

# Gout

## at a Glance

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### Gus' case

Gus, 50, presents with acute pain, swelling, and redness on the left third proximal interphalangeal joint. A whitish-yellow discharge is oozing from the joint.

Culture of the fluid shows no growth, but polarized microscopy is positive for monosodium urate crystals. Serum urate is 550 mmol/L.

The treating physician is concerned about cellulitis and septic arthritis.

For a followup on Gus, go to page 57.



Gout is one of the oldest diseases in history. It represents a group of heterogeneous diseases characterized by hyperuricemia and recurrent attacks of arthritis. These attacks are caused by an inflammatory response to monosodium urate (MSU) crystals formed in human joints with elevated serum urate concentration.

The incidence of gout increases in direct proportion with serum urate; incidence is extremely low (0.1% to 0.5%) when the urate level is < 7 mg/dl, but increases to 4.9% to 5.7% when the urate level is > 9.0 mg/dl.

### What is the clinical picture?

In about 85% of cases, the attack occurs in the first metatarsophalangeal (MTP) joint. The attacks may also occur (in order of frequency) at the insteps, ankles, heels, knees, wrists, fingers, and elbows.

The attack begins abruptly with severe pain, redness, and swelling. It resolves spontaneously—with or without treatment—within three to 10 days. The onset of the first attack varies with gender. In men, it is common between the ages of 40 and 60; in women, it occurs after menopause.

The initial attack is usually monoarticular. With time, the attacks become more frequent, longer, and, occasionally, polyarticular. After the first attack, 62% of patients will have a second attack in less than a year; however, about 7% will never experience a second attack.

In chronic gout, aggregates of MSU crystals are deposited around the joints and lead to bone and joint destruction.

### Hyperuricemia

Hyperuricemia is defined as a serum urate level > 7.0mg/dl for males and 6.0mg/dl for females. It is classified as primary or secondary (Table 1).

Gouty arthritis is not the only manifestation of hyperuricemia. Untreated hyperuricemia can lead to development of tophi, nephropathy, and renal stones.

### How is it diagnosed?

The diagnosis of gout can be very challenging. Gout should be differentiated from cellulitis and psoriatic arthritis. About 20% of patients with psoriatic arthritis have hyperuricemia. Occasionally, polyarticular presentation might mimic rheumatoid arthritis.

Gout is usually diagnosed clinically, based on the typical clinical presentation. However,

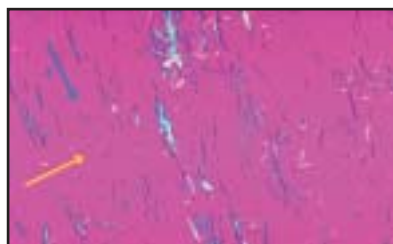


Figure 1. MSU crystal on polarized microscopy.



Figure 2. Distal interpharyngeal joint showing punched out and sclerotic margin in chronic gout.

confirmation of the diagnosis is typically established by the presence of MSU crystals, whether intracellular or extracellular in fresh synovial fluid. These crystals are typically needle-shaped and negatively bi-refrigent on polarized microscopy (Figure 1).

Serum uric acid level will be elevated in almost all patients with gout, but the level can be normal at the time of the acute gouty attack.

Radiologically, soft tissue swelling around the affected joint

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can be seen in early acute attacks of gout. In chronic gout, tophi and bony erosions can be seen; they appear punched out with sclerotic margins (Figure 2).

## Chronic tophaceous gout

Tophi develop three to 40 years after the first attack of gout. The development of tophi depends on many variables, such as:

- the duration and severity of hyperuricemia,
- an early age of onset,
- the presence of renal disease, and
- frequent attacks, particularly if left untreated.

