

## Acute Gout Management:

Affected joints should be rested, elevated and kept cool.<sup>1,5</sup>

Start anti-inflammatory/analgesic therapy straight away and continue for 1-2 weeks<sup>2</sup>

**1<sup>st</sup> line: NSAIDs Ibuprofen or Naproxen**, continued for 48 hrs after attack has resolved.

There is no evidence to suggest 1 NSAID is more effective than another, ibuprofen/naproxen are preferred based on safety profile, indomethacin is best avoided due to severe side effects and salicylates should be avoided as they interfere with uric acid excretion<sup>2</sup>.

NSAIDs benefit from being quick acting, but are associated with GI and renal safety concerns. The NICE NSAIDs advice note recommends using doses of Ibuprofen 1200mg/day (or less) or naproxen 1000mg/day (or less)<sup>3</sup>. In practice higher doses may be required to achieve adequate pain relief for patients with an acute flare of gout. Consider a PPI for patients at high risk of GI bleeding or ulceration. (See also [NICE KTT13](#))<sup>3</sup>

**2<sup>nd</sup> line: Colchicine<sup>1,2&5</sup>** 500 micrograms 2-3 times per day, until symptoms are relieved

Can be used in patients on warfarin and in patients with heart failure but use is limited by side effects (can cause profuse diarrhoea) and the development of toxicity at higher doses. (See also [colchicine prescribing information](#))

**3<sup>rd</sup> line: Corticosteroids<sup>1,5</sup>**

Oral -Prednisolone 20-40mg daily for 5 days

STAT Intramuscular injection -Methylprednisolone 40-120mg or  
-Triamcinolone acetonide 40-80mg

STAT Intraarticular injection (Off license use. Suitable for gouty monoarthritis only)

- Methylprednisolone 10-80mg or
- Hydrocortisone acetate 12.5- 25mg
- Triamcinolone acetonide 20-40mg

### Does the patient have any of the following?<sup>2</sup>

- Definite diagnosis of gout  $\geq 2$  attacks within 1yr
- Tophi
- Erosive arthropathy (x-ray)
- Uric acid nephropathy or nephrolithiasis
- Polyarticular flare at presentation<sup>13</sup>

If NO: Uric acid lowering therapy is not indicated. If 1<sup>st</sup> attack check serum urate (sUA) 4 weeks post flare.

Reconsider diagnosis: refer persistent symptoms without definitive diagnosis secondary care<sup>1,13</sup>

If YES: consider LONG TERM TREATMENT with uric acid lowering therapy.

## Chronic Gout Management<sup>13</sup>

(See also allopurinol and febuxostat [prescribing information](#))

- Ensure serum urate (sUA) levels are obtained during the convalescent period (4 weeks post-acute attack)
- Commence **allopurinol 1<sup>st</sup> line** ensuring at least 1-2 weeks have passed from last attack

- Start at 100 mg/day, and titrate to achieve sUA  $<360 \mu\text{mol/l}$  \*
- Recheck sUA 4 weeks after initiation
- If  $>360 \mu\text{mol/l}$  increase to 200 mg/day
- Recheck sUA after a further 4 weeks
- If  $>360 \mu\text{mol/l}$  increase to 300 mg/day
- Continue monthly sUA levels, increasing by a further 100mg/day each month until sUA  $<360 \mu\text{mol/l}$  or maximum tolerated dose is reached (The BNF max dose = 900 mg per day,<sup>8</sup> but locally it has been agreed that treatment should be reviewed at 600 mg per day)
- Once sUA target is achieved, annual sUA measurements are recommended
- **Co-prescribe prophylactic colchicine** (500 micrograms twice daily for up to 6 months) or a NSAID (ibuprofen 200mg twice daily or naproxen 250mg daily for up to 6 weeks)<sup>13</sup> to prevent an acute gout flare.<sup>3,5</sup>

If allopurinol is contraindicated, not tolerated or there is inefficacy despite titration to 600 mg per day.

