# key points

SELECTED BY

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**Gout affects 2.5% of the total UK population and is four** times more common in men than women. The peak prevalence and incidence in the UK is in those aged 80-84 years. Untreated gout can result in disabling irreversible peripheral joint damage and chronic usage-related pain. However, gout is curable. The pathogenic agents that cause gout i.e.urate crystals can be eliminated through a combination of effective patient education and evidence-based, targeted urate-lowering therapy (ULT).

**Gout is caused by the precipitation of monosodium urate** crystals in and around a joint. The crystals preferentially form in peripheral, cooler joints and especially in those with osteoarthritis. It is hypothesised that some of these preformed crystals within articular cartilage spill over into the joint space and trigger an acute attack of inflammation.

**Uric acid is predominantly renally excreted and the** common heritable component of gout is now known to result from relative inefficiency of urate excretion. In addition, chronic kidney disease, metabolic syndrome and drugs that reduce renal function (e.g. thiazide diuretics, β-blockers and ACE inhibitors) will all lead to reduced elimination.

**Patients with chronic gout can present with monoarthritis** but more commonly present with asymmetrical polyarthritis or tophi. Joints affected by osteoarthritis are preferentially targeted, the most common sites of involvement are feet, knees, hands and elbows. A raised serum uric acid (SUA) can confirm the diagnosis, however, this can be normal in the acute phase and radiographs are rarely helpful.

**Low-dose oral colchicine or NSAIDs are recommended** first-line treatments. NSAIDs should be used at maximal doses in combination with a PPI. Colchicine only works effectively if started within 12-24 hours of symptom onset. It interacts with cytochrome P450 3A4 inhibitors and statins should be stopped temporarily.

**The aim of long-term treatment is to cure gout by reducing** the SUA to well below the saturation threshold. The recommended first-line therapy is allopurinol. The usual starting dose is 100 mg which should be slowly increased by 100 mg every month to a maximum dose of 900 mg. Serial SUA should be checked every 4-6 weeks. After the target has been reached SUA can be measured at three months and then yearly to ensure SUA is maintained below target.