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Gout

Gout can be defined as arthritis due to deposition of monosodium urate (MSU) monohydrate crystals within joints causing acute inflammation and eventual tissue damage. It has been aptly described as, "... one of the most painful acute conditions that human beings can experience ..."^[1]

The four types of gout are:

- Asymptomatic
- Hyperuricaemia
- Acute gout
- Intercritical gout and chronic tophaceous gout

Classification

The condition can be classified into primary or secondary gout depending on the cause of hyperuricaemia:

- Primary gout occurs mainly in men aged 30-60 years presenting with acute attacks.
- Normally, secondary gout is due to chronic diuretic therapy. It occurs in older subjects, both men and women, and is often associated with osteoarthritis.

Pathogenesis

It affects both upper and lower limbs with acute attacks. Less often it presents with painful, tophaceous deposits (\pm discharge) in Heberden's and Bouchard's nodes.

- Most patients with hyperuricaemia never develop gout and gouty patients may not have hyperuricaemia at presentation.
- Patients can be over-excretors of uric acid, normo-excretors or under-excretors.
- Most cases of primary gout are due to undersecretion.^[2]
- About 10% are due to overproduction.^[3]
- Genome-wide association studies have found that the urate transporter genes SLC2A9, ABCG2 and SLC22A12 modulate serum uric acid (SUA) levels and gout risk.^[4]
- Proinflammatory cytokine interleukin-1 (IL-1) has been identified as having a central role in the inflammatory process associated with gout.^[5]

Epidemiology

- Research using the UK primary care database reported the incidence of gout per 1,000 person-years to be 2.68 (4.42 in men and 1.32 in women) for the years 2000-2007. The prevalence increased with age.^[6]
- Asian populations and people of the Pacific Islands have a much higher prevalence and more severe disease.^[7]
- The male to female ratio is 9:1.^[8] The prevalence increases in women after the menopause although this is partly reduced by hormone replacement therapy.^[9]
- Factors such as the introduction of fructose-high corn sweetener and the rise in obesity have led to a dramatic increase in the incidence of gout in developed countries such as America.^[10]

Risk factors

The European League Against Rheumatism (EULAR) produced evidence-based guidelines in 2006.^[2] It identified the following risk factors:

- Male sex

- Meat
- Seafood
- Alcohol (10 or more grams per day)
- Diuretics
- Obesity
- Hypertension
- Coronary heart disease
- Diabetes mellitus
- Chronic renal failure
- High triglycerides

Other factors since identified include chemotherapeutic drugs, psoriasis and heart failure.^[6] The presence of previous joint morbidity and trauma may influence which joint is affected.

Presentation^[11]^[12]

- The EULAR guidelines for diagnosis suggest that the development of acute pain in a joint which becomes swollen, tender and erythematous and which reaches its crescendo over a 6- to 12-hour period is highly suggestive of crystal arthropathy, though not specifically of gout.^[2]
- 50% of all attacks and 70% of first attacks affect the first metatarsophalangeal joint.
- Other sites often affected are:
 - Knee
 - Midtarsal joints
 - Wrists
 - Ankles
 - Small hand joints
 - Elbows
- The inflammation reaches its peak within 24 hours, often with fever and malaise.



Some patients may only present with connective tissue tophi.^[13]

Signs

- There is florid synovitis and swelling and extreme tenderness with overlying erythema. Untreated, the attack resolves spontaneously over 5-15 days, usually with itching and desquamation of overlying skin.
- Atypical attacks can occur with tenosynovitis, bursitis and cellulitis, with mild discomfort without swelling lasting a day or two.
- Chronic tophaceous gout - in this condition large crystal deposits produce irregular firm nodules mainly around extensor surfaces of the fingers, hands, forearms, elbows, Achilles tendons and ears.

- Typically, tophi are asymmetrical with a chalky appearance beneath the skin. Damage is usually found in the first metatarsophalangeal joints, mid-foot, small finger joint and wrist, with restricted movement, crepitus and deformity.

Differential diagnosis

- Acute attacks - sepsis and other forms of crystal-related synovitis.
- Chronic tophaceous - **rheumatoid arthritis**, generalised nodal **osteoarthritis**, xanthomatosis with arthropathy, multicentric reticulohistiocytosis.

