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PRACTICE POINTER

Ask an expert: Gout

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What you need to know

- Preferred treatments for flares of gout include non-steroidal anti-inflammatory drugs (NSAIDs), colchicine, and corticosteroid drugs
- Do not offer NSAIDs to older patients with multiple comorbidities, such as cardiovascular disease and chronic kidney disease
- Consider offering urate lowering therapy early in the course of the disease, and aim for a target serum urate level below 360 µmol/L (6 mg/dL)
- Evidence to indicate that any specific diet prevents flares or lowers serum urate levels is insufficient; therefore advise patients to follow a healthy, balanced diet

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Rheumatologist Edward Roddy provides expert answers to GPs' questions on common challenges with managing gout, including treating flares, initiating urate lowering therapy in patients and appropriate target serum urate levels, and counselling patients on the diagnosis and treatment of gout.

Test yourself

You are treating a 62 year old woman for her recurrent flares of gout and tophi with allopurinol. Over a period of four months, you gradually increase the dose of allopurinol. Her serum urate level is lowered successfully to 297 µmol/L. She then experiences a further flare of gout, which affects her first metatarsophalangeal joint.

With regard to this new flare of gout, which one of the following is correct?

- Treat the flare with colchicine and continue allopurinol during the flare
- Treat the flare with colchicine and stop allopurinol during the flare
- The occurrence of a flare suggests that treatment is not working, and you should increase the dose of allopurinol
- The occurrence of a flare suggests that treatment is not working, and you should switch the allopurinol to febuxostat
- The occurrence of a flare suggests that treatment is not working, and you should refer the patient to a specialist.

What do you recommend in terms of management and follow-up for patients with a gout flare?

Most flares should respond quickly to one of the preferred treatments (described below), which are recommended in UK national guidance.[1] In addition, the guidelines recommend applying an ice pack to the affected joint.[1 2]

A non-steroidal anti-inflammatory drug (NSAID)

There is no evidence that any particular NSAID is superior to any other, so offer any immediate release NSAID at full dose (eg, naproxen) guided by your patient's preference, renal function, and comorbidities.[3] Avoid indometacin because of the risk of gastrointestinal, renal, and cardiovascular adverse events.[3-5] Offer gastro protection in addition to a NSAID, for example 20 mg of omeprazole.[3]

