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Original Article

How is Gout Managed in Primary Care? A Review of Current Practice and Proposed Guidelines

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Abstract: Twelve practices with a total list of 74 111 patients were audited; 429 patients were identified with a diagnosis of gout. A wide variation in various clinical and laboratory assessments was detected. Similar variations were also noted regarding dietary advice and medical treatment. Monitoring of patients was infrequent. As a result of this audit, guidelines are proposed to improve the diagnosis and management of gout in the community.

Keywords: Alcohol; Allopurinol; Gout; Hypertension; Monitoring; Monosodium urate crystals; Obesity; Primary care

Introduction

Gout is perhaps one of the most well described diseases and can be said to be one of a few rheumatic conditions where treatment is most rewarding and almost 'curative'. It is a common condition and recent estimates have indicated that gout may affect more than 6/1000 men and 1/1000 women [1] and that its incidence may be increasing [2,3].

Management of this condition, which is related to hyperuricaemia and deposition of monosodium urate crystals in the joints with acute inflammatory features, occurs principally in general practice but our own clinical impression over the years has raised concerns about aspects of diagnosis, treatment and prophylaxis of gout in the community. In addition, there is still some debate on the best treatment of a subgroup of 'gouty patients' who have intermittent (interval or intercritical) gout and who have no tophi or renal impairment [4,5]. Furthermore, there is no consensus on optimum management, in particular, there are no formal guidelines that could be used by general practitioners in the community [4,5].

Our aim was, therefore, to review the management of gout in primary care with a view to detecting any deficiencies or areas of concern. Accordingly, guidelines would be prepared for general use but which may be of particular benefit to general practitioners. Having circulated 'standards' to the participating practices, our intention in the future would then be to undertake a further audit to assess the impact of our guidelines.

Patients, Methods and Results

Twelve practices with a total list of 74 111 patients participated in the study. We were able to identify 429 patients (5.8 per 1000) with a diagnosis of gout. Computer entries for any patients with a diagnosis of gout or for those who were on known treatment for gout, e.g. allopurinol, were identified and their medical records scrutinised in detail. The male to female ratio was 4:1 and the mean age of the patients was 64.5 years (Fig. 1). A wide variation was observed in the assessment or recording of blood pressure (12–80%), weight (21–100%), history of alcohol intake (29–100%) and investigations such as biochemistry (8–100%) and serum lipids (1–100%) (Figs 2, 3).

Variations were also noted with regard to the advice and treatment offered, e.g. dietary advice ranged from 8

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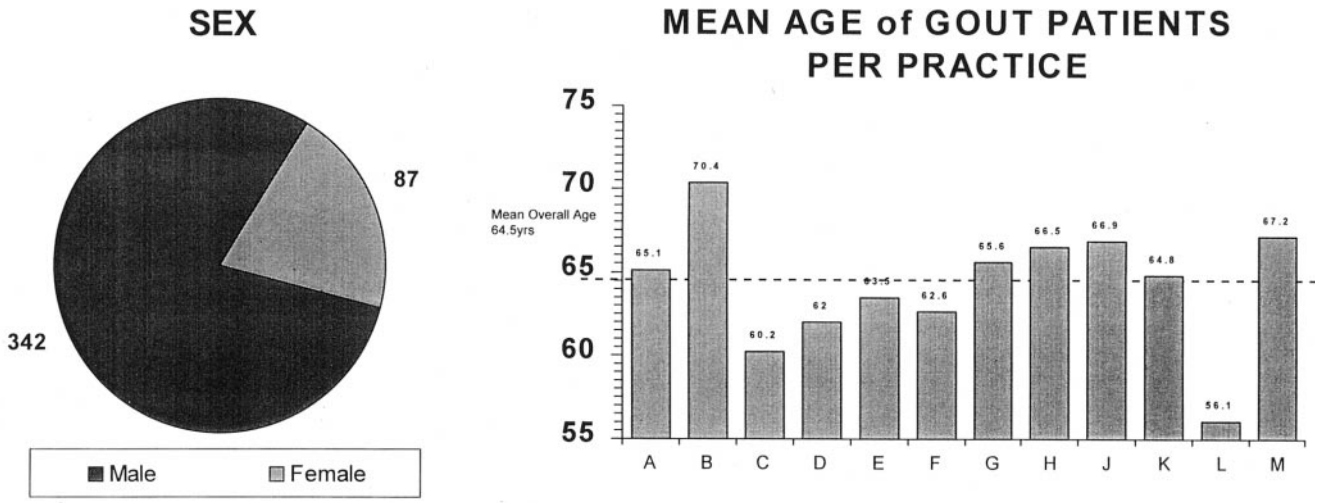

