Gout

Following the release of the 2017 Gout guideline from the British Society for Rheumatology, we ask if there is a role in the management of gout for the General Practice Nurse.

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Background

People commonly think of gout as a 'rich man's disease' caused by dietary excess and overconsumption of alcohol, evoking stereotypical images of historical figures such as King Henry VIII [1]. This negative perception of the condition with an historical link to a life of dissipation leads to underestimating the impact of gout[2]. Characterised by recurrent sudden flares of excruciating joint pain, swelling and inflammation, poorly treated gout can cause disabling joint damage and is associated with multiple comorbidities (renal impairment, metabolic syndrome, heart disease and depression)[3].

The prevalence of gout has increased dramatically in the UK and now affects 2.5% of the UK adult population; making it the most prevalent form of inflammatory arthritis[4,5]. It is four times more common in men than women and is more common in

older age groups, reaching its peak prevalence between the ages of 80-84 where it affects 15% of men and 6% of women[3].

The majority of patients with gout are managed in primary care within general practice. Gout is considered to be the only curable form of arthritis[3] yet GP-led treatment for gout is sub-optimal and not concordant with current national and international recommendations[3]. In view of this, a new approach is required to improve the management of gout and therefore we ask 'is there a role for the GPN?'.

So what is gout?

Essentially gout is a form of arthritis caused by the formation of monosodium urate crystals in and around joints. Gout results from sustained elevated serum urate (often reported as serum uric acid (sUA)) levels (hyperuricaemia) beyond a saturation point which can lead to formation and deposition of urate crystals.

Myths and misconceptions that hyperuricaemia and gout is simply caused by excessive amounts of alcohol and over-eating do not tell the whole true story. Whilst risk factors for gout include obesity; excess consumption of beer or spirits, red meat, seafood, fructose-sweetened beverages, and fruit juices; in many patients gout is due to genetic factors; comorbid medical conditions such as hypertension, chronic kidney disease and obstructive sleep apnoea; or some medications (particularly diuretics) rather than life-style factors[6].

Hyperuricaemia is caused by over-production or renal under-excretion of urate[5]. Over 90% of people with gout have hyperuricaemia because of renal under-excretion of urate. Over a period of time, monosodium urate crystals form in and around the joints and can eventually trigger an acute gout flare. Such flares usually present with an excruciatingly painful, red, hot and swollen joint(s). The skin often appears shiny[7]. Acute flares come on rapidly (typically overnight) with symptoms peaking within 12-24 hours and usually subsiding within one to two weeks[5]. Gout most commonly affects the first metatarsophalangeal (MTP) joint but can also present in other joints within the foot as well as the knees, ankles, elbows and hands[8]. It usually affects a single joint at a time, but several joints can be affected at once (polyarticular gout). Monosodium urate crystals can also form outside of the joint and under the skin

forming discrete firm lumps called tophi[8]. Gout can also cause chronic joint pain and erosive destructive arthritis.

Diagnosing gout: Diagnosis is most commonly based on symptoms, clinical history and an examination of the affected joint. An acute flare in one joint with excruciating pain that peaks within 24hrs is highly characteristics of gout, particularly when the first MTP joint is affected[3]. When diagnostic uncertainty exists, joint aspiration and microscopy of synovial fluid can give a definitive diagnosis. Although the serum urate level is elevated in people with gout, it should be noted that serum urate on its own is not a useful diagnostic test as most people with hyperuricaemia do not have gout and serum urate can be falsely low when measured during an acute flare[3].

Managing gout: Due to the extreme pain associated with acute flares, all patients should be offered or advised to use analgesia that is commenced as early as possible. Treatment options include non-steroidal anti-inflammatory drugs (NSAIDs) (such as naproxen) with a proton-pump inhibitor, low-dose colchicine, or oral corticosteroids, according to patient preference, past experiences and comorbidities. Non-pharmacological treatment such as ice-packs placed over the affected joint can be useful adjunctive therapy[9].