Investigations^[2]

The EULAR guidelines recommend the following evidence-based approach:

- For typical presentations such as inflammation of the first metatarsophalangeal joint (also known as podagra) with hyperuricaemia, a clinical diagnosis can be made with reasonable accuracy but is not definitive unless the presence of uric acid crystals can be demonstrated.
- Demonstration of MSU crystals in synovial fluid or tophi confirms the diagnosis of gout.
- Since gout can present atypically, an opportunity should be taken to examine all samples of synovial fluid aspirated from joints for MSU crystals, even if not inflamed at the time.
- Gram staining and culture of synovial fluid should be arranged, even if MSU crystals are found, since gout and sepsis can co-exist.
- Although a raised SUA level is an important risk factor for gout, the use of SUA as a diagnostic test is limited. It can be normal during acute gout, whilst patients with hyperuricaemia may never develop an attack. Studies suggest that the cut-off point above which a level can be considered raised is 360 $\mu\text{mol/L}$.^[2]
- Renal uric acid secretion (as detected by a 24-hour urine sample) may be helpful in diagnosis, particularly in patients with a family history of young-onset gout, patients whose first attack of gout was under the age of 25 and patients with renal stones. Such patients are likely to be over-excretors of uric acid.
- Radiographs may be useful in chronic gout, when punched-out lesions, areas of sclerosis and, in the latter stages, tophi may be seen. The first lesions usually occur in and around the first metatarsophalangeal joint. CT scanning may be helpful in less accessible areas.^[14] Radiography is less helpful in early gout or during an acute attack.^[2]
- Fasting glucose and lipids should be performed to rule out **hyperglycaemia** and **hyperlipidaemia**, as gout is commonly associated with **metabolic syndrome**.

Management^[12]

General points

An ice pack may be useful, as may rest. The joint should be elevated and trauma avoided.

Pharmacological therapeutic options include:

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Colchicine
- Corticosteroids
- Other primarily analgesic compounds

The choice for a particular patient will depend on:

- Contra-indications
- The gap between onset of symptoms and the start of treatment
- Risks versus benefits

EULAR guidelines recommend colchicine and/or NSAIDs as the first-line option for acute gout.^[15]

The opportunity should be taken to discuss lifestyle issues such as weight loss, exercise, diet, alcohol consumption and fluid intake.^{[12] [16]}

Canakinumab

Canakinumab is a recombinant monoclonal antibody active as an inhibitor of proinflammatory cytokine IL-1. It is licensed for use in patients whose condition has not responded adequately to treatment with NSAIDs or colchicine, or who are intolerant of them. It can be used for the symptomatic treatment of frequent gouty arthritis attacks (at least three in the previous 12 months).^{[17] [18]}

Non-steroidal anti-inflammatory drugs

NSAIDs are the first-line treatment. The sooner medication is started, the more rapid the response.^[19] Consider giving the patient a stock to keep at home.

Indometacin has been traditionally used first-line in the past but there is no convincing evidence to support the use of any particular NSAID.^[20] Eight drugs are licensed for use in gout. Diclofenac, naproxen and indometacin are generally preferred.^[12]

For patients with a high risk of gastrointestinal adverse events, use a gastro-protective agent, simple analgesia, or colchicine.^[21]

Tailor the dose to the needs of the patient, bearing in mind age, comorbidity and interactions with other drugs.^[22] Aim for the highest tolerable licensed dose but be aware of the Commission on Human Medicine's guidance to use NSAIDs for the shortest possible time in view of cardiovascular risk.^[12]

Colchicine

Colchicine is an effective treatment for gout. The British National Formulary (BNF) recommends 500 micrograms 2–4 times daily until symptoms are relieved - maximum 6 mg per course; the course is not to be repeated within three days. In practice, the maximum dose is often limited by the development of toxicity symptoms (nausea, vomiting, diarrhoea).^[17]

Colchicine is particularly appropriate when NSAIDs are poorly tolerated, in patients with heart failure and in those who are on anticoagulants.^{[23] [24]}

The drug can be effective at lower doses.^[25] Titrate up to the maximum licensed dose, according to response.

Corticosteroids

These can be given orally, intramuscularly, intravenously or intra-articularly.^[20] They are useful where NSAIDs or colchicine are contra-indicated.

