Whittington Health NHS

Gout - Management of Acute Disease

Subject:	Gout (Acute)
Policy Number	N/A
Ratified By:	Clinical Guidelines Committee
Date Ratified:	May 2011 (original), reviewed August 2015
Version:	2.0
Policy Executive Owner:	Dr C Murdoch, Clinical Director (Medicine, Frailty and Networked Service ICSU)
Designation of Author:	Consultant Rheumatologist(s) Dr I Wamuo (version 1) Dr A Nuttall (Version 2)
Name of Assurance Committee:	As above
Date Issued:	August 2015
Review Date:	3 years hence
Target Audience:	General internal medicine doctors, Emergency department, Orthopaedics, Rheumatology and Care of the Elderly.
Key Words:	Gout, uric acid, urate, monoarthritis, oligoarthritis, polyarthritis, crystal arthropathy, hyperuricaemia

Version Control Sheet

Version	Date	Author	Status	Comment
1.0	May 2011	Dr Rachel Byng-Maddick (SpR) Dr Wamuo	OFF LINE	New guideline ratified at Clinical Guidelines Committee
2.0	August 2015	Dr Anna Nuttall	LIVE	Reviewed with very minor change. Deletion of sentence: *Prescribe a maximum of 6 mg per course; course not to be repeated within 3 days* Updating of contacts

➢ Introduction

Gout attacks occur in patients with high levels of uric acid. It is a common disorder of uric acid metabolism caused by deposition of monosodium urate crystals in soft tissue, causing recurrent episodes of debilitating joint inflammation. If untreated, may lead to joint destruction and renal damage.

Gout is definitively diagnosed by demonstrating uric acid crystals in synovial fluid. Early diagnosis and definitive treatment can significantly improve prognosis. Uric acid levels are generally elevated for 20 years before onset of symptoms. Tophi are often clinically detectable 10 years after the first gout attack.

Gout is unlikely to present in premenopausal women, or men < 30 years unless they have renal insufficiency or a genetic abnormality of purine metabolism (e.g. Lesch-Nyhan Syndrome). The higher prevalence in the elderly may reflect an increased prevalence of metabolic syndrome, high rates of diuretic treatment, and the use of low-dose aspirin.

➢ History

- Acute attacks begin abruptly, often at night, reaching maximal intensity within 8-12 hours
- Hot, red, exquisitely tender joints
- Untreated, attacks may resolve spontaneously in < 2 weeks
- In chronic gout, attacks may be less intense, but last longer
- 90% presentations involve one joint (monoarticular)
    - Typically smaller, lower extremity joints
    - Podagra (inflammation of the 1st metatarsophalangeal joint) is the initial joint manifestation in 50% cases, and eventually involved in 90% cases
- Oligo- or polyarticular arthritis i.e. > 3 joints
    - More common in chronic gout
    - Tends to affect upper limb more commonly
- Attacks may occur as a bursitis or tendonitis e.g. olecranon bursa

Please see Whittington Health Guideline:
The management of the red, hot swollen joint.

Risk factors for hyperuricaemia:

Raised levels of serum uric acid may be due to overproduction of uric acid, or under excretion through the kidney.

Uric acid over production:

Common causes include
- Drugs – chemotherapy agents
- Psoriasis
- Food with high purine content – red meat, marmite, yeast
- Myeloproliferative or lymphoproliferative disorders

Less common causes include
- Haemolytic or pernicious anaemia
- Ineffective erythropoiesis (e.g. Vitamin B12 deficiency)
- Excess exercise

Very rarely uric acid overproduction may be due to inherited syndromes e.g. Lesch-Nyhan, Glucose-6-phosphatase deficiency (G6PD), Fructose 1-phophat aldolase deficiency.

