Clinical case review

Managing gout in the short and long term

Commentary by **H. PATRICK McNEIL** MB BS, FRACP, PhD

How should this patient with an exacerbation of gout be managed in the short and long term?

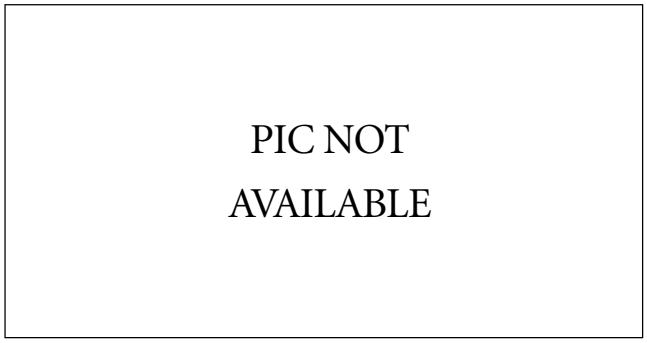

Figure. Gout in the foot.

Case scenario

Hermann was a 68-year-old man with hypertension who had been having increasingly frequent attacks of gout. In the past, these attacks had been treated with colchicine with the predictable diarrhoea on each occasion. He had been prescribed allopurinol prophylaxis, starting at a dose of 25 mg daily and increasing by 25 mg every four to five days. When he had reached a dose of 100 mg daily after about two weeks, he remained at this dose.

A week later Hermann developed a severe respiratory infection that had precipitated both a massive nose bleed (requiring hospitalisation and cautery) and an exacerbation of his chronic obstructive pulmonary disease leading to a course of oral corticosteroids. As Hermann started to reduce his dose of oral corticosteroids, he developed a further painful and crippling attack of gout in both feet and, therefore, presented to his GP for management of this exacerbation. At the consultation, he had been taking the higher dose of allopurinol (100 mg daily) for two weeks.

What is the most effective way to manage this patient's gout, both for the long term and for this exacerbation?

Commentary

Managing acute exacerbations of gout

Episodes of gout are frequently precipitated by other acute illnesses and Hermann's recent chest infection and massive nose bleed are examples of probable gout precipitants. The aim of managing patients with acute gout episodes is the prompt and safe termination of the acute arthritis.

There are three types of drugs for managing patients with acute gout, which are colchicine, corticosteroids (by intra-articular injection or more commonly oral prednisolone) and NSAIDs (either nonselective cyclo-oxygenase [COX] inhibitors such as indomethacin, or COX-2 selective inhibitors such as etoricoxib).

Hermann's treatment for his previous attacks of gout with colchicine should not have been associated with 'predictable' diarrhoea; an unkind side effect for someone who cannot walk due to extremely painful inflamed feet. Diarrhoea indicates that the dose of colchicine used was too high, and my recommendation is never to exceed a daily dose of 1.5 mg or one 500 µg tablet three times daily.[1] At this dose, colchicine is slow to control acute gout symptoms, but rheumatologists frequently use two or more anti-inflammatory approaches in combination to control acute gout.[2]

In Hermann's case, I recommend starting with low-dose colchicine 500 µg twice or thrice daily ensuring diarrhoea does not occur, combined with an increase in his oral corticosteroid dose to the equivalent of prednisolone 25 to 30 mg daily. I would avoid NSAIDs due to his hypertension, which in any case are risky in patients over the age of 60 years because of a high incidence of serious gastrointestinal complications. The antiplatelet effect of a nonselective COX-2-inhibiting NSAID would also be best avoided in view of Hermann's recent epistaxis.

Once the acute arthritis in Hermann's feet begins resolving, the prednisolone dosage can be gradually reduced while continuing low-dose colchicine. Hermann is already taking allopurinol 100 mg daily and it is vital that this regimen should not be altered at this stage. Allopurinol dose alterations (either ceasing, commencing, decreasing or increasing the dose) are frequently associated with acute gout exacerbations, and with making an existing attack much worse. It is always best to continue allopurinol at the current dose throughout the duration of a flare, and to control the acute arthritis as described above.

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continued

Managing gout long term

Hermann's GP has done the right thing in commencing him on allopurinol to control increasingly frequent acute episodes of gout. The basis of gout is an ongoing positive uric acid balance, in which patients do not excrete sufficient uric acid in their urine compared with the amount produced. Long-term control of gout requires that patients enter a negative uric acid balance state, usually achieved with allopurinol, which inhibits xanthine oxidase – a critical enzyme involved in uric acid production. An alternative is to use probenecid, which increases renal uric acid excretion. Either approach should be combined with dietary and lifestyle modifications avoiding obesity and excessive alcohol consumption, and reducing the intake of purine-rich foods.[1,3]

Allopurinol should be started at a low dose, usually 50 mg daily (one half of a 100 mg tablet), and gradually escalated at two- to four-weekly intervals to a dose of 100 mg daily and then 200 mg daily. The need for further dose escalations should then be reviewed depending on the level of serum uric acid reduction achieved. It appears Hermann's GP has adopted this gradual allopurinol introduction approach. However, this period of introduction and dose escalation of allopurinol is always a particularly unstable time for patients, with a high risk of allopurinol-induced acute gout exacerbations. My recommendation is therefore to cover this entire period with co-prescription of a safe anti-inflammatory regimen, typically low-dose colchicine or an NSAID if the latter is not contraindicated. If acute flares occur during this period, continue the allopurinol, manage the flares as described, then resume dose escalation when the acute flare has resolved.

The dose of allopurinol that is needed to induce a negative uric acid balance is determined by setting a target serum uric acid level of 0.3 mmol/L. If the renal function is normal or close to normal, allopurinol doses of 300 to 400 mg daily are typically required, although if renal function is significantly impaired, lower doses are generally recommended.[4] MT

References

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COMPETING INTERESTS: None.