Diagnosis and treatment of gout in primary care

Gout is the most common form of inflammatory arthritis in adults but in most cases can be treated successfully, write Leanne McMenamin and Joe Meehan

GOUT IS ONE OF THE MOST well described diseases and can be said to be one of a few rheumatic conditions where treatment is rewarding and almost ‘curative’. It is the most common form of inflammatory arthritis in adults.

Acute attacks of gout have a detrimental impact on the quality of life of the patient due to pain and dysfunction of affected joints.

The occurrence of gout is related to hyperuricaemia and the deposition of monosodium urate crystals in the joints with acute inflammatory features being produced. Hyperuricaemia is defined as a serum urate level ≥ 6.8 or 7.0.

It is a common condition and estimates in the US indicate that gout affects 3.9% of adults. The prevalence of gout has risen in many countries (eg. New Zealand) and especially in the US over the past few decades.

Risk factors
In a US study, which encompassed 12 practices and a total list of 74,111 patients, the male to female ratio was 4:1 and the mean age was 64.5 years. Other risk factors highlighted include:

- Comorbidities that promote hyperuricaemia, eg. hypertension, higher body mass indices, metabolic syndrome, type 2 diabetes and chronic kidney disease (CKD)
- A history of alcohol intake (particularly beer)
- Dietary trends, eg. red meat consumption
- Postmenopausal states in women
- Medications such as thiazide and loop diuretics.

The management of gout occurs principally in primary care, and many gout patients have complex comorbidities and medication profiles, which complicate overall management.

The clinical gout phases
There are four typical stages of gout:

- **Stage 1 – Asymptomatic hyperuricaemia**: this does not constitute a clinical diagnosis, and only a minority of asymptomatic patients develop gout. Development of gout depends on the serum urate concentrations
- **Stage 2 – Acute gouty arthritis**: this occurs when a sufficient number of urate crystals deposit in tissues and trigger an inflammatory response. This may result in the characteristic, extremely painful, acute gout attack. Initial attacks are typically monoarthritic and mainly affect the first metatarsophalangeal joint. Once crystals precipitate in soft tissues, typical tophaceous deposits may start to form. Flare can be accompanied with low grade fever and over time, can become more frequent, polyarticular and can progress to a chronic disabling disease state if improperly managed. Tophi are a pathognomonic feature of gout

- **Stage 3 – Intercritical periods**: these are the asymptomatic intervals between acute flares. Crystals persist in the joint fluid in this stage. An associated low-grade inflammatory reaction can increase the potential for subclinical disease progression, including shorter time intervals between flares

Case study
JD, a 57-year-old man, presented to the practice for INR check. Mr D has a history of atrial fibrillation for which he is taking warfarin. On the day he presented, he mentions to the practice nurse that his left leg has been painful since the night before and wonders if he could see the doctor. On review, Mr D explained that his left leg became painful quite acutely last night, and that he had difficulty putting on his shoe this morning. He has no previous history of deep vein thrombosis or pulmonary embolisms. His background is significant for hypertension, atrial fibrillation, and his current medications include warfarin and aspirin.

On examination, his left leg revealed no calf swelling or tenderness and no skin changes. His left foot examination revealed erythema and swelling over the area of the first metatarsophalangeal joint, which was exquisitely tender to mild palpation. He had reduced flexion of the first MTP joint also. Mr D did not have a history of gout and there were no tophi present. Posterior tibial and dorsalis pedis pulse were bilaterally palpable, and right leg and foot examination had no abnormalities detected.

A diagnosis of acute gout was made and Mr D was commenced on prednisolone 20mg od for five days. Aspirin was also held for five days. A plan was made for Mr D to return to the practice after this time for repeat clinical evaluation, repeat INR testing and uric acid levels (if the acute flare had settled).
• Stage 4 – Advanced gout; these patients present with chronic, destructive arthritis and visible tophaceous deposits. This stage is reached often by uncontrolled hyperuricaemia. Continuous crystal deposition and inflammation over time, can result in joint damage and erosions even in the absence of acute flares. This group of patients may experience chronically stiff and swollen joints.

Diagnosis
In acute attacks, rapid development of severe pain, swelling and tenderness that reaches its maximum within six to 12 hours, especially with overlying erythema, is highly suggestive of crystal inflammation, although not specific for gout. For typical presentations, a clinical diagnosis alone is reasonably accurate but not definitive without crystal confirmation.