Uric acid underexcretion:

Common causes include
- Drugs – diuretics, cyclosporine
- Chronic alcohol abuse (especially beer or fortified wine)
- Renal insufficiency*
- Starvation or dehydration (including trauma or haemorrhage)

* Chronic urate nephropathy can also contribute to renal insufficiency from the deposition of urate crystals in the medullary interstitium and pyramids, resulting in an inflammatory reaction that can lead to fibrotic changes

Less common causes include
- Hypothyroidism
- Hypoparathyroidism

These patients are 1000x more likely to develop renal stones than healthy individuals. 80% have pure uric acid stones, 20% develop calcium oxalate or calcium phosphate stones with a uric acid core.

Particular risk factors for an acute attack:
- Sudden reduction in level of serum uric acid
    - e.g. radiocontrast dye, allopurinol and uricosuric agents
- Metabolic syndrome (insulin resistance or diabetes, hypertension, hypertriglyceridaemia, and low levels of HDLs)

➢ Examination

During an acute gout attack:

- Examine all joints for swelling, warmth, erythema, and tenderness – remember gout can be very painful!
    - If the joint capsule is swollen, there is reduced range of motion
- Erythema over the joint may resemble cellulitis and skin may desquamate as the attack subsides
- Look for tophi (small subcutaneous collections of uric acid crystals) along pinna of the ear, fingers, toes, and along olecranon (can resemble rheumatoid nodules) or other tendon sheaths
- The patient may be pyrexial – particularly if polyarticular involvement

Septic arthritis and gout can co-exist, so complete a general examination to ensure you have looked for other sites of infection.

➢ Differential Diagnosis

Other crystal arthropathies – including Pseudogout (calcium pyrophosphate/CPPD) and hydroxyapatite
Cellulitis
Septic Arthritis
Reactive Arthritis
Rheumatoid Arthritis
Psoriatic arthritis
Sarcoidosis
Seronegative inflammatory monoarthritis

➢ Investigation

Blood tests

- Full blood count(FBC): raised white cell count in acute attacks (mainly a neutrophilia)
- C-reactive protein(CRP)/ Erythrocyte sedimentation rate(ESR): likely to be raised in acute attacks
- Urea and electrolytes may be raised / eGFR may be impaired
- Serum uric acid: elevated (NB. may be normal during acute attacks)
- Blood cultures – if suspicious of septic arthritis

NB. An isolated finding of raised serum uric acid (without joint symptoms) does not equal a diagnosis of gout, but does predispose to the condition.

Synovial fluid analysis

- Aspiration and culture of the joint is the only way to definitively rule out septic arthritis and confirm the presence of uric acid crystals
- Send for urgent analysis: - type ‘SYNOVIAL FLUID’ into ICE requests

- Microbiology - gram stain and culture
  - If clinically suspicious for AFB smear (to exclude TB)
- Cytology - for crystal analysis

NB. Septic arthritis and gout may co-exist, so presence of crystals does not necessarily exclude infection if highly suspicious. The synovial fluid white cell count (polymorphonuclear neutrophils) is likely to be raised in both conditions.

Imaging
Is not diagnostic in acute gout, unless suspecting trauma or osteomyelitis.

➢ Treatment

Management of acute attacks

Affected joints should be rested appropriately e.g. splinting and bed rest. Treatment should be started immediately and continued for until symptoms resolve. This usually takes 1-2 weeks.
NB. Do NOT stop allopurinol during an acute attack (if patient is already taking it)

1. **Non steroidal anti-inflammatory drugs (NSAIDs)**

   – Fast acting oral NSAIDs at maximum dose are the drugs of choice e.g. Diclofenac sodium 50mg tds po

   – Caution in: renal failure, peptic ulcer disease, GI haemorrhage or perforation, asthma, concomitant warfarin use or abnormal liver function

   – Consider co-prescribing gastroprotection using a proton pump inhibitor(PPI) - Lansoprazole 15mg od po

If unable to take NSAIDs – alternative treatment:

2. **Colchicine**

   – An alternative to NSAIDs, but slower onset of action – a starting dose of 500 micrograms bd po is usually adequate. Continue until symptoms relieved or if side effects such as diarrhoea and vomiting occur.