Low dose colchicine

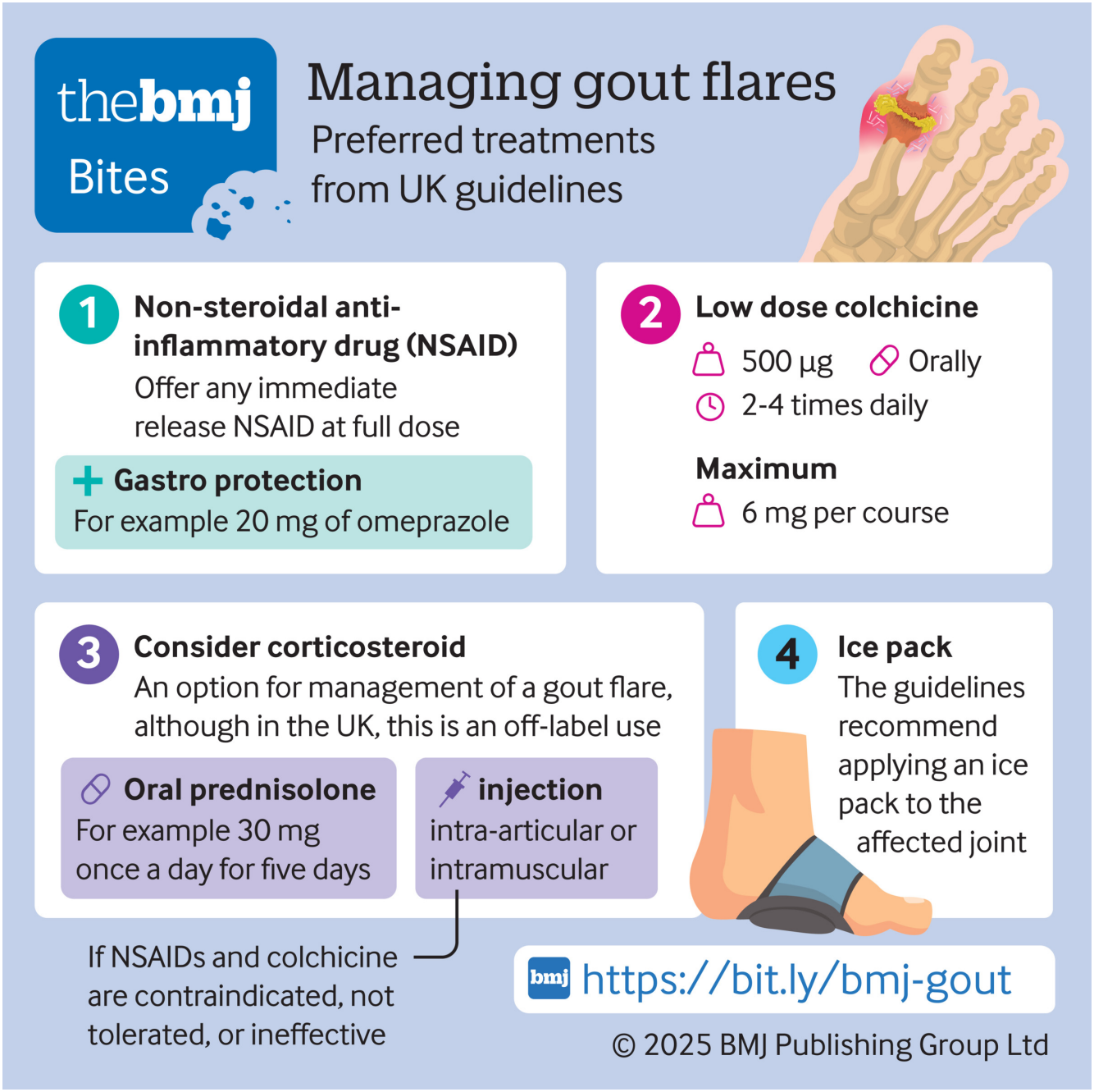
Offer colchicine at a dose of 500 µg orally two to four times daily until symptoms are relieved, up to a maximum of 6 mg per course.[6] A randomised trial compared naproxen or colchicine for people with a gout flare and found no difference between the groups in pain intensity for seven days, but adverse events were more common in the colchicine group with the most common one being diarrhoea.[7]

Short course of a corticosteroid drug

A corticosteroid drug is an option for management of a gout flare, although in the UK, this is an off-label use.[1]

- Oral prednisolone is an effective option, for example 30 mg once a day for five days.[3 8]
- You might consider an intra-articular or intramuscular injection of a corticosteroid drug if NSAIDs and colchicine are contraindicated, not tolerated, or ineffective, especially in patients who are older and have multiple comorbidities.

This article was adapted from a BMJ Learning module Ask an expert: Gout: https://new-learning.bmj.com/course/10055705



Follow-up

Guidelines from both National Institute for Health and Care Excellence (NICE) and the British Society for Rheumatology recommend reviewing a patient after a gout flare has settled, or four to six weeks later.[1 3] This is to:

- Measure the serum urate level and renal function
- Provide information on how to self-manage and reduce the risk of future flares
- Assess comorbidities including cardiovascular risk factors (eg, obesity, hypertension, hyperlipidaemia, diabetes) and chronic kidney disease
- Assess predisposing lifestyle factors such as excessive alcohol consumption
- Review medications (eg, diuretics)
- Discuss the benefits of long term urate lowering therapy.

Referral criteria

In general, if the diagnosis of gout is uncertain or if treatment is contraindicated, not tolerated, or ineffective, you should refer the patient to a specialist, as outlined in UK national guidance.[1] The most important alternative diagnosis that should not be missed is infection: septic arthritis (box 1) or osteomyelitis. If there is clinical suspicion of infection, then refer the patient as an emergency to a rheumatologist or an orthopaedic surgeon, according to the local care pathway.

