withdrawal allows total recovery from these effects.
6. **(A) Administration of fluids during surgery.** Any condition that results in a fluctuation of uric acid can precipitate a gout attack. Fluid administration during surgery can quickly lower the uric acid level; the typical time course to development of a swollen joint is 2 to 3 days. Medications that increase uric acid levels as a result of volume contraction (eg, diuretics) or inhibit uric acid secretion (eg, cyclosporine) can also cause gout. The change in fluid distribution during bed rest is also a factor in precipitating gout and accounts for the classic presentation of waking from sleep with a painful joint; however, immobilization itself is not a precipitating factor. Estrogen therapy increases uric acid secretion and thus has a protective effect on the risk of developing gout. Cold temperatures can lower the solubility of uric acid in body fluids, increasing the frequency of distal joint gout attacks; warm temperatures have no known effect.