



## REVIEW ON DIAGNOSIS & MANAGEMENT OF GOUT IN PRIMARY HEALTH CARE

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### ABSTRACT

With a countrywide incidence of 2.5% in the adult population, gout is the most prevalent inflammatory joint disease affecting men over 40 in the UK. Prevalences are similar in other industrialized nations, including the US and New Zealand. Monosodium Urate (MSU) crystals occur and are deposited in joints, most frequently the first metatarsophalangeal joint (MTP), as a result of the hyperuricemia that causes gout. Most gout patients in the UK are treated exclusively in primary care. To ensure that care is provided in accordance with need, it is critical to assess the socioeconomic status (SES) of a community afflicted by a condition. Being from a lower SES group is linked to more GP visits and worse health, according to numerous studies on social disadvantage and health consequences. Some of these studies have been conducted at the neighborhood and individual levels to determine whether any associations are caused by characteristics specific to the community or the patients.

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### Introduction

With a countrywide incidence of 2.5% in the adult population, gout is the most prevalent inflammatory joint disease affecting men over 40 in the UK [1]. Prevalences are similar in other industrialized nations, including the US and New Zealand [2, 3]. Monosodium Urate (MSU) crystals occur and are deposited in joints, most frequently the first metatarsophalangeal joint (MTP), as a result of the hyperuricemia that causes gout [4]. Most gout patients in the UK are treated exclusively in primary care. To ensure that care is provided in accordance with need, it is critical to assess the socioeconomic status (SES) of a community afflicted by a condition. Being from a lower SES group is linked to more GP visits and worse health, according to numerous studies on social disadvantage and health consequences [5, 6]. Some of these studies have been conducted at the neighborhood and individual levels to determine whether any associations are caused by characteristics specific to the community or the patients.

Little research has examined the link between SES and gout, producing conflicting findings. In a recent study conducted in

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the UK, it was discovered that those with gout who were 50 years of age or older were more likely than those without gout to see their financial income as inadequate. However, there was no correlation between having gout and occupational class, amount of education, or level of poverty in your neighborhood [7]. In Australia, no correlation with area-level disadvantage was discovered [8, 9]. Gout and SES were linked in research from Western Germany but not in a study of men [10].

More socioeconomically impoverished communities have the highest prevalence of gout, according to studies from England and New Zealand [4, 11]. Most of these earlier research contrasted SES between gout sufferers and non-gout sufferers. There are hardly any investigations on the connection between SES and the severity of gout. One study from Mexico found no association between tophi presence and socioeconomic class or educational attainment [12]. People frequently miss work due to acute gout severity, resulting in temporary sick leave [13]. People with gout have been demonstrated to be highly concerned about how their condition may affect their productivity, employability, and capacity to work [14]. Employees with gout take roughly five times as many days off from work each year due to illness as those without gout [15]. Employees with at least three gout attacks annually miss more days from work than those who experience fewer attacks [16]. In a study of individuals with severe chronic gout that was resistant to conventional therapy (mean number of attacks 8.8 per year), 78% of those under 65 reported missing work for at least one day owing to gout during a year, with the mean yearly number of workdays lost being 25 days [17].

It has been demonstrated that persons with insufficiently controlled gout, even receiving urate-lowering medication, have lower work productivity than those with satisfactory management [18]. Contrarily, a tiny study revealed no connection between the frequency of assaults or tophi and productivity at work [19].

### *Epidemiology*

Globally, gout is the most prevalent kind of inflammatory arthritis. Gout is more prevalent in aging populations since the danger of developing it rises with advancing years. Gout is caused by a persistent increase in serum urate levels (hyperuricemia), which deposits monosodium urate crystals in joints, tendons, and other tissues. Gout flares are periodic episodes of severe acute inflammation. Despite being one of the few rheumatic diseases that can be "cured" with pharmacological urate-lowering treatments (ULTs), gout management remains insufficient in many parts of the world due to low ULT adoption and patient adherence. Gout frequently coexists with several comorbid illnesses, such as obesity, chronic renal disease, and cardiovascular disease [20].

Understanding changes in gout prevalence is crucial for facilitating adequate healthcare resource planning. Gout is the most prevalent inflammatory arthritis worldwide, not to mention that gout may be "cured" using readily available and affordable therapies. Due to the absence of data for many nations and the widely varying prevalence estimates across various geographic regions and demographics obtained using different illness classifications, it is challenging to determine the global prevalence of gout [20].

### *Diagnosis*

Gout diagnostic criteria include two of the following:