The British National Formulary recommends that Colchicine should be used in doses of 0.5mg two to four times daily. Diarrhoea is a common side-effect particularly at higher doses[3]. Careful consideration should be given to drug interactions with Colchicine. Statins should be stopped whilst taking colchicine and there should be an awareness of interactions with ciclosporin, ketoconazole, ritonavir, clarithromycin, erythromycin, verapamil and diltiazem[10]. Oral prednisolone at a dose of 30-35mg for five days has been shown to be as effective as NSAIDs[7].

Gout is often viewed as an acute episodic condition which results in a focus on the short-term treatment of acute flares and detracts from ”curative” long-term management of the condition and therefore, the most serious complications are not prevented[2]. Monosodium urate crystals can be eliminated through a combination of effective patient education and evidence-based ‘treat-to-target’ urate-lowering therapy (ULT) such as allopurinol[3].

Long-term use of ULT is strongly recommended when patients have recurrent flares, tophi or chronic arthritis[11]. However, recently updated British and European guidelines state that the benefits of ULT should be explained and offered to all patients at diagnosis[11]. Optimal ULT involves a treat-to-target approach to reduce serum urate below the physiological saturation threshold, thereby preventing further crystal formation and dissolving existing crystals to prevent further flares[3]. European and American guidelines recommend lowering serum urate below 360µmol/L[11,12] whereas BSR advocates a more stringent target below 300µmo/L which leads to more rapid clinical improvement[3,13].

Allopurinol should be commenced at an initial dose of 100mg per day (50mg per day in the presence of stage-4 CKD) with a gradual monthly increase in dose (50/100mg increments) until the therapeutic target is achieved. The maximum daily dose of allopurinol is 900mg although the maximum daily dose is lower in patients with chronic kidney disease[5].

Despite clear guidance and recommendations, only 30-40% of people with gout take ULT and only 40% of those that do have the dose escalated to achieve the 360µmol/L target[14,15]. Such suboptimal treatment causes frequent flares, chronic pain and disability, long-term joint damage, and impaired quality of life[3].

Continual monitoring and follow-up: Once ULT has been commenced patients should be followed-up and monitored every 4-6 weeks with regular serum urate measurements until the target serum urate level has been achieved. Once the serum urate target is reached, levels should re-checked annually as part of on-going assessment. Patients may still experience acute flares during the first two years after the target serum urate level is reached whilst existing crystal deposits dissolve, but these flares should eventually cease[3]. The critical serum urate level (the saturation point) is 360 µmo/L although this will show as ‘in range’ and within normal limits on practice computer screens, so it is crucial to know whether or not serum urate is above or below this critical level[8].

Is there a role for the GPN?

It is clear that although there are multiple single disease guidelines for patients with multiple morbidities, the core interventions for many of the long-term condition (LTC)

guidelines have considerable overlap. Gout is no exception and the BSR targets three key areas in their 2017 guideline update:

- Management of acute attacks
- Modification of lifestyle and risk factors
- Optimal use of urate-lowering therapy

A revision of their earlier guideline was required because of the availability of the new pharmacological treatments, increases in incidence and prevalence, sub-optimal management in both primary and secondary care, and a better understanding of barriers to effective care[16,17].

The role of the GPN has evolved such that they are now considered fundamental to the care of patients with LTCs[18] and policy dictates that future expansion should bring new aspects of complex care within the GPN role, including assessment and diagnosis[19]. Over recent years, the role of GPNs has already extended within primary care. In 2014, NHS England published its 'Five Year Forward View' in which the need for a more flexible workforce to 'future proof' the NHS was made explicit[20]. GPNs have extended their working boundaries to take on roles previously considered outside their remit and are increasingly representing the first point of contact within primary care[21]. Specifically-trained nurses can provide care that is at least equivalent to that provided by GPs for 'treat-to-target' LTCs such as diabetes[22].

