NHS
Vale of York
Clinical Commissioning Group

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Referral Support Service

Rheumatology

RH05 Gout

Definition

Gout is an inflammatory arthritis caused by accumulation and deposition of sodium urate crystals. Gout is the most common inflammatory arthropathy, affecting 2.5% of adults in the UK. Whilst characteristically it is intermittent nature it is increasingly thought of as a chronic condition. It affects the great toe most often, although any joint can be involved. 62% of patients with gout will have a second attack within 12m with the risk increasing with higher urate levels.

Chronic crystal deposition can be seen in multiple peripheral joint and peri-articular sites at the first attack of gout and likely predates symptoms by some years. These intra-articular crystals lead to ongoing joint damage and chronic arthritis.

Patients diagnosed with hyperuricaemia are at increased risk of metabolic syndrome.

Complications of Gout (from NICE CKS)

- Tophi occur in approximately 50% of people with untreated gout after 10 years. This increases to 72% after 20 years [BMJ Best Practice, 2017].
- Tophi may create problems with activities of daily living, become inflamed, exude tophaceous material, or develop secondary infection, and adversely impact on quality of life [Dalbeth, 2016].
- Urinary stones are found in around 14% of people with gout [Roughley, 2015].
- Gout is an independent risk factor for Chronic kidney disease, myocardial infarction and cardiovascular disease mortality [Clarson, 2015; Liu, 2015; Roughley, 2015].
- Other comorbidities include hypertension (74%), hyperlipidaemia, osteoarthritis, obesity (53%), diabetes (26%), congestive heart failure (11%) and ischaemic heart disease (14%) [Roddy, 2013].

The following pathway does not include patients who are under long term follow up by the renal team.

Diagnosis

Joint aspiration

The ‘gold standard’ is aspiration of synovial fluid with microscopy for crystals. However this is time consuming and false negative results can be seen if there is delay in the sample reaching the lab. It is not expected this becomes a standard in general practice.

Clinical diagnosis

The typical history of gout is sudden-onset acute pain in one joint, peaking within 12-24h and then subsiding in 1–2w. 50–75% of the time it affects the 1st MTP joint but can also affect the knee, wrist, fingers, ankles and midfoot.

Differential diagnosis

- Acute calcium pyrophosphate crystal arthritis (also known as pseudogout)
- Septic arthritis.

NICE CKS says: Gout may be present without hyperuricaemia and a normal level of urate does not exclude the diagnosis. Normal levels are often found during an acute flare of gout, when plasma urate levels may fall to normal. Serum uric acid (SUA) is usually measured **4–6 weeks after an acute attack of gout** to confirm hyperuricaemia.

The formation and deposition of monosodium urate crystals occur when urate levels are persistently above 380 micromol/L. Hyperuricaemia may be present without gout. The presence of hyperuricaemia does not equate with a diagnosis of gout, as **most people with hyperuricaemia do not develop gout.**

There is a primary care scoring system that does not require aspiration and that performs better than clinical judgement alone (Rheumatology 2015;54:609). Clinical assessment has a PPV of 64% and an NPV of 87%. The tool offers a PPV of 87% and an NPV of 95%.

Features of Presentation	If Yes score:
Male	2
Previous patient reported arthritis attack	2
Onset within 1day	0.5
Joint Redness	1
1st MTP Joint involved	2.5
History of Hypertension or at least one of these: IHD/CHC/Stroke/PVD	1.5
Serum uric acid > 350µmol/L	3.5
Interpretation of scores	
Gout unlikely, consider alternative diagnoses	<4 points
This group are most likely to benefit from aspiration to establish the diagnosis*	4-8 points
Gout highly likely: start empiric treatment (may be falsely low during an attack)	>8 points

* doesn't need to be undertaken routinely and not urgently either. Joint aspiration can be done in primary care or primary care clinicians can seek advice from Rheumatology colleagues who can decide whether it's necessary to undertake themselves or advise to refer for USS guided aspiration

Management

Treatment of Gout and will be determined by risk, frequency and severity of attack and the management of it should be undertaken in Primary Care.

Only refer to Secondary Care if intolerant of conventional urate lowering therapies or troublesome disease control on standard therapies.

Acute attacks

These events should be treated with **non-steroidal anti-inflammatory drugs** with appropriate GI protection if indicated or **colchicine** 500mcg 2-4 times per day, maximum 6mg per course and do not repeat treatment within three days. Be familiar with NICE guidance on colchicine. **Colchicine** has a narrow therapeutic index and is extremely toxic in overdose. Lower the dose and dose interval in renal impairment.

It should be noted that **aspirin** is not indicated in gout.

The British Society of Rheumatology suggest prednisolone 35mg daily for 5 days if first line choices are contraindicated. Intramuscular or intra-articular steroids are also effective in the

acute event. All treatments are similarly effective and prescribers should opt for the safest and best tolerated option for individuals.

