Management of gout

In their article discussing gout, Drs Robinson and Stamp advise using non-steroidal anti-inflammatory drugs (NSAIDs) as first-line treatment for acute gout (AFP May 2016).1 I did this for my first 25 years in general practice but for the last 15 years have used nothing but prednisone.

Patients will report that improvement in pain and swelling occurs within hours, as opposed to days with NSAIDs. In many cases, they need only 25 mg at 12-hourly intervals for three or four doses. I have never encountered any side effects with this regime apart from reminding patients never encountered any side effects with this regime apart from reminding patients

I am in agreement with the authors that, when initiating allopurinol, it is essential to use NSAIDs concurrently (eg diclofenac 50 mg daily or twice daily for the first six weeks or so) to prevent flare-ups of gout.

When used appropriately, prednisone truly is a desert island drug.

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Reply

We thank Dr Golder for his correspondence related to our gout management article.1 The American College of Rheumatology recommends the use of corticosteroids, NSAIDs or colchicine as first-line treatment, and advises clinicians to make a decision based on the number of involved joints and severity.2 We agree with Dr Golder that steroids can be very effective in the management of gout and are a useful therapeutic option.

Regarding Dr Golder’s observation of corticosteroid superiority, this question has been addressed in a large trial involving 120 patients, which compared naproxen 500 mg twice a day and prednisone 35 mg daily over five days and found no difference.3

The choice of agent should be guided by the patients’ comorbidities and concomitant medications, and for patients with contraindications to NSAIDs or colchicine, corticosteroids are particularly useful.

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References

Antidepressants as analgesics

Some important points are worth making in response to the article by Janakiraman, Hamilton and Wan regarding the use of antidepressants as analgesics (AFP March 2016).1 The cited meta-analysis of trials of duloxetine for neuropathy, chronic pain or fibromyalgia raised concerns of trial bias in that of 18 included trials, 17 were performed by the drug manufacturer Eli Lilly, and only two trials were conducted over periods longer than 12 weeks, one of which reported no benefit.2

In addition, fibromyalgia shows high placebo response rates to drug treatment. In a meta-analysis of 18 studies of four drugs, including duloxetine and pregabalin, in 3546 patients, Hauser et al3 showed that the ‘pooled estimate of a 50% pain reduction by placebo was 18.6%’.

I agree with the authors that research limitations need to be carefully considered before making clinical decisions. Unfortunately, the opacity of the trials, data, and even meta-analyses, makes this careful consideration a chore that few clinicians would readily take on.

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References

Reply

We thank Dr Main for his valuable comments regarding the use of antidepressants as analgesics. As Dr Main has correctly mentioned, most of the clinical trials on the effectiveness of duloxetine in chronic pain management were performed by the manufacturers of duloxetine, which is a funding bias. Lunn, Hughes and Wiffen have concluded that there is moderate-quality evidence in managing pain in diabetic peripheral neuropathy with a dose of 60 mg and 120 mg of duloxetine.1
Serotonin (5-hydroxytryptamine) and noradrenaline have been implicated in the mediation of endogenous pain-inhibitory mechanisms via the descending pain-inhibitory pathways in the brain and spinal cord. In view of this, duloxetine was evaluated in neuropathic pain, widespread pain syndrome (WSPS), chronic pain due to osteoarthritis (OA), and chronic lower back pain. There is wide inter-individual variation in response to duloxetine. The beneficial and harmful responses to treatments cannot be predicted for individuals with chronic pain condition.

A recent systematic review published by Finnerup et al showed serotonin–noradrenaline reuptake inhibitors to be moderately effective in managing neuropathic pain with a number needed to treat (NNT) of 6.40 (5.2–8.4). Duloxetine has been recommended as first-line management for neuropathic pain.2 In WSPS, there is lower quality evidence. The effect in WSPS may be achieved through a greater improvement in psychological symptoms than in somatic physical pain.

WSPS is a chronic pain condition in which the main features are widespread musculoskeletal pain and tenderness accompanied by a number of non-specific symptoms that include fatigue, headache, low mood, unrefreshed sleep, abdominal pain and cognitive symptoms.3,4 Pathophysiological hypotheses for WSPS are neuroendocrine dysfunction, neurotransmitter dysfunction and neurosensory dysfunction.5 Complete resolution of symptoms is, unfortunately, almost never achieved, but significant improvement can be obtained through adequate pharmacological and non-pharmacological management. The European League Against Rheumatism (EULAR) published a series of recommendations in 2007.6 They stress the importance of a multidisciplinary approach to treatment of WSPS. Biopsychosocial, cultural and spiritual management with lifestyle changes, such as improving quality and quantity of sleep, avoiding emotional and physical stress, improving posture and body mechanics, eating nutritiously, building a strong support system, developing and maintaining a positive attitude, are recommended. Antidepressants are often used as they decrease pain and improve function. Specific EULAR recommendations on non-pharmacological management include heated pool treatment with or without exercise and, in some cases, individually tailored exercise programs (aerobic exercise and strength training). On the basis of the patient’s specific needs, relaxation, rehabilitation, physiotherapy and psychological support are also beneficial.5

It is also essential to consider the possibilities of hypothyroidism, polymyalgia rheumatica, systemic lupus erythematosus, Sjögren’s syndrome, vitamin D deficiency and autoimmune conditions, as these may complicate the clinical presentation and remain a significant challenge for assessment and management.

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References

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