There are no definitive trials regarding dosage but UK practice is to use short courses of lower doses - 15 mg/day of prednisolone or less.^[12] Randomised trials using 30-35 mg of prednisolone reported a low incidence of side-effects. Systemic steroids are widely used although a Cochrane review found limited evidence of effectiveness.^[26]

An intramuscular corticosteroid injection can be useful in podagra.^[12]

Intra-articular administration of long-acting steroids has been shown, in small trials, to be safe and effective.^{[15] [27]} However, further work is needed to clarify effectiveness.^[28] It can be paired with aspiration of the joint, making it convenient to both aid diagnosis and manage the condition. It is particularly useful for those patients with a severe monoarthritis and contra-indications to NSAIDs and colchicine. It is also useful as it is associated with minimal adverse effects and a lower risk of drug interactions. It should not be undertaken if septic arthritis is suspected.

Analgesics

These are useful where all other drug groups are contra-indicated or as an adjunct for pain relief. Start with paracetamol, with or without codeine, taken regularly rather than 'prn'.^[12]

What next?

If there is no improvement after 2-3 days:

- Review the diagnosis (differentials include septic arthritis, non-urate arthropathy, other arthritides and haemochromatosis).
- Check medicine compliance.
- Increase doses to the maximum.
- Canakinumab.
- If the patient still fails to improve, consider combining treatments, or seek specialist advice.

Emerging treatments

The central role recently identified for proinflammatory cytokine IL-1 has led to the development of inhibitors such as anakinra, canakinumab and rilonacept.^[5] Other advances in genome technology have helped to increase our understanding of urate metabolism and are likely to lead to further therapeutic innovations.^[29]

Complications

- Renal disease:
 - An American study reported that 24% of patients with gout had nephrolithiasis.^[30]
 - Chronic urate nephropathy results from widespread deposition of urate crystals in the interstitium of medulla and pyramids causing inflammation and fibrosis. Reduced glomerular filtration rate is a recognised complication of gout. It was associated with older age of onset, longer duration of gout and higher levels of maximum SUA.^[31]
 - Gout patients who have a 24-hour urinary excretion of uric acid above 780 mmol/L have a 50% risk of developing urate and oxalate kidney stones.^[12] Those with a measured urate excretion greater than 800 mg per 24 hours may benefit from **allopurinol** prophylaxis to prevent urate nephropathy.
- Severe degenerative arthritis.
- Secondary infections.
- Recurrent painful episodes.
- Carpal tunnel syndrome (rare).
- Nerve or spinal cord impingement.

Prognosis

Further attacks will usually occur within the first year, if at all.^[32]

Prognosis is usually good with early treatment.

Sometimes the attacks become more frequent and involve more sites, eventually causing joint damage and chronic pain (usually after ten10 years).

Prevention

Lifestyle modification^[12] ^[33]

Observational studies support the link between lifestyle factors and gout, although it is interesting to note that there is a lack of high-quality evidence from randomised controlled trials either to support or refute the use of lifestyle modifications for improving outcomes in chronic gout.^[16]

Asymptomatic hyperuricaemia is NOT gout and does not warrant management with drugs. However, evidence suggests that hyperuricaemia may well play a role in the development of neurological and cardiorenal pathology. Further research is required to establish whether normalisation of uric acid levels would result in clinical benefit. Irrespective of pharmacological treatment, patients with asymptomatic hyperuricaemia should be given advice on lifestyle modification: [34]

- Drink alcohol sensibly (eg, keep to recommended limits and have three alcohol-free days a week). Beer or spirits should be avoided (there is a particularly strong link with beer, stout and port wines) but wine in moderation is not associated with an increased risk. [19]
- Avoid dehydration.
- Dietary intervention - reduction of purine-based foods.
- Meat or seafood significantly increase the incidence of gout.
- Highest purine levels are found in heart, herring, sardines and mussels.
- Other foods which are very rich in purines include liver, kidneys, yeast extracts and oatmeal.
- Soya foods are also high in purines but are less likely to lead to gout than meats and seafood.
- It is the quantity of purine-rich food consumed that is more important than the absolute purine content in each food.
- Soft drinks containing high levels of fructose can affect the levels of purines and should also be avoided. [35]
- There is no evidence to support a reduction in purine-rich vegetables such as peas, beans, mushrooms or cauliflower. [36]
- Weight reduction - there is increasing evidence to support a link between obesity and gout. [36]
- Regular exercise is beneficial.
- Smoking cessation should be encouraged,

