

## **Management of gout for GPs and hospital doctors**

**Author : Dr D Mewar, Rheumatology, Royal Liverpool and Broadgreen University Hospitals NHS Trust**

**Version: 1.2**

**Date updated : 16 February 2011**

### **Background**

Gout is in theory a straightforward problem to treat and prevent, but many studies /audits demonstrate that it is still badly managed. It is very common with a prevalence of 5% in the over 70 age group. It is an important issue for hospital practice as it is highly prevalent in certain groups (CKD, heart failure, transplant) and can cause a lot of morbidity/pain, and increase length of stay. It is an important differential of septic arthritis. Attacks can be triggered by any acute medical or surgical illness (pneumonia, MI, post-op etc). There are now national and international guidelines which have reached consensus about how to manage gout.

### **Diagnosis**

This is often a clinical matter. Bear in mind the usual pattern, which is *acute attacks* affecting the big toe first (in 70%), which then can spread over time (many months or years) to involve the midtarsal joints, ankles, knees and then upper limb joints. *Hip and shoulder or spinal involvement is very unusual* and should prompt evaluation for alternative diagnoses. In hospital populations a lot of gout is secondary, and more likely to be polyarticular, and less likely to involve the big toe. Foot/mid tarsal involvement can look almost *cellulitic*, and it often moves around, first affecting one side of the foot, then the other etc over a few days. It can appear as a *'flitting' arthritis*. Gout can cause a *bursitis* especially the olecranon bursa which may contain lumpy tophi. There will often be a prior history – it is worth asking the patient if they have ever had 'gout' or anything like it before. Look for *tophi* which can be on the ears but are more commonly over the DIP joints of hands and feet, in the finger pulps, or other soft tissue sites eg Achilles tendon or olecranon bursa.

A large joint can be *aspirated* and finding crystals is obviously diagnostic. Joint fluid can be negative however late in the attack.

*Serum uric acid /urate(SUA)* should be measured, but falls during an acute attack so a normal SUA during an attack does not rule it out. If it is normal and gout can't be confirmed, it should be repeated when the attack has settled ('intercritical SUA').

### **Treatment of the acute attack**

The three options are NSAIDs, colchicine and corticosteroids.

NSAIDs are entirely appropriate in younger fit individuals, especially those who have used them before. However many unwell inpatients will have contraindications/cautions such as CKD, cardiovascular disease, peptic ulceration etc. Risks of adverse effects are also much higher in older individuals. We would not recommend using NSAIDs in patients >65 years old for gout as first line treatment.

***We recommend colchicine*** as first line treatment in hospital or the community.

Try colchicine 500 microgrammes (1 tablet) 3-4 times a day initially. Doses above 500 microgrammes 4x/day are likely to cause diarrhoea. The advice in the BNF about giving colchicine every 2 hours until diarrhoea is induced is obsolete and should not be followed. It

has been revised in the most recent BNF. As the patient improves gradually cut the dose down over 2-4 weeks.

If intolerant to colchicine, an alternative is to use a short course of oral prednisolone 30mg daily tapering over 2-3 weeks. Patients who are intolerant to colchicine should be referred to rheumatology. Large joints (knees, ankles) can be injected with steroid, which is extremely effective.

Most patients will respond within 24 hours – some take longer and require a combination of treatments.

### **Who needs treatment with urate-lowering therapy ('ULT')?**

#### **In the community -**

Anyone with persistently raised SUA and

Recurrent attacks of gout

Tophi

Any evidence of Destructive gouty arthritis e.g. xray changes

Anyone with stones or nephropathy

#### **In hospital -**

Most patients with attacks of gout in hospital will have ongoing risk factors and will need ULT. We suggest treatment with allopurinol initially for all patients in hospital because of gout or with gout complicating their stay, assuming the diagnosis is secure, that SUA is found to be significantly elevated and it is not their first ever attack ( in which case it may be appropriate to wait).

### **How to start urate lowering therapy**

ULT can trigger an acute attack of gout, hence the advice to wait until the acute attack has settled before starting. This is not always necessary *as long as the advice below is followed* and at times it is appropriate to take the opportunity to commence treatment in hospital.

***Acute attacks of gout will be prevented by ensuring the patient has concomitant cover/prophylaxis. This should be given routinely to all patients when starting ULT regardless of whether or not they are suffering attacks of gout. We use colchicine 500 microgrammes bd for this.*** (NSAIDs could be appropriate in selected patients). This is consistent with advice in the BNF. Prophylaxis should be continued until the SUA is controlled (can take many weeks) and a little while longer eg 6-12 weeks, and then ceased..

### **A low dose of allopurinol should be used initially and titrated until urate is below 340micromo/l.**

Allopurinol is renally excreted. We suggest commencing 100mg daily in those with eGFR <40ml/min, 200mg if eGFR between 40 and 60ml/min, or 300mg if eGFR is >60ml/min. SUA should be measured 4 weeks or so after starting and the dose of allopurinol increased if necessary (by 100mg steps every 4-8 weeks) until SUA is in the target range (<340micromol/l). ***This is essential as attacks will not cease until the urate level is controlled.***

Once the SUA is controlled, acute attacks of gout should cease, although this can sometimes take many months or longer if there is a particularly high urate burden (untreated gout for prolonged period of time). Tophi take many months or longer but often resolve.

There are some important drug interactions with allopurinol –

*Can increase anticoagulant effect of warfarin* – concomitant use is **NOT** contraindicated but closer monitoring of INR is required when commencing treatment.

*Can increase toxicity of azathioprine* – try to avoid using the two concomitantly

**What should I do if a patient develops an acute attack of gout whilst on allopurinol?**

If an attack develops whilst on ULT, *do not stop the allopurinol* but simply treat the acute attack – it may be appropriate to continue with prophylaxis for longer.

**When should I refer to rheumatology?**

***Inpatients-***

If diagnosis not clear, especially if not responding to treatment within 24 hours, or other serious condition suspected.

Intolerant of, or not responding to colchicine.

***Outpatients-***

As above.

Gout resistant to allopurinol, or intolerant of allopurinol. (Other options are benzbromarone, sulphinyprazole or febuxostat – some physicians will be happy to start these agents themselves.)