   – Side effects include nausea, diarrhoea and vomiting – warn patient about these symptoms

   – Do not use if eGFR < 10 ml/min/1.73m^2, avoid in hepatic dysfunction or biliary obstruction

Steroids (to be used in discussion with the rheumatologist)
– Used if intolerant of NSAIDs and colchicine

- Prednisolone 10-15mg once daily for 10-14 days po (higher dose and longer duration if polyarticular symptoms)

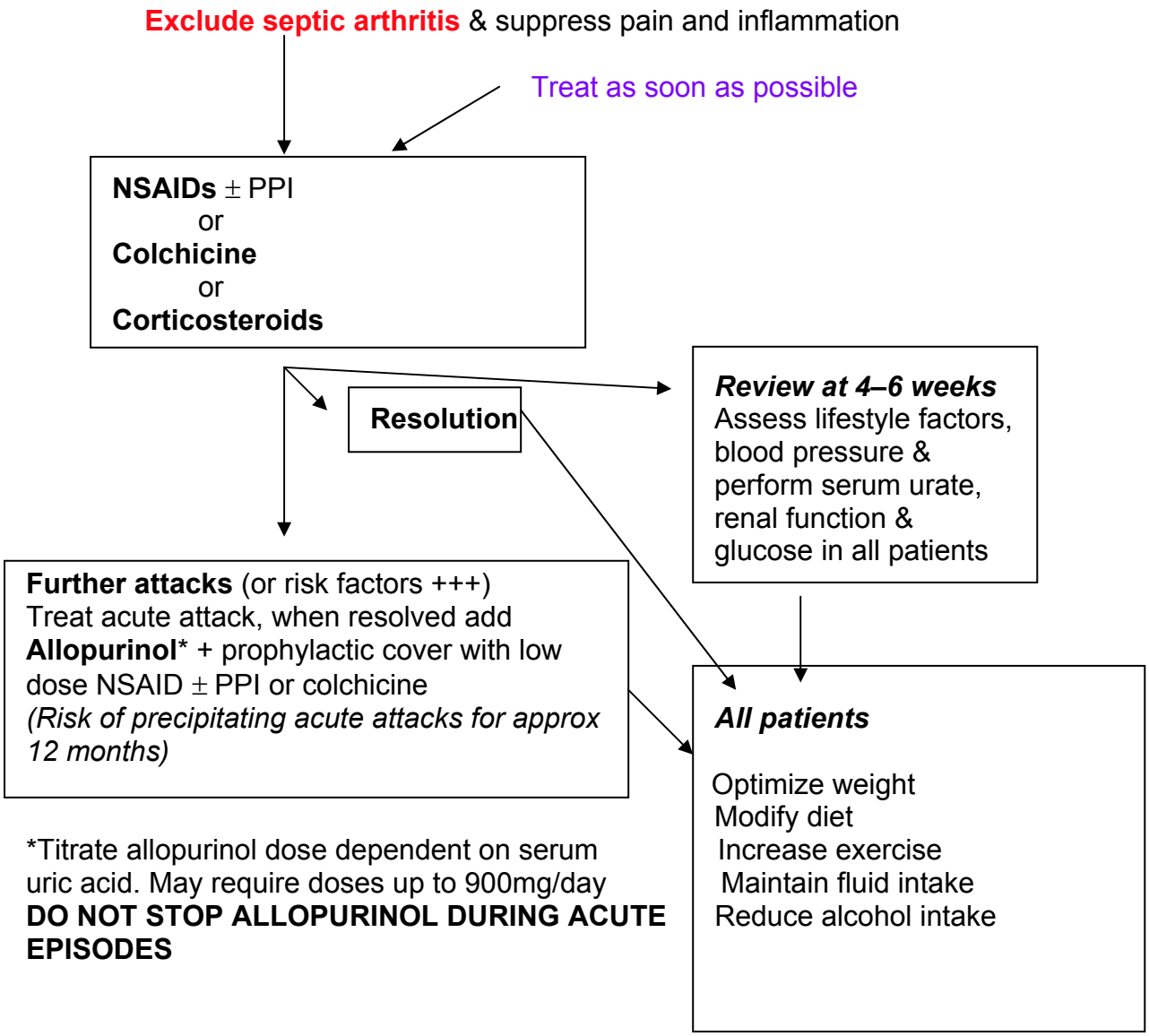
- Intra-articular steroids can be used provided microscopy shows no evidence of infection

