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Improving management of gout in primary care:

a new UK management guideline

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INTRODUCTION

Gout is the most prevalent inflammatory arthritis, affecting 2.5% of adults in the UK.[1] Despite its prevalence and the availability of potentially 'curative' urate-lowering therapies (ULT) such as allopurinol, gout remains undertreated in both primary and secondary care. Under half of patients receive ULT and many do not have ULT escalated sufficiently to reduce serum uric acid (sUA) below recommended target levels.[1]

THE 2017 GUIDELINE

The British Society for Rheumatology and British Health Professionals in Rheumatology (BSR/BHPR) have recently revised their evidence-based guideline for the management of gout,[2] first published in 2007.[3] The multidisciplinary guideline development group agreed key clinical management questions and undertook a systematic literature review and Delphi process to inform evidence-based consensus management recommendations. These were grouped into three categories: management of acute attacks ($n = 6$), modification of lifestyle and risk factors ($n = 5$), and optimal use of ULT ($n = 10$). The guideline has been reviewed and endorsed by the Royal College of General Practitioners (RCGP).

MANAGEMENT OF ACUTE ATTACKS

For acute attacks of gout, a non-steroidal anti-inflammatory drug (NSAID) (with gastro-protection) or low-dose colchicine 500 mcg two to four times daily are recommended as first-line treatment, depending on patient preference, renal function, and comorbidities. In patients who are intolerant of, or have contraindications to, NSAIDs and colchicine, intra-articular, intra-muscular, or oral corticosteroids are recommended (for example, oral prednisolone 35 mg daily for 5 days). Patients should be advised to treat attacks as soon as possible after onset to minimise severity and impact. Non-pharmacological adjunctive treatment with bed-cages, elevation, rest, or topical ice are also recommended.

LIFESTYLE AND RISK FACTORS

People with gout are at increased risk of cardiovascular disease and commonly have risk factors such as hypertension, diabetes mellitus, and dyslipidaemia. They should therefore be screened for such risk factors annually and managed appropriately. Thiazide and loop diuretics are an important cause of hyperuricaemia and alternatives should be considered to treat hypertension in people with gout. Although the effect of dietary modification on gout is unclear, patients should observe a well-balanced diet and avoid excessive consumption of alcoholic drinks, high-purine foods (for example, meat or seafood), and sugar-sweetened soft drinks. Those who are overweight should be supported to lose weight gradually and maintain weight loss.

OPTIMAL USE OF URATE-LOWERING THERAPY

The most important change from the previous guideline[3] is that ULT should be explained and offered to all patients with gout at first diagnosis, rather than waiting for complications such as frequent attacks, chronic symptoms and disability, or tophi to develop. Gout is now considered to be a chronic inflammatory arthritis rather than an acute episodic condition, because crystal deposition occurs early in many hyperuricaemic patients who are yet to develop gout, and sub-clinical joint inflammation frequently persists between attacks. Earlier initiation of ULT prevents attacks from becoming more troublesome and achieves clinical remission sooner. In a proof-of-concept study in primary care, all participants chose to receive ULT after a full explanation of gout and the benefits of treatment, with excellent adherence and achievement of target sUA levels.[4]

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Competing interests

Graham Davenport has received honoraria for *ad hoc* advisory board meetings relating to gout from AstraZeneca. George Nuki was a member of the Independent Disease Monitoring Committee for trials of Lesinurad (Ardea/AstraZeneca) and has received honoraria for advisory boards from Grünenthal and Menarini, and research funding from Menarini for the FAST trial. Christian D Mallen, Michelle Hui, and Edward Roddy have declared no conflicts of interest.

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Allopurinol remains the first-line ULT and should be commenced at low dose (for example, 50–100 mg daily) and increased gradually in 50–100 mg increments every 4 weeks until the target sUA level has been achieved. The maximum dose of allopurinol in the UK is 900 mg daily in divided doses, although the starting, incremental, and maximum doses are lower in patients with chronic kidney disease, depending on the degree of renal impairment.

The biochemical treatment target is to reduce the sUA level below 300 µmol/L, which is unchanged from the previous BSR/BHPR guideline[3] but lower than that recommended in European and American guidelines, which advocate reducing to below 360 µmol/L.[5,6] This more stringent target is recommended because, although lowering below 360 µmol/L prevents further urate crystal formation and dissolves existing crystals, causing attacks to cease and tophi to shrink, the speed at which these occur is greater at lower sUA levels. However, recent studies have suggested that hyperuricaemia may protect against neurodegenerative diseases such as dementia and Parkinson's disease. Whether lowering sUA levels with ULT to treat gout increases the risk of these diseases is unknown. In the face of such uncertainty, the guideline advises that very low sUA levels should be avoided in the long term, and that, although the initial treatment target should be below 300 µmol/L, once tophi have resolved and the patient remains free of symptoms, the dose of ULT can be adjusted to maintain the sUA below 360 µmol/L.

Since the previous guideline was published in 2007,[3] febuxostat has been approved by the National Institute for Health and Care Excellence (NICE) for the management of hyperuricaemia in people with gout. The revised guideline recommends that febuxostat can be considered in patients in whom allopurinol is not tolerated or whose renal impairment prevents allopurinol dose escalation sufficient to achieve the therapeutic target, in accordance with the NICE technology appraisal. Febuxostat is not recommended for use in people with ischaemic heart disease or congestive cardiac failure.

Acute attacks of gout commonly occur following initiation of ULT. Such attacks do not require discontinuation of ULT and indicate that sUA lowering and crystal dissolution have commenced. The guideline recommends that colchicine 500 mcg b.d. or o.d. should be considered as prophylaxis against attacks for up to 6 months when initiating or uptitrating any ULT. This is particularly important when starting febuxostat that, in its two licensed doses of 80 mg and 120 mg daily, lowers sUA to a greater extent than fixed-dose allopurinol 300 mg daily and therefore more commonly triggers attacks of gout. In patients intolerant of colchicine, a low-dose NSAID with gastroprotection can be used in the absence of contraindications.

EFFECTIVE MANAGEMENT OF GOUT

The revised guideline strongly emphasises the importance of patient education and provision of information about gout and its treatment to all patients. Inadequate provision of information is an important barrier to effective management, and adherence to ULT and attainment of target sUA levels is best achieved by including high-quality information in an evidence-based package of care.[4] Practitioners in both primary and secondary care should take gout seriously and ensure that all patients with gout are aware of the benefits of ULT, which should be initiated, escalated, and monitored appropriately to achieve target sUA levels.

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