Box 1: Distinguishing septic arthritis from a gout flare

Flares of crystal arthritis (either gout or pseudogout) are characterised by sudden onset, severe joint pain that reaches its peak intensity within 12 to 24 hours of onset, joint swelling and erythema, and complete resolution, typically within one to two weeks.[1 4]

If these features are present, and the first metatarsophalangeal joint is affected, then gout is the most likely diagnosis.[9] Onset of septic arthritis is typically less acute.

Traditional markers of infection such as fever and raised white cell count, erythrocyte sedimentation rate, and C reactive protein might be absent or only modestly raised in people with septic arthritis.[10] Conversely, fever might be a feature of a gout flare, and white cell count, erythrocyte sedimentation rate, and C reactive protein might be grossly raised. As a result, traditional markers of infection are not useful to distinguish septic arthritis from gout flares.[11 12]

The only way to definitively distinguish septic arthritis from a gout flare is to aspirate the affected joint before starting antibiotics, and arrange crystal examination, Gram stain, and culture of the aspirated fluid.[9]

Non-infective causes of an acute hot, swollen joint include calcium pyrophosphate crystal deposition and reactive arthritis. Therefore, microscopic examination of synovial fluid that has been aspirated from the affected joint provides an opportunity to confirm a diagnosis of crystal arthritis and differentiate gout from calcium pyrophosphate crystal deposition. The necessary experience and practical skills to perform joint aspiration might not be present in all settings (eg, primary care), and you might need to refer a patient to a specialist for this.

In an older patient with multiple comorbidities, what is the best option to treat a gout flare?

NSAIDs are contraindicated for use in older patients with multiple comorbidities (eg, cardiovascular disease and chronic kidney disease). In accordance with the British National Formulary, in a gout flare, the normal dose of colchicine is 500 µg orally two to four times daily until symptoms are relieved, up to a maximum of 6 mg per course.[6] In an older patient with comorbidities, you can cautiously treat a flare with colchicine, but increase the interval between doses (ie, 500 µg two times daily).[6] Review the drug interactions that potentially increase the toxicity of colchicine (box 2).

Box 2: Possible drug interactions that increase the risk of colchicine toxicity[4]

- Amiodarone
- Ciclosporin
- Digoxin
- Diltiazem
- Fibrate drugs
- Antifungal drugs (itraconazole, ketoconazole)
- Macrolide antibiotic drugs
- Protease inhibitor drugs
- Statin drugs
- Verapamil

Corticosteroid drugs are an effective treatment for a gout flare and provide a valuable treatment option in patients who cannot tolerate, or have contraindications to, NSAIDs and colchicine.

- According to small observational studies, expert consensus, and clinical experience, intra-articular aspiration and injection of a corticosteroid drug are highly effective in a monoarticular gout flare and could be the treatment of choice in patients with a gout flare in a large joint and comorbidity.[3 5] However, there are no randomised controlled trials of intra-articular injection of a corticosteroid drug for a flare of gout, and it is an off-label indication.[13] You should refer a patient to a specialist for this treatment.
- Intramuscular injection of a corticosteroid drug is a useful option in patients with oligoarticular or polyarticular flares.[3]
- Oral prednisolone is also an option. Four randomised controlled trials of oral prednisolone for a gout flare show comparable pain relief to NSAIDs.[8 14 -16] In these trials, a four or five day course of prednisolone was given at a dose of either 30 mg or 35 mg daily. Following these trials, a clinically credible approach is to offer a short course of prednisolone (eg, 30 mg daily for 5-7 days). In my experience, a rebound gout flare after stopping oral steroid drugs does not appear to be a common problem.

When should I offer a patient urate lowering therapy to prevent a gout flare?

UK national guidelines recommend that you should offer urate lowering therapy to all patients with gout who have[1]:

- Multiple or troublesome flares
- Chronic kidney disease stages 3 to 5
- Diuretic therapy
- Tophi
- Chronic gouty arthritis.

The British Society of Rheumatology guidelines also recommend urate lowering therapy for patients with[3]:

- Uric acid urolithiasis
- Primary gout starting at a young age
- Evidence of joint damage on radiograph.

Both guidelines recommend making patients aware of the option of urate lowering therapy after their first or subsequent flare.[1 3] In my experience, many people are not offered urate lowering therapy until they have frequent troublesome flares or have developed joint damage or tophi.

