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Case report

Case study: A case of debilitating gout in the 1st metatarsophalangeal joint

Natalie Tanner*, Ross Diaper, Mathew King, Stuart A. Metcalfe

Department of Podiatric Surgery, Turner Centre, St James Hospital, Locksway Road, Portsmouth PO4 8LD, UK

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ABSTRACT

Gout is a painful arthritic condition that affects many people worldwide. The disease has been associated with hyperuricaemia and life style risk factors such as obesity, alcohol intake, meat and seafood consumption.

We present a case of a 67-year-old male with a history of gout, who attended the clinic with a painful 1st metatarsophalangeal joint, which had progressively worsened in pain, mobility and deformity in the last 20 years. Although lifestyle changes had been advised by the GP some years earlier such as a low purine based diet, management had only consisted of NSAID's, which had not significantly improved symptoms.

Surgical excision of chalky white material from around the 1st metatarsophalangeal joint rendered the patient symptom free with increased mobility after 6 weeks. Histopathology confirmed the excised tissue as gouty tophus. Following this, the patient was placed on allopurinol, a xanthine oxidase inhibitor to prevent recurrent attacks.

This case study highlights the importance of early recognition and prophylactic management in gout sufferers. In joints where the disease process is well-established surgical excision of the gouty tophus may help mitigate further disease progression, and restore quality of life to individuals.

1. Introduction

Gout is an arthritic disease whereby monosodium urate crystals form, primarily in joints and other tissues. An acute flare can be excruciatingly painful and is associated with impaired quality of life [1]. The incidence is on the increase, both in primary care and hospital practice, triggering a recent rise in media coverage [2]. Gout has been strongly linked to elevated blood urate levels (hyperuricaemia) along with life style related factors such as obesity, alcohol intake, meat and seafood consumption [3,4].

Poor or late diagnosis with suboptimal management has been reported to have possibly contributed to the increase in disease burden over the last few decades [5]. This paper showcases the potential for the severe destruction of joints in this patient group.

Historically, gout was one of the earliest clinical afflictions to be recognised, with evidence dating back to 400BC from Hippocratic writings [6]. Once described as the "king of diseases and the disease of kings" [7] due to purine rich food being restricted to the wealthy classes, in recent decades this has changed to a more widespread phenomenon.

Evidence from epidemiological surveys from the United Kingdom, China, United States and other countries suggest that gout is on the increase [8]. In the UK in 1993, surveys reported 0.95% of gout in a survey of 1000 people. Both the UK General Practice Research database in 1999 [9] and the IMS Disease Analyzer (2000–2005) [10] found the prevalence of gout in the UK to be 1.4%. A report published in 2011 also found the incidence significantly increased with age [11].

An American study in 2004 which looked at the increasing prevalence of gout over a 10-year period showed a male to female ratio of 4:1 in under 65s and 3:1 for over 65s. This highlights both the rarity of gout in younger women and the age associated increase of gout in post-menopausal women [12]. This corresponded to the UK study of 2011 mentioned above where the incidence rate increased from 0.4 per 1000 person-years in women of 40–49 years to 3.6 in women of 70–79 years. However male prevalence was higher in those under 60 [11]. This increase in gender difference over 65 may be due to hormonal status. Oestrogens increase the excretion of uric acid in the urine which could be protective against gout in premenopausal women [13,14]. This is also recognised in women on hormone replacement therapy; Cea Soriano et al. in 2011

* Corresponding author. Tel.: +44 7774 181009; fax: +44 7774 181009.
E-mail address: natalie.tanner@solent.nhs.uk (N. Tanner).

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observed a 14% decrease in the risk of developing gout in women on HRT. This effect disappeared once therapy had stopped [11].

2. Pathophysiology

Uric acid is produced as the final metabolite of purine metabolism. When ionised it is known as urate. At a serum pH of 7.4, 98% of uric acid is in the form of urate, this binds to sodium in the extracellular compartment forming monosodium urate.

There is a direct link between hyperuricaemia and gout. Interestingly, humans are the only mammals in whom gout is known to develop unprompted. This may be because humans do not process the enzyme uricase, present in animals such as fish, amphibians and non-primate mammals. This enzyme oxidatively degrades uric acid to allantoin, a more soluble compound. The lack of uricase in humans therefore increases their susceptibility to gout as the less soluble uric acid is more likely to precipitate out into joint spaces [4].

Gout is classified into primary and secondary forms. Both present in the same way and differ only in the initial cause of the hyperuricaemia. In primary gout, hyperuricaemia results from either uric acid overproduction or decreased excretion. In contrast, secondary gout develops due to extrinsic factors such as drugs (thiazide diuretics, low-dose aspirin), alcoholism, chronic renal insufficiency and anorexia. Secondary gout is usually encountered in patients younger than 20 years of age [15] (Fig. 1).