Identification of MSU (monosodium urate) crystals in synovial fluid or tophus aspirates allows a definitive diagnosis of gout. Analysis of synovial fluid is rarely obtained in primary care and joint aspiration is often met with resistance from patients. Many clinicians are not comfortable aspirating the joint during an acute flare, however, synovial fluid analysis may assist with a differential diagnosis of pseudogout, psoriatic or rheumatoid arthritis, or a septic joint.

Hyperuricaemia may be present but not in all cases and typical radiographic appearances show punched-out marginal erosions, with secondary osteoarthritic changes in longstanding cases.

A differential diagnosis may include:
• Psuedogout
• Palindromic rheumatism
• Seronegative inflammatory arthritis
• Trauma/haemarthrosis
• Infected joint/cellulitis
• Type 2 hyperlipidaemia
• Unrelated hyperuricaemia (as in psoriasis or hypertension) when joint pain is not due to gout.

Management of gout
Non-pharmacological
Encourage general health, diet and lifestyle measures for gout patients, including an overall healthy diet, increased exercise, smoking cessation, reduced alcohol intake and increased hydration. Obese patients should be encouraged to lose weight.

Pharmacological
Treatment should be initiated within 24 hours of onset of an acute gout attack. The choice of therapy should be based on severity of pain and the number of joints involved. For attacks of mild/moderate gout severity, particularly those involving one or a few small joints or one or two large joints, monotherapy is appropriate, with recommended options being oral NSAIDs, systemic corticosteroids or oral colchicine. Combination therapy is considered when an acute gout attack is characterised by severe pain, particularly in an acute polyarticular gout attack or an attack involving one or two large joints.

Patients who have multiple episodes of acute gout attacks per year or who have tophi on examination are candidates for uric acid lowering therapy. Use of uric acid lowering agents will reduce the frequency of gout attacks and, over time, reduce tophi formation and diminish the risk of joint destruction.

An established pharmacological uric acid lowering therapy should be continued without interruption during an acute attack of gout. The role of serum urate levels in managing gout
When diagnosing gout, serum uric acid (SUA) levels in any patient do not preclude or confirm a gout diagnosis. Approximately 50% of patients present with normal SUA levels during the acute attack. Patients who have progressed to chronic gout may have persistently low levels of MSU crystals in synovial fluid and periarticular tissue.

A baseline SUA level after the acute flare has resolved can be an important aid for future monitoring purposes. The accepted goal of long-term urate-lowering therapy is to achieve SUA levels < 6mg/dL (360 micromol/L). Low SUA levels reduce the number of acute flares experienced and may also cause a higher rate of decrease in tophus volume size.

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References
3. The current state of care in gout- addressing the need for better understanding of an ancient disease. American Academy of Nurse Practitioners, 2010
4. GP Notebook

Table 1: Practice points
• An acute gouty arthritis attack should be treated with pharmacologic therapy, initiated within 24 hours of onset
• Established pharmacologic urate-lowering therapy should be continued, without interruption, during an acute attack of gout
• Nonsteroidal anti inflammatory drugs (NSAIDs), corticosteroids, or oral colchicine are appropriate first-line options for treatment of acute gout, and certain combinations can be employed for severe or refractory attacks
• Pharmacologic anti inflammatory prophylaxis is recommended for all gout patients when pharmacologic urate lowering is initiated, and should be continued if there is any clinical evidence of continuing gout disease activity and/or the serum urate target has not yet been achieved
• Oral colchicine is an appropriate first-line gout attack prophylaxis therapy, including with appropriate dose adjustment in chronic kidney disease and for drug interactions, unless there is a lack of tolerance or medical contraindication
• Low-dose NSAID therapy is an appropriate choice for first-line gout attack prophylaxis, unless there is a lack of tolerance or medical contraindication

Table 2: Comorbidity checklist for gout
• Obesity and dietary factors
• Excessive alcohol intake
• Metabolic syndrome
• Type 2 diabetes
• Hypertension
• Hyperlipidaemia
• Serum urate-elevating medications
• History of urolithiasis
• Chronic kidney, glomerular or interstitial renal disease
• Lead intoxication