- Intra- muscular depomedrone 80mg-120mg can be used

3. Opiates

- Can be used as adjuncts for pain – although generally not very helpful in gout e.g. co-dydramol (paracetamol/dihydrocodeine) two tablets qds po

GOUT: MANAGEMENT PATHWAY – adapted from BSR Gout Guidelines
http://www.rheumatology.org.uk/includes/documents/cm_docs/2009/m/management_of_gout.pdf



*Titrate allopurinol dose dependent on serum uric acid. May require doses up to 900mg/day
DO NOT STOP ALLOPURINOL DURING ACUTE EPISODES

➢ Evaluation of the guideline

Clinical audit of this guideline will be undertaken if deemed appropriate by the author(s). Results to be discussed at Rheumatology Department meetings in the first instance with escalation of findings, only if relevant.

	Please see Whittington Health Guideline: ***Management of Chronic Gout***

➢ Contacts (inside and outside the Trust including out-of-hours contacts)

Monday-Friday 09.00-17.00:

Rheumatology Consultants:	ext 5259 (secretary) Or via switchboard
Dr Anna Nuttal Dr Elena Nikiphorou Dr Ihuoma Wamuo	
Rheumatology SpRs	bleep 3033 / 3088
Out of hours: Duty Medical Spr	bleep 3300

Appendix A

Plan for Dissemination and implementation plan of new Procedural Documents

To be completed and attached to any document which guides practice when submitted to the appropriate committee for consideration and approval.

Acknowledgement: University Hospitals of Leicester NHS Trust

Title of document:	**Acute gout**		
Date finalised:	**Re-issued August 2015**	**Dissemination lead: Print name and contact details**	**Rheumatology Consultants**
Previous document already being used?	**No** **(Please delete as appropriate)**		
If yes, in what format and where?			
Proposed action to retrieve out-of-date copies of the document:			

To be disseminated to:	**How will it be disseminated/implemented, who will do it and when?**	**Paper or Electronic**	**Comments**
Medical and surgical teams	**Intranet- audit training sessions via rheumatology department**	**electronic**	
Is a training programme required?	**no**		
Who is responsible for the training programme?			

Appendix B

Equality Impact Assessment Tool

To be completed and attached to any procedural document when submitted to the appropriate committee for consideration and approval.

Impact (= relevance) 1 Low 2 Medium 3 High	Evidence for impact assessment (monitoring, statistics, consultation, research, etc	Evidential gaps (what info do you need but don't have)	Action to take to fill evidential gap	Other issues
Race	**1**	**N/A**		
Disability	**1**	**N/A**		
Gender	**1**	**N/A**		
Age	**1**	**N/A**		
Sexual Orientation	**1**	**N/A**		
Religion and belief	**1**	**N/A**		

Once the initial screening has been completed, a full assessment is only required if:

- The impact is potentially discriminatory under equality or anti-discrimination legislation
- Any of the key equality groups are identified as being potentially disadvantaged or negatively impacted by the policy or service
- The impact is assessed to be of high significance.

If you have identified a potential discriminatory impact of this procedural document, please refer it to relevant Head of Department, together with any suggestions as to the action required to avoid/reduce this impact.