- Consider **Febuxostat 2<sup>nd</sup> line** ensuring at least 1-2 weeks have passed from last attack (See also [NICE TA 164](#) and [SPC](#))<sup>7</sup>
- The licensed starting dose is 80 mg once daily, but starting with a 40mg dose (half an 80mg tablet), may decrease the incidence of acute flares<sup>1</sup>
- If after 4 weeks, the sUA is  $>360 \mu\text{mol/l}$  then the dose can be increased by 40mg & sUA rechecked in a further 4 weeks
- Max dose = 120 mg daily
- **Co-prescribe prophylactic colchicine** (500 micrograms twice daily for up to 6 months) or **NSAID** (ibuprofen 200mg twice daily or naproxen 250mg daily for up to 6 weeks) to prevent an acute gout flare.<sup>3,5,13</sup>

NB: Stop febuxostat immediately if hypersensitivity occurs, do no restart (See [MHRA warning](#))<sup>7</sup>

## Refer to Secondary Care<sup>1&13</sup>

**Immediately if Septic Arthritis is suspected**  
(Please note it is possible for both gout and septic arthritis to co-exist)

Or by routine referral if:

- The sUA is unresponsive to uric acid lowering therapy
- If gout persists despite uric acid levels  $<360 \mu\text{mol/l}$ \*
- Patient suffers complications relating to gout e.g. nephropathy
- Patient requires Intra-articular therapy and primary care are not able to provide
- There is diagnostic uncertainty

## In Addition to Acute Management

- **Assess lifestyle factors** and provide advice. E.g. reduce alcohol intake (particularly beer) and intake of purine rich foods (shellfish & meat)<sup>1,2</sup>. Click on the relevant link for patient information on<sup>4</sup>:

- [Gout](#)
- [Diet](#)
- [Related health problems](#)
- [Treatments](#)

- **Treat cardiovascular risk factors.**

There is a connection between sUA levels and CV disease, with high sUA being part of metabolic syndrome. Additionally components of metabolic syndrome are independent risk factors for gout<sup>2, 14 & 16</sup>

- **Consider drug induced gout**

• Low dose aspirin (75-150mg/day) has insignificant effects on plasma urate but higher doses interfere with uric acid excretion and should be avoided<sup>1&5</sup>

- **Review antihypertensives:** Diuretics (Inc. thiazide), <sup>1</sup> B-blockers, ACE inhibitors and non-losartan angiotensin II receptor blockers<sup>15</sup> increase sUA. Losartan and calcium channel blockers decrease sUA<sup>2&15</sup>

**DO NOT interrupt uric acid lowering therapy unless there is a clinical reason**  
**Gout flare is NOT a clinical reason<sup>1</sup>**

Table 1: Renal Dose Adjustments<sup>2,8-11</sup>

Allopurinol	CrCl 10-20ml/min: Max 100-200mg/day CrCl <10ml/min: Max 100mg/day
Colchicine	CrCl 10-50ml/min: Reduce dose or increase interval CrCl < 10ml/min: Do not use
Febuxostat	No dose adjustment is necessary in mild or moderate impairment.  Efficacy and safety have not been fully evaluated in patients with CrCl <30 ml/min
NSAIDs	Avoid if possible/use with caution in renal impairment <sup>1</sup> Use the lowest effective dose for the shortest possible duration and monitor renal function.

\*The BSR guidelines recommend titrating uric acid lowering therapy to sUA  $<300 \mu\text{mol/l}$ , but some other guidance<sup>1,2,6</sup> use  $<360 \mu\text{mol/l}$  which is locally considered more achievable and is supported by cohort studies<sup>5</sup>

## Supporting Information

Gout is the most common inflammatory arthritis in the UK and mainly diagnosed and managed in primary care. The clinical course of gout is largely predictable and divided into three stages.<sup>2</sup>

### Stage 1: Asymptomatic

#### Hyperuricaemia

sUA levels rise but no clinical features of gout are present.

### Stage 2: Acute Gout and “intercritical periods”

sUA reaches saturation, monosodium urate (MSU) crystals are precipitated and deposited in joints causing an inflammatory response and painful arthritis. Attacks are self-limiting and followed by asymptomatic “intercritical” periods before the next flare.

### Stage 3: Chronic

#### Tophaceous Gout

Crystal deposits in the joints and surrounding soft tissue. Symptoms are persistent.

**Diagnosis<sup>2</sup>:** sUA is the most important risk factor for gout, but should only be considered in combination with other clinical features, since on its own it does not confirm or exclude a diagnosis of gout. Many patients with hyperuricaemia do not develop gout, and sUA can be normal during an acute attack.