With respect to timing, offer urate lowering therapy two to four weeks after a gout flare has settled, but if flares are more frequent, it can be started during a flare.[1] There is growing consensus that you should consider starting urate lowering therapy early in the course of the disease, and discuss this with your patient.[1 3 4] This is because most patients with gout experience recurrent flares, and imaging studies show chronic crystal deposition both at the time of the first flare, and in hyperuricaemic patients who are yet to have a gout flare.[17 -19] Two small placebo controlled randomised trials found that starting allopurinol during a flare, first with flare treatment and then with continued colchicine or NSAID prophylaxis, did not increase pain, inflammation, or flare recurrence when assessed at 28 to 30 days.[20 21]

Delaying the start of urate lowering therapy, however, until after the flare has settled and the patient is no longer in pain, allows the patient to better absorb information about their treatment.[4] For

example, the patient should know how to treat a further flare, and not to discontinue urate lowering therapy if a flare occurs.[3] Continuing urate lowering therapy once a flare has occurred does not affect the duration or severity of the flare. Furthermore, if it is stopped during a flare, the patient might be reluctant to start it again afterwards because of the perception that the drug has worsened their gout. If urate lowering therapy is restarted, start at a low dose and gradually increase it again.

Should urate lowering therapy be used to reach a target serum urate level?

The objective of urate lowering therapy in all patients is to lower serum urate below its physiological saturation threshold in body tissues (the target level). Lowering serum urate to below this level causes existing crystals to dissolve and prevents new crystals from forming. Treating to target stops flares, shrinks tophi so they eventually disappear, and prevents long term joint damage.[22-24] Even once the therapeutic target is achieved, it can take several months for existing monosodium urate crystals to clear, flares to stop, and tophi to reduce in size.

The NICE guideline recommends that you aim for a target serum urate level below 360 µmol/L (6 mg/dL).[1] The guidelines also suggest a lower target serum urate level, below 300 µmol/L (5 mg/dL), for people with gout who:

- Have tophi or chronic gouty arthritis
- Continue to have ongoing frequent flares despite having a serum urate level below 360 µmol/L (6 mg/dL).

Start with a low dose of urate lowering therapy and measure serum urate levels every four weeks to guide dose increases, as tolerated, until the target serum urate level is reached.[1]

UK national guidelines recommend offering either allopurinol or febuxostat as the preferred treatment when starting urate lowering therapy, taking into account the patient's comorbidities and preferences.[1] In patients who have major cardiovascular disease (eg, previous myocardial infarction or stroke, or unstable angina), offer allopurinol as the preferred treatment.

How to manage a flare when starting urate lowering therapy

Warn your patient that they might experience a flare when starting or titrating urate lowering therapy because all urate lowering drugs can cause a flare of gout.[25] Advise patients that such a flare is a "sign of successful treatment" rather than a side effect of the medication. Patients should not stop the urate lowering drug in the event of a flare.

The following might help to reduce the risk of a flare of gout:

- Start urate lowering therapy at a low dose and titrate the dose up slowly[26]
- Consider co-prescribing prophylactic medication, such as colchicine, but be aware of drug interactions (box 2).[1] If colchicine is contraindicated, not tolerated, or ineffective, consider a low dose NSAID, or a low dose oral corticosteroid drug[1]
- As an alternative to prophylaxis, you can prescribe a short course of colchicine, an NSAID, or an oral corticosteroid drug for the patient to keep in reserve in case a flare occurs.[1]

If neither colchicine, NSAIDs, nor corticosteroid drugs are suitable, an interleukin 1 inhibitor could be considered to prevent gout flares when starting or titrating urate lowering therapy, but this would require referral to a rheumatology service before prescribing.[1]

Do you have any tips on how we can counsel patients in primary care?

All patients with gout should be given a clear verbal explanation of the nature, causes, associations, consequences, and treatment of gout.[1 27] It is helpful to support this with written information, such as a patient information booklet on gout from Arthritis UK.[28] Box 3 outlines examples of key messages I give to patients related to the cause of gout, how it affects people, and how it is treated.