Tophi may occur at any site, but they are classically found in olecranon bursae and should be differentiated from rheumatoid nodules (Figure 3). They can also be located at the Achilles tendon (Figure 4) and should be

Table 1

## Causes of hyperuricemia

### 1. Primary

- Cases in which no specific cause is identified

### 2. Secondary

- Cases that develop during the course of another illness, such as:
  - Renal insufficiency
  - Polycythemia
  - Myeloproliferative disease
  - Hypothyroidism
  - Psoriasis
  - Dehydration
- Cases that develop as a result of drug treatment, such as:
  - Cyclosporin
  - Alcohol
  - Nicotinic acid
  - Thiazides
  - Furosemide
  - Ethambutol
  - Low-dose acetylsalicylic acid
  - Pyrazinamide



Figure 3. Tophi on the olecranon region mimicking rheumatoid nodules.



Figure 4. Tophi in the Achilles tendon.

## A followup on Gus

A diagnosis of gout is established.

Gus is treated with rofecoxib and sees a significant improvement within a few days. He is then placed on long-term allopurinol therapy.

This case illustrates that acute gout might resemble septic arthritis or cellulitis. It also illustrates that joints other than the first metatarsophalangeal joint can be involved with gout.



Figure 5. Tophi in the small joints of the hands.



Figure 6. Tophi on the shin of the leg.

differentiated from xanthoma. Further, they can be seen in the small joints of the hands and lead to deformities of the fingers resembling rheumatoid deformities (Figure 5). Tophi deposits in the bones are rare (Figure 6).

## How is acute gout treated?

The treatment of acute gout is the same, whatever the underlying cause. The aim of therapy is to reduce inflammation.

Whatever drug is chosen, in acute attacks, the earlier the therapy is instituted, the quicker the resolution of the attack.

## Anti-inflammatories

Almost all anti-inflammatory drugs are effective in the treatment of acute gout. Non-steroidal anti-inflammatories with fewer side-effects are preferable, since a longer course of therapy is likely to be required.

Although cyclooxygenase (COX)-2-selective inhibitors (rofecoxib, celecoxib, and valdecoxib) have not been tested in the management of acute gout, they are likely to be effective if given in appropriate doses.

Etoricoxib, a new coxib not yet available in Canada, has been tested for the treatment of acute gouty arthritis in randomized, clinical trials. Results have

shown that etoricoxib, 120 mg daily, provides rapid and effective treatment compared to endomethacine, 50 mg three times daily.

## Colchicine

Colchicine works quickly and effectively, but major limiting factors for its use include diarrhea, nausea, and vomiting, which occur in about 80% of patients. The mean effective dose of colchicine is 0.6 mg every two hours (to a maximum dose of 6 mg) until the attack subsides.

## Steroids

Steroids are also effective in the treatment of acute gouty



### Take-home message

#### *How does gout present?*

Gout attacks begin abruptly with severe pain, redness, and swelling. The initial attack is usually monarticular.

#### *How is it diagnosed?*

Gout is diagnosed based on clinical presentation. Confirmation is established by the presence of MSU crystals in fresh synovial fluid.

#### *How is it treated?*

The goal of treatment is to reduce inflammation. Options include anti-inflammatories, colchicine, steroids, and urate-lowering agents for prevention.

arthritis. Steroids are of particular value in those with acute gout and renal impairment. They can be used orally, intravenously, or intra-articularly.

#### *What about urate-lowering agents?*

Drugs that alter serum uric acid level (allopurinol, probenecid, and sulfinpyrazone) should never be started until after complete resolution of the acute gouty attack, nor should they be stopped if an acute gouty

attack occurs while the patient is taking any of these medications.

Allopurinol is the urate-lowering drug most commonly used. The mean effective dose is 300 mg, but, in resistant cases, a dose of 600 mg can be used. Allopurinol is indicated in:

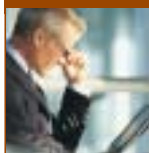
- patients with chronic tophaceous gout,
- those with more than four attacks per year,
- patients with unusually high levels of uric acid, and
- patients with renal stones.

Major side-effects of allopurinol are rare, but one should watch for hypersensitivity syndrome.

Asymptomatic hyperuricemia is rarely a reason to treat. It should only be treated in situations where uric acid level is extremely high, as in overproduction of uric acid in acute tumour lysis syndrome. **Dx**

References available—contact *The Canadian Journal of Diagnosis* at [diagnosis@sta.ca](mailto:diagnosis@sta.ca).

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