**Acute Gout Diagnosis:** Acute joint pain is the most common presenting complaint.

Joints affected include the big toe (up to 78% of 1<sup>st</sup> attacks) foot, ankle, knee, wrist, finger, and elbow.

Urate crystals in fluid aspirated from the affected joint is the “gold standard” for diagnosis, but in reality, this test is only applied to a minority of patients and most patients are diagnosed clinically.

**Features which strongly predict a diagnosis of gout are<sup>2</sup>:**

- First MTP joint involvement
- Rapid onset of severe joint pain (over 6-12 hrs)
- Joint swelling and tenderness
- Overlying erythema
- Self-limiting with complete resolution
- Tophus (proven or suspected)

Laboratory and radiological investigations are not necessary as sUA is often normal during an attack, and x-rays are unlikely to be helpful

**Chronic Gout Diagnosis:** In between attacks MSU crystals will be seen in joint fluid providing definitive diagnosis.

In the absence of this, **the following features are strong predictors of gout<sup>2</sup>:**

- History of classical acute attacks
- Presence of tophi
- Asymmetrical swelling within a joint, or subcortical cysts without bony erosions on x-ray
- Double contour sign” (hyperechoic band seen parallel to the edge of the joint line) on ultrasound – nb not available in all regions.

## Prescribing Information. Please Refer to Individual [SPCs](#) for Full Details

### Allopurinol<sup>8</sup>

**Renal impairment:** Dose adjustment required see [Table 1](#).

**Side Effects:** Most common are GI intolerance.<sup>2</sup>

**Pruritic maculopapular skin rashes** may occur in up to 10% of people who take allopurinol, the rash can be the 1<sup>st</sup> sign of a severe but rare hypersensitivity syndrome. Patients should be advised to stop allopurinol immediately and seek medical advice promptly. When the rash is gone if it was mild it can be gradually reintroduced, if the rash recurs immediately discontinue the allopurinol.

**Interactions:** Allopurinol potentiates the anticoagulant effect of warfarin (increase monitoring during dose titration). Do not co-prescribe with azathioprine (it inhibits the metabolism of azathioprine, leading to accumulation of toxic metabolites).<sup>2</sup>

**Do not start during an acute attack** ensure at least 1-2 weeks have passed before initiation, (can prolong the attack indefinitely). Continue if an attack develops whilst already on treatment and treat attack separately.<sup>12</sup>

### Febuxostat<sup>10</sup>

**Cautions:** Max 80mg daily in mild liver impairment (no information available in moderate-severe liver impairment) Use with caution in patients with thyroid disorders, ischaemic heart disease and/or heart failure.<sup>2</sup>

**Side Effects:** Most common are GI, abnormal LFTs and oedema. **Serious hypersensitivity reactions**, including Stevens-johnson syndrome and acute anaphylactoid/shock reactions have been reported, mostly during the 1<sup>st</sup> month of therapy. Treatment should be stopped immediately if signs of hypersensitivity reactions occur and treatment must not be restarted.<sup>8</sup> [See MHRA warning](#)

**Interactions:** Avoid concomitant use with azathioprine and mercaptopurine

**Do not start during an acute attack** ensure at least 1-2 weeks have passed before initiation, (can prolong the attack indefinitely). Continue if an attack develops whilst already on treatment and treat attack separately.<sup>12</sup>

### Colchicine<sup>9</sup>

**Dose and duration of treatment:** For acute attacks, this guidance recommends 500mcg 2-3 times daily, until symptoms are relieved. This is lower than the dose listed in the SPC, because there is trial and clinical practice evidence that the higher dosing regimen is frequently associated with diarrhoea and other toxic side effects.<sup>5</sup> If colchicine is used for treatment of an acute attack, it can be continued as **prophylactic treatment** at a reduced dose of 500mcg twice daily upon initiation of uric acid lowering therapy and used for up to 6 months.

**Renal impairment:** Dose adjustment required see [Table 1](#).

**Cautions:** Increased risk of toxicity in elderly and debilitated patients. Avoid in patients with blood disorders.

**Side Effects:** Use is limited by side effects and toxicity at higher doses. Can cause profuse diarrhoea, nausea, vomiting and abdominal pain.

## References

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## Version 1.0

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