Ice packs may be used as safe adjuncts to pharmacological treatment for acute gout, or when drugs are contraindicated because of multiple active co-morbidities

Urate lowering therapy

Urate lowering therapy should be started 1–2 weeks after the inflammation has settled

Urate lowering therapy should be considered in anyone with an episode of gout and should be strongly offered to patients if

- there have been two or more attacks in a year,
- tophi are present, there is radiographic evidence of erosion,
- renal impairment (eGFR <60)
- uric acid stones
- in patients on long term diuretic therapy.

Therapy should be aimed at suppressing serum urate to below 300 micromol/L

If not contraindicated: Start **allopurinol**. Be familiar with NICE guidance on allopurinol. Initially 100mg daily and titrate the dose every few weeks until the serum uric acid (SUA) level is below target level. The maximum dose of **allopurinol** for gout prophylaxis is 900mg daily in divided doses (lower in renal impairment). Monitor urate and renal function whilst increasing doses. NICE recommend up titration every four weeks until the serum uric acid (SUA) level is below 300 micromol/L. There is specific guidance in patients with renal impairment here.

If patients are intolerant of **allopurinol** consider **febuxostat** as second-line therapy. However, note the local formulary advises: Monitor liver function tests prior to initiation and then periodically thereafter based on clinical judgement. Avoid treatment with febuxostat in patients with pre-existing major cardiovascular disease. A prior history of hypersensitivity to Allopurinol and/or renal disease may indicate potential hypersensitivity to **febuxostat**.

As prophylaxis against acute attacks secondary to when starting or increasing the dose of urate lowering therapy, co-prescribe either

- low-dose **colchicine** (500mcg up to twice daily), for six weeks to prevent acute attacks of gout and continue this prophylaxis for up to 6 months, though three is often sufficient.

- In patients who cannot tolerate colchicine, a low-dose **NSAID** or **coxib**, with gastroprotection, can be used as an alternative providing there are no contraindications.

- NICE say carefully consider the risk to benefit balance when considering long-term gout flare prophylaxis, particularly in people with comorbidities or taking medication with potential for interaction.

If **NSAIDs** and colchicine are contraindicated, Y&SHFT rheumatologists suggest considering low-dose (up to 0.25mg/kg) oral **prednisolone** once a day for 4 to 12 weeks though this increases their risk of infections and other complications of steroids. **This is not in NICE or BSR guidelines**. Ensure there is careful tapering to reverse any adrenal suppression that has occurred. Give patients a steroid card and warn patients about not abruptly stopping treatment.

Advise the person that:

- Urate-lowering medication is **normally lifelong** and regular monitoring is needed If used as described above **allopurinol** nor **febuxostat** should not cause acute attacks of gout on initiation. A radical change of lifestyle may have removed some of the triggers for hyperuricaemia so a cautious reduction and cessation of urate lowering therapy can be tried.
- Explain that they should start their anti-inflammatory treatment as soon as possible and **not to stop** their **allopurinol** or **febuxostat** during acute attacks

It may be useful in practices to regard gout as a long-term condition to be managed by trained practice nurses. A UK-based RCT study demonstrated that over two years nurse-led care can be more clinically-effective and cost-effective than GP-led care (Lancet 2018;392;1403). The nurses achieve normal serum urates in 95% of patients compared to only 30% in those receiving normal care albeit with higher doses of allopurinol and with many more contacts. This meant it was a more expensive service to provide although well within the NICE investment threshold for the NHS.

Lifestyle advice

Advise people with gout to:

- Aim for an ideal body weight — but avoid crash dieting and high protein/low carbohydrate diets
- Eat sensibly — by restricting the amount of red meat and avoiding a high protein intake. Avoid excessive consumption of foods rich in purines (such as liver, kidneys, and seafood)
- Drink alcohol sensibly — by avoiding binge drinking and restricting alcohol consumption especially avoiding beer and other high purine drinks with at least two alcohol-free days a week. Note not all alcohol-free beers are low in purines. Detail on purines in alcohol here.
- Avoid dehydration by drinking water (up to 2 litres/day unless there is a medical contraindication)
- Drink skimmed milk or consume low-fat dairy products (up to 2 servings daily)

- Limit consumption of sugary drinks and snacks (especially carbonated drinks)
- Take regular exercise — but avoid intense muscular exercise and trauma to joints
- Stop smoking

Indications for referral

If intolerant of conventional urate lowering therapies or troublesome disease control on standard therapies.

Patient with impaired renal function should be discussed via A and G with nephrology.

Investigations prior to referral, and ideally 4-6w after an attack

- Full blood count
- Serum urate
- Renal and liver function
- X-ray of affected joint
- Calculate the Q risk

Information to include in referral letter

- Date of onset
- Frequency of attacks
- Medication history

Patient information leaflets/PDAs

- Versus Arthritis – information on gout
- NHS leaflet on Gout

References

Gout - NICE Clinical Knowledge Summary

The British Society for Rheumatology Guideline for the Management of Gout

Determination of purine contents of alcoholic beverages using high performance liquid chromatography

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