Fig. 1. Audit of mangement of gout: demographic data.

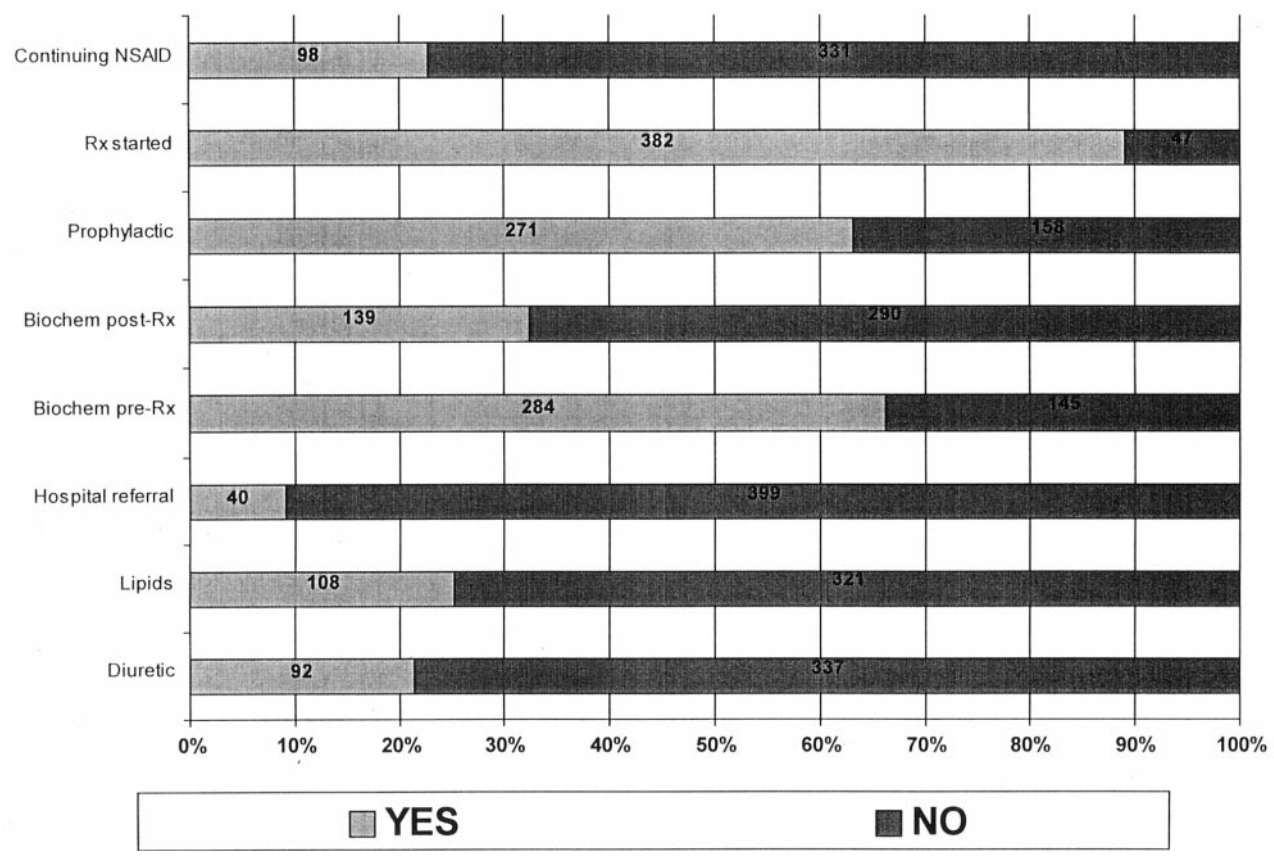

Fig. 2. Audit of management of gout: clinical data. Rx, treatment.

to 75%, counselling on alcohol intake from 5 to 42% and prophylaxis such as allopurinol was variably prescribed (5–97%) (Fig. 2). The dosage of allopurinol also varied enormously with inadequate doses frequently being given, e.g. 81 (19%) patients were on only a 100 mg daily dose (Fig. 4). Some patients were on non-steroidal anti-inflammatory drugs (NSAIDs) long after allopurinol had been started, e.g. more than 2 years in 25 (6%) patients (Fig. 4). In most patients, allopurinol had been started apparently before the acute gout had had a chance to settle with NSAIDs. Most patients (62%) had had no biochemistry investigations performed whilst on drug treatment (Fig. 2). Referral to the Rheumatology Department was infrequent (9%). (Fig. 2).

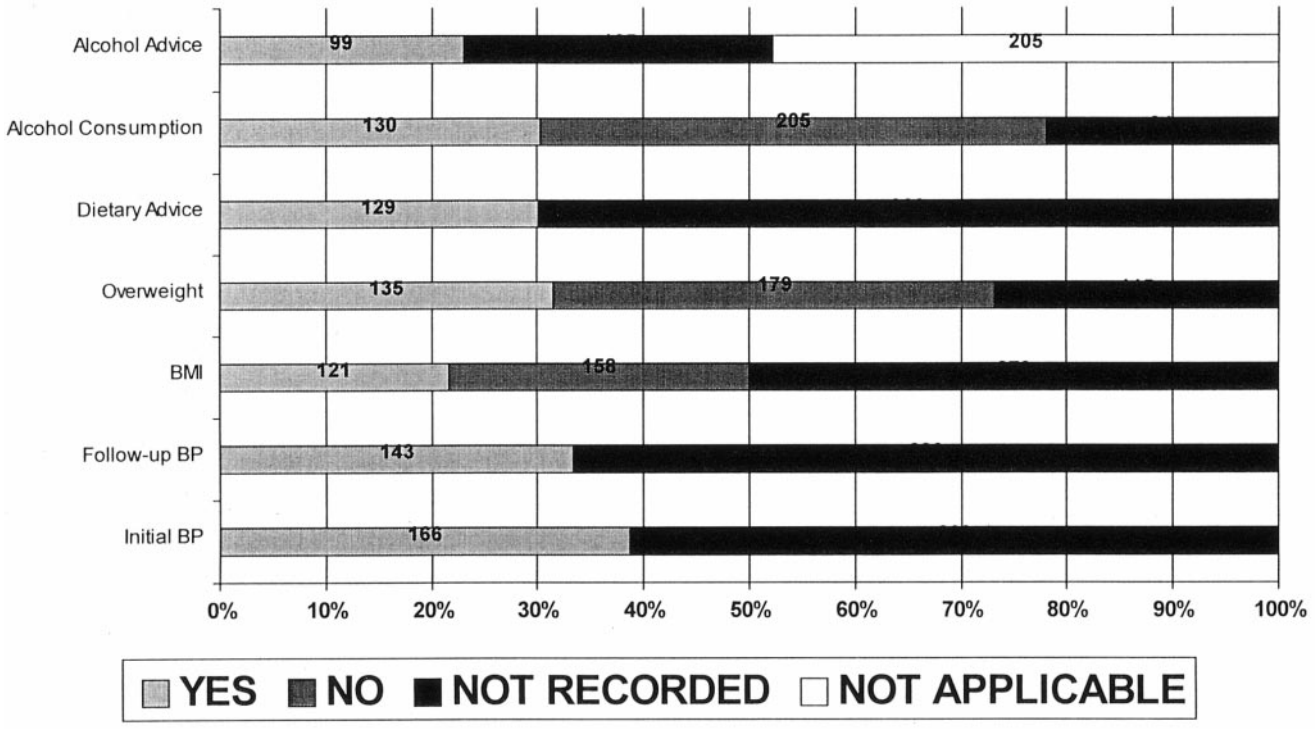

Fig. 3. Audit of management of gout: laboratory and clinical data. BMI, body mass index; BP, blood pressure.

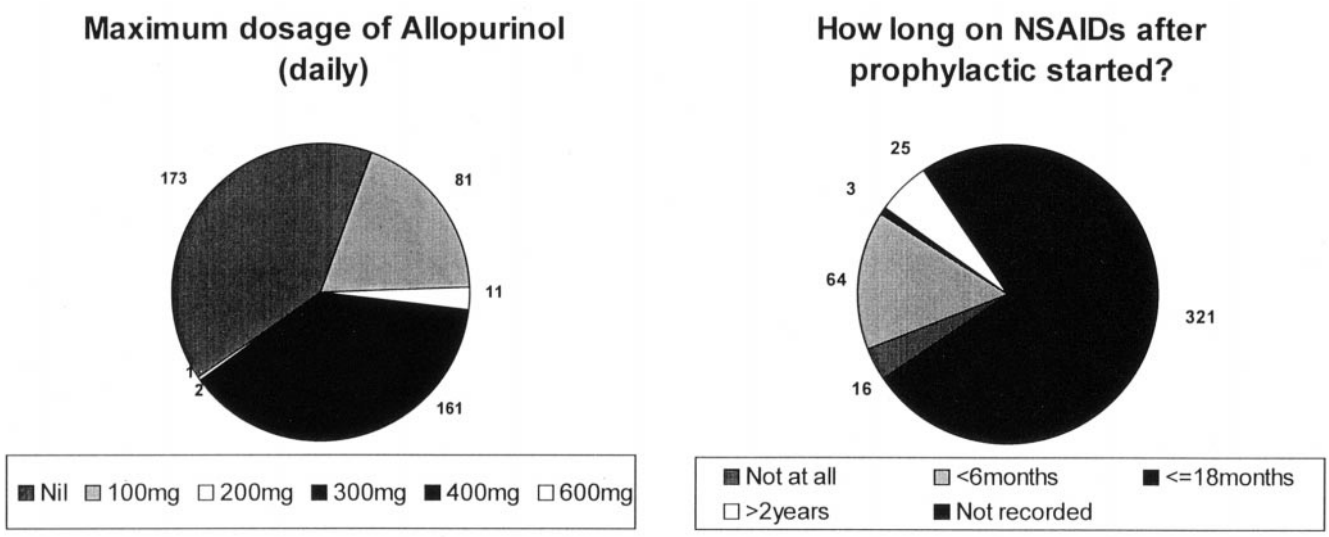

Fig. 4. Audit of management of gout: data on drugs prescribed.

Discussion

We are not aware of any previous large-scale reviews of gout management in the community such as ours. We found enormous variations in the diagnosis and management of gout in this study. The prevalence of gout in different practices varied to such an extent that it raises the possibility of under-recognition in some practices and over-diagnosis of gout in others. It is possible that some patients were on unnecessary treatment whilst others were on inadequate treatment, as shown by our finding that a third of patients were on an insufficient dose of allopurinol, if indeed the underlying diagnosis was truly gout.

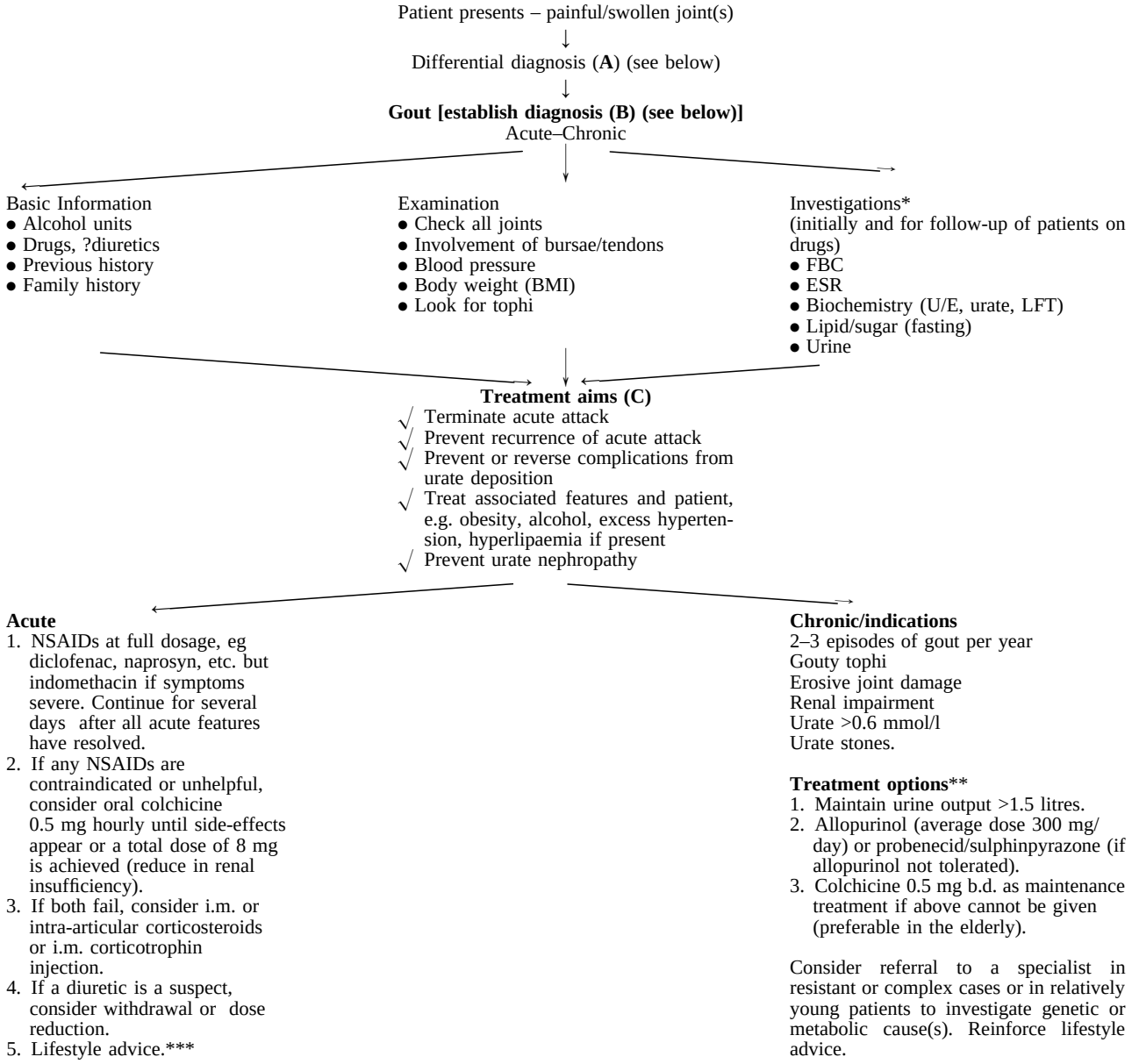

Fig. 5. Proposed guidelines for the assessment and treatment of gout. *Aspirate joint for crystal analysis and culture if unexplained monoarthritis/suspicion of infection (referral to a specialist may be necessary). **Start (only after acute gout has settled) at a low dose and slowly build up, e.g. allopurinol 100 mg o.d., to 300 mg o.d. gradually; occasionally higher doses up to 900 mg o.d. may be required. NSAID or colchicine cover to prevent gouty exacerbations for the first few weeks. Probenecid and sulphinpyrazone are uricosuric agents indicated in patients under the age of 60 years with normal renal function, uric acid excretion <700 mg/24 h and no renal calculi. Allopurinol is a xanthine – oxidase inhibitor and indicated in patients with renal calculi, gouty tophi, impaired renal function, intolerance of uricosuric drugs, urate excretion >700 mgs/24 h and myeloproliferative disease. Reduce doses in renal failure. ***Advice on alcohol, low-purine diet and weight reduction when indicated.

A. Differential diagnosis
1. Pseudo-gout, such as pyrophosphate crystal related arthritis.
2. Palindromic rheumatism.
3. Seronegative inflammatory arthritis.
4. Trauma/haemarthrosis.
5. Infected joint/cellulitis.
6. Type II hyperlipidaemia.
7. Unrelated hyperuricaemia (as in psoriasis, hypertension) when joint pain is not due to gout.

B. Diagnosis
1. History and clinical features.
2. Hyperuricaemia may be present (not in 100% of patients).
3. If any synovial fluid can be aspirated, monosodium urate crystals can be found on polarised microscopy.
4. Typical radiographic changes (punched-out marginal erosions) and secondary osteoarthritis changes in longstanding cases.

Note: Hyperuricaemia on its own is not enough to diagnose gout. Many patients with hyperuricaemia remain asymptomatic and do not have gout.

The management of gout occurs principally in primary care. However, in a small proportion of cases hospital referral for investigations and further management advice may be appropriate [6–9]. A low rate of referral to local rheumatology departments (less than 10% of patients) was noted in this review. Whether or not a higher rate of referral may be appropriate is a question that needs debating.

There are frequent known associations of gout with alcohol excess, hypertension, hyperlipidaemia and obesity [10,11] and we feel that no opportunity should be lost in recognising these factors in patients and offering appropriate advice [12]. Issues associated with gout such as obesity and hypertension are of public health importance, incorporated in NHS targets, and they should not be underestimated when assessing patients with gout. Surveillance of the conditions indicated and appropriate intervention in the early stages would be of obvious benefit, as indicated by other authors [10–12]. Post-treatment surveillance, which appeared to be inadequate in this review, is also an important part of gout management, because a wide range of drugs may be prescribed in this condition [13–15].

It is important that ongoing, continuing care is given to patients with gout because there are areas of concern that may be addressed. Reiteration of initial advice can also be given with regard to hypertension and compliance with treatment, counselling against alcohol excess and appropriate reduction where necessary. Furthermore, monitoring the response to medications and monitoring biochemistry, including serum urate, is of vital importance [9–12]. This needs special care because some patients on long-term treatment with allopurinol may develop adverse reactions, in particular those pertaining to the liver [8,13–15].

It was also our experience that gout had been misdiagnosed in patients with joint pain just because the serum urate level was raised. Conversely, gout had been ruled out because of normal serum urate levels. It is useful to point out that in acute gout, the serum urate level is often normal initially (only to rise later) and low at other times [16]. It is our opinion that this point is not sufficiently emphasised in medical or rheumatology textbooks. If laboratory reports also highlight these facts, general practitioners would be able to increase their diagnostic accuracy and improve patient management.

In view of the deficiencies identified in this audit, we convened a meeting with local general practitioners and asked for their participation in a discussion of the results with a view to preparing a mutually agreed protocol for the diagnosis and management of gout in the community. As a result of these meetings and our findings, we propose the guidelines shown in Fig. 5 for management of gout in the community. These guidelines may also be equally useful in hospital practice.

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