Manage risk factors

These may include:

- Drugs causing hyperuricaemia: [37]
- Thiazides and loop diuretics.
- Low-dose salicylates - eg, aspirin <1 g/day, pyrazinamide, ethambutol, nicotinic acid, ciclosporin.
- Hypertension. [19]
- Impaired renal function. [12]
- Hyperlipidaemia, especially hypertriglyceridaemia. [19]
- Vascular disease. [12]
- Chemotherapy - consider starting prophylaxis before treatment begins. [38]
- Myeloproliferative disease. [39]

NB: aspirin in low doses (75–150 mg/day) has insignificant effects on the plasma urate and should be used as required for cardiovascular prophylaxis.

Prophylactic drugs

General principles

It is important to explain that medication is normally lifelong and regular monitoring is needed. Advise the person that allopurinol may cause acute attacks of gout just after initiating treatment and for some weeks afterwards. Explain that they should start their anti-inflammatory treatment as soon as possible and not stop their allopurinol during acute attacks.

Pharmacological management [17] [40]

Manage recurrent attacks of gout by starting allopurinol after two or more attacks within a year.

- Uric acid-lowering drug therapy should also be offered to patients with: [15]
 - Tophi.
 - Renal insufficiency.
 - Uric acid stones and gout.
 - The need for continuing diuretic treatment.
- Allopurinol should never be started during an acute attack. Wait for 1-2 weeks after the attack resolves.

- Start with a low dose (50-100 mg) and increase in 50-100 mg increments every 2-4 weeks until SUA level is below 300 $\mu\text{mol/L}$.
- Maximum dose is 900 mg daily.
- The timescale for increasing in dose may need to be slower in some patients, with frequent checks of renal function, if renal function is known to be impaired.
- Co-prescribe colchicine or a low dose non-steroidal anti-inflammatory drug (NSAID) to prevent an attack of gout whilst initiating therapy, and continue until after hyperuricaemia has settled (usually a total of three months).
- If an acute attack develops during treatment, maintain the dose but add colchicine or NSAIDs. [41]
- If neither NSAIDs nor colchicine are tolerated, or both are contra-indicated, consider low dose oral prednisolone. [12] However, it may be preferable to seek specialist advice.

Allopurinol

This drug is traditionally the first choice for long-term control of gout. [40] [41] It is not indicated for asymptomatic hyperuricaemia. [27] [41] It is useful where renal function is impaired or renal stones are present. [12]

Febuxostat [17] [42]

This is recommended as an option for the management of chronic hyperuricaemia in gout but only in patients where urate deposition has already occurred and not in cases where urate formation has greatly increased such as malignancy. The Medicines and Healthcare products Regulatory Agency (MHRA) issued advice in 2012 that febuxostat can cause serious hypersensitivity reactions, including Stevens-Johnson syndrome and acute anaphylactic shock. The National Institute for Health and Care Excellence (NICE) has issued guidance recommending that febuxostat should be reserved for patients intolerant of allopurinol, (ie adverse effects severe enough to warrant discontinuation, or to prevent full dose escalation for optimal effectiveness).

Sulfinpyrazone

This can be used as an alternative to allopurinol if toxicity occurs, or as an adjunct in resistant cases. It can be difficult to obtain (a generic form is available) and is contra-indicated in renal failure and urolithiasis.

The dose is 200-800 mg/day in patients with normal renal function.

Colchicine

Although not a urate-lowering drug per se, colchicine is sometimes prescribed at low dose in early gout - to 'buy time' in patients undergoing lifestyle modification, before a commitment to urate-lowering drugs is made.

Other options

Low-dose corticosteroids and NSAIDs have also been used to buy time, in the same way as colchicine. [27]

Probenecid is a less powerful uricosuric agent and is relatively contra-indicated in urolithiasis.

Benzbromarone can also be used in patients with mild/moderate renal insufficiency at a dose of 50-200 mg/day. However, it carries a small risk of hepatotoxicity. [15]

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