Purines accumulate in various ways: they are absorbed from the gut in proportion to dietary intake; synthesised in the body or reclaimed from endogenous DNA or RNA. They are then oxidised by xanthine oxidase to form the end product uric acid [16]. The kidney excretes the majority of uric acid, although some is reabsorbed in the proximal tubule.

Formation of monosodium urate crystals in tissues depends on its local concentration. As it has a low solubility limit of 380 μmol/L [17] the urate is more likely to precipitate out as gout. The solubility depends on the articular hydration state, temperature, pH and the presence of extracellular matrix proteins such as proteoglycans, collagens and chondroitin sulphate [18]. Chronic depositions of monosodium urate crystal form chalky deposits known as tophi [19].

Acute gouty arthritis usually presents in the lower limb and affects one joint, most commonly the 1st metatarsophalangeal joint (MTPJ) in 85–90% of cases. Other frequent locations include the mid tarsi, ankles, knees and elbows. The predilection of gout for the 1st MTPJ (a joint which is peripheral and therefore has a lower temperature) and osteoarthritic joints (which have decreased collagen and proteoglycan content) is widely documented [20,21].

A systematic review of population based epidemiological studies found that the estimated prevalence of 1st MTPJ OA in those above middle age may be up to 39% [22]. This may indicate why there are a high number of gout sufferers in this population.

Chronic gout often causes boney erosions, which lead to debilitating joint damage. Characteristic features of joint damage boney erosions are; new bone formation, tophi within tendons and focal cartilage loss with eventual joint destruction. Chhana and Dalbeth recently looked the pathophysiology of joint damage in advanced gout. They reported that the increase in osteoclasts and their activity with the reduced osteoblast viability, function and differentiation, contribute to bone erosions seen in gout. Damage to the articular cartilage is caused by reduced viability of chondrocytes, reduced matrix production in combination with the increased catabolic enzyme activity and inflammation [23].

A study in New Zealand analysed paired plain radiographs and CT scans of 798 individuals with gout in order to establish a relationship between the size (mm) of radiographic erosions and the presence of intraosseus tophus. They found a significant correlation between boney erosion size and tophus diameter. They suggested that the tophus infiltration into bone is the dominant mechanism for the development of bone erosion and joint damage in gout.

A number of comorbidities have been strongly associated with gout. In practice patients have a high prevalence of cardiovascular disease, hypertension, renal impairment and metabolic syndrome [24]. A recently published US survey looked to estimate the latest prevalence of major comorbidities associated with hyperuricemia and gout. They looked at data from 5707 participants aged 20 years and older and found that among the individuals with gout 74% had hypertension, 71% chronic kidney disease (stage 2), 53% were obese, 26% had diabetes, 24% has nephrolithiasis, 14% had myocardial infarction 11% had heart failure and 10% had suffered a stroke. These proportions were substantially higher than the individuals not suffering from gout [25]. These results were echoed in their stating that they found that patients with prior histories of ischaemic heart disease, heart failure, obesity, chronic renal impairment were associated with increased risk of gout [11].

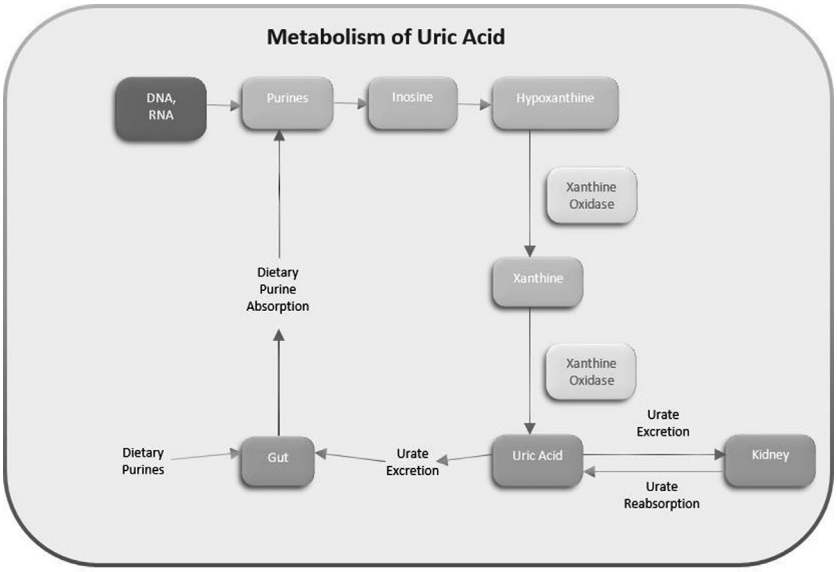

Fig. 1. Diagram depicting the metabolism of uric acid.