How GPNs might best become involved in the management of gout is not certain, but there are sufficient parallels with the treat-to-target approach in conditions such as diabetes to suggest that similar approaches could work. Gout information should be tailored to the individual patient, targeting its management (i.e modification of lifestyle and risk factors), and initiation and upward dose titration of ULT as per BSR recommendations. GPNs already have these skills but not the knowledge to utilise them for gout. However, a nurse-led approach to the management of gout has been tested[23] and a recently-completed UK community-based randomised controlled trial[24] demonstrated that nurse-led care of people with gout can be superior to standard GP care and such a model of care is likely to be cost-effective. Table 1 provides a case study of a patient whose gout may be identified opportunistically.

Table 1. Case Study, Fred

Fred is a 64 year old gentleman who has presented for an annual hypertension review with the GPN. On examination: BP 138/82 Pulse 78 regular BMI 31 Alcohol 16 units a week CVD risk assessment 17.3% **Last serum uric acid level 512 µmol/L** Medication: Lercanidipine 20mg, **Allopurinol 100mg** Investigations: U+Es, LFTs, Lipids, HBA1c, fasting glucose, serum UA Identifying the gout medication the GPN asks about flares. Fred reports having two acute attacks of gout in the last 6 months that were excruciating and debilitating. He feels anxious about the risk of further attacks and describes feeling 'down and depressed'.

The GPN in this case study is ideally placed to address the lifestyle issues raised within this hypertension consultation such as weight management, alcohol intake and the fact that ideally the BP would benefit from being reduced slightly. However, the GPN has the further opportunity to enquire and encourage the patient to discuss his medication regime, noting Fred to be taking Allopurinol, this is an indicator and opportunity to discuss gout and to find out how he is managing.

Fred discloses that he has had two flares since his last annual LTC review. He describes a classical presentation of pain, inflammation and swelling in his right great toe. He self-managed both episodes with non-steroidal anti-inflammatories.

CVD risk assessment found that Fred was at moderate risk of a cardiovascular event and this lifestyle intervention was crucial to support Fred's understanding of the potential complications of gout if untreated. It had been around 2-3 months since his last flare and Fred's serum urate level was measured and found to be 512µmol/L, his renal function was within normal range

Follow up

Following discussions with Fred, it was agreed to increase the Allopurinol dose to 200mg daily with a repeat serum urate level at 4 weeks. Further titration of Allopurinol up to a maximum of 900mg daily (as renal function is normal) would continue 4-6 weekly until the serum urate levels were below 300 µmol/L.

NSAIDs or Colchicine cover would be provided for potential acute flares during the titration period. This could be managed and supported by the GPN and is protocol driven.

The GPN role is fundamental to the management of gout for patients such as Fred, their expertise in lifestyle advice and intervention with regard to LTC management is an accepted element of the role and knowledge and experience is transferable to the management of gout. Fred gained an understanding of the lifestyle implications of weight, alcohol and CVD risk which supported him to look at behaviour change and modification. GPNs are ideally placed and experienced to facilitate this lifestyle modification and where necessary to discuss the potential use of statins in line with the NICE guidance for primary prevention of cardiovascular disease[25]. Further risk assessment should be undertaken following these changes and if risk remains above 10% a statin should be offered to support the patient

Within 12 weeks and using 400mg Allopurinol, Fred's serum urate level had reduced to 298 µmol/L, BP was 136/78, and BMI 30.2. He had had no further flares in this 3-month period and had a good understanding of the importance of managing this condition with lifestyle modifications and adherence to ULT.

Conclusion

Alongside the recent publication of new guidelines for the management of gout[11], two recent studies have demonstrated the effectiveness of suitably trained nurses to manage the condition[23,24]. For this to be so, a package of care should include individual patient education and patient involvement in management decisions to improve lifestyle and treat-to-target ULT adherence. GPNs are in a unique position to make a difference for patients with gout and should be encouraged to 'take the first step' to better understand gout and its management by raising the subject with their GPs and practice managers.

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