Box 3: Examples of key messages when discussing gout with patients

Cause

- People get gout because the level of urate in their blood is too high, causing urate crystals to form in the cartilage lining the joint. If urate levels remain high, crystals form slowly but continuously, often without causing symptoms
- Urate levels can be high for many reasons including a person's medical conditions, the drugs they take, their genetic make up, and whether they are overweight or obese. Offer the patient an individualised explanation of their personal risk factors for gout
- Gout is often assumed to be caused by drinking too much alcohol or eating too much red meat and seafood, but this is often not true for many people

Effects

- Occasionally, some crystals "spill out" into the joint causing a flare of gout when the joint becomes painful, inflamed, and swollen
- Over many years, these flares can become more frequent and spread to affect other joints
- Lumps of crystals called tophi can form over the joints under the skin and cause damage to the joint cartilage in the joint and bone. This damage to the joint can cause regular day to day pain

Treatment

- Flares can be treated with colchicine, NSAIDs, or corticosteroid drugs
- The aim of treating gout (with drugs such as allopurinol) in the long term is to lower the level of urate below the target level. If the level can be lowered enough and kept low, then new crystals will stop forming and existing crystals will dissolve and eventually disappear altogether. Drugs to lower urate levels usually need to be taken for life
- When there are no crystals left, flares of gout stop, tophi reduce in size (and may eventually disappear), and joint pain due to gout improves
- Starting allopurinol can sometimes set off a gout flare. If this happens, it is not a side effect but a sign that allopurinol is working. The flare has occurred because the urate level is being lowered and crystals are starting to dissolve. The flare can be treated in the same way as any other flare of gout and the allopurinol does not need to be stopped

Risks of stigma about alcohol and diet might prevent people from seeking healthcare for gout, so questions about diet and alcohol consumption should be asked sensitively. Although people often assume gout is caused by excessive consumption of alcohol and certain foods such as red meat and seafood, studies show that the risk of gout increases only with high levels of consumption.[29] People with gout who are overweight or obese or drink alcohol excessively should be supported to lose weight and reduce their alcohol consumption, respectively. There is insufficient evidence that any specific diet prevents flares or lowers serum urate levels and people with gout should be advised to follow a healthy, balanced diet.[1]

This is a lot of information to convey in a typical 10 to 15 minute consultation, so it should be reinforced at future appointments.[1] It could be effectively delivered in a clinic led by nurses, as shown by a randomised trial comparing care led by nurses with usual care led by GPs in people with gout.[30] Nurses delivered an individualised package of care over multiple appointments, consisting of holistic assessment, discussion of perceptions of illness, and providing information about gout including its nature, causes, associations, consequences, and treatment options. The nurses shared decision making with the patient, which was combined with gradual escalation of urate lowering therapy to achieve the target level of serum urate. Results at two years showed that with care led by nurses, 95% of participants achieved a target urate level compared with 30% of participants with usual care led by GPs, which was statistically significant (risk ratio 95% *v* 30%, relative risk 3.18, 95% confidence interval 2.42 to 4.18, P<0.001). Patient centred outcomes such as number of flares, tophi, and quality of life were also better with care led by nurses.

Test yourself (revisited)

A (Treat the flare with colchicine and continue allopurinol during the flare) is the correct answer.

This patient has achieved the therapeutic target serum urate level, indicating that she is taking an appropriate dose of allopurinol. However, even once the therapeutic target is achieved it can take several months for existing monosodium urate crystals to clear, flares to stop, and tophi to reduce in size, although flares will become less frequent. Treat this flare as any other.

Although it is standard advice to delay starting urate lowering therapy until the flare has resolved in order to prevent worsening of the flare, do not stop allopurinol if a flare occurs once urate lowering treatment has already begun; instead, continue at the same dose alongside the treatment for the flare, in this instance with colchicine.[3]

There is no need to switch the allopurinol to febuxostat. UK national guidelines suggests that if people have ongoing symptoms, you can consider referring them to a rheumatologist.

Since she has only had one flare you should be able to manage her in primary care and referral to a specialist is not warranted at present.

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Further details of *The BMJ* policy on financial interests are here: https://www.bmj.com/about-bmj/resources-authors/forms-policies-and-checklists/declaration-competing-interests

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