Why is gout so poorly managed?

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Gout has been recognised since ancient times, and we currently have a deep understanding of its pathophysiology. The disease results from a deposit of monosodium urate (MSU) crystals in joint structures and in other, generally periarticular, sites in the form of tophi. High serum uric acid (SUA) is required for the formation of these crystals. The most characteristic features of gout are acute attacks of joint inflammation, which frequently occur at the first metatarsophalangeal joint, although their occurrence in other joints and in bursae is also very common. Oligoarticular, polyarticular, and more protracted and lingering forms of the disease occur, and if untreated or poorly managed, the disease can become very persistent and disabling. Fortunately, we now have highly effective drugs enabling us to deal with gouty joint inflammation and to prevent their recurrence.1 An unequivocal diagnosis can be obtained by identifying MSU crystals in joint fluid obtained either during the attacks2 or at intercritical periods from previously inflamed asymptomatic joints of untreated subjects,3,4 or from a tophus. This procedure requires only skill in performing arthrocentesis and familiarity with the use of the polarised microscope, both a part of the core curriculum in rheumatology. The deposited crystals responsible for the disease dissolve when SUA levels are brought back to normal,3,5 and the time for total dissolution relates to the duration of the disease; after 1 year of adequate hypouricaemic treatment, crystals have usually disappeared from the joints if the disease duration has not been too long.6 In the absence of MSU crystals, attacks of gout are impossible, and the disease can be considered cured. Patients who have formed MSU crystals on one occasion appear to easily form them anew if SUA levels are allowed to rise again, prompting the return of the disease.7,8 To avoid this, lifelong hypouricaemic drug treatment is necessary after the crystals have dissolved, although for some patients dietary and lifestyle modifications may suffice. Finally, in an important proportion of patients with gout, hyperuricaemia is a part of metabolic syndrome and the presence of gout should make clinicians aware of associated morbidities, which are reversible, at least to some extent, by modifying dietary and lifestyle habits. The recent publication of guidelines on gout diagnosis and management9 by the European League Against Rheumatism (EULAR), the quality of care indicators from the US,10 and outcome measures for clinical trials11 outline the current interest in gout.

Despite such a reassuring background, the standard of gout management in practice continues to lag behind the research. A precise diagnosis is the first step for the correct management of any disease. In gout, identification of MSU crystals allows this, as has been stressed in the recently published EULAR guidelines9 and quality of care indicators.11 In addition, the technique is consistent when performed by trained personnel.12 However, in practice, gout is too frequently diagnosed on clinical grounds and a high SUA level, an approach that appears supported by the frequently referred ACR diagnostic criteria, published in 1977 as Preliminary criteria for the classification of the acute arthritis of primary gout,13 but which have not been revised nor received further validation. Reports showing inadequacies of serum urate-lowering treatment14–16 and of gout-related inflammation therapy17 show that in treatment the gap between clinical practice and potential treatment possibilities is large. In this issue of the Annals of the Rheumatic Diseases,18 we are reminded that according to the recent EULAR recommendations for the management of gout,19 primary care gout management in the UK is poor.

If the knowledge is widely available, why should gout management be inappropriate? Gout appears to be considered a minor disease by many rheumatologists, and the care of patients with gout often rests on the shoulders of the general practitioner (GP). We searched in the PubMed database for all papers published in the past 10 years on gout, using the MeSH terms “patient care management”, “quality of care” and “gout”, and found 11 papers. Five of these directly investigated the way gout is managed by different medical specialties: two papers audited gout management in primary care practices,20–22 one recent paper conducted a survey of US rheumatologists24 and the remaining two explored treatment trends by both primary care doctors and rheumatologists in China and Mexico.25–26 Of interest, in the four papers in which the management of gout by GPs was evaluated, GPs were fully responsible for the management of the disease, including its diagnosis. In contrast, rheumatologists play a leading role in the management of other inflammatory joint conditions such as rheumatoid arthritis (RA). Using a similar search approach as above, we found 61 papers published in the past 10 years relating to the way in which RA is managed by different specialties. Six of those papers centred around management of RA in primary care,27–32 48 papers (79%) reported management by rheumatologists and the remaining 7 compared or included both types of doctor.27–32 Many of these papers stress the need for early and easy access to a rheumatologist when RA is suspected; this contrasts sharply with data from the UK, which found that most patients with gout never see a rheumatologist.21

Any one of us with sufficient experience has seen (1) patients with less typical clinical presentations posing difficulties for a clinical approach to the diagnosis, (2) patients with severe gout seriously affecting the patient’s quality of life, or even its duration, (3) patients undiagnosed or misdiagnosed despite repeated evaluation, and thus (4) have an appreciation how notoriously difficult gout can be to manage. In addition, in the UK, rheumatologists act essentially as the consultants to whom the less typical and difficult cases ought to be referred, but a large proportion of rheumatologists are unfamiliar with synovial fluid analysis for crystal identification. Thus, when they are consulted about a patient with possible gout, they often have to rely on clinical diagnosis, and their accuracy may be no better than that of the GP who made the referral (who is likely to know that urate crystal identification provides an unequivocal, evidence-based diagnosis, and perhaps may think that the rheumatologist’s clinical approach to gout is an indication of the way all diseases are approached in his specialty). This relaxed attitude of rheumatologists towards gout may be perceived by GPs and other doctors, stimulating them to take over the care of patients with gout.

Those writing in scientific journals have to support their assertions with evidence, and the available evidence essentially indicates that (1) most patients with gout are cared for at the primary care level, (2) gout is often diagnosed on less accurate clinical
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doi: 10.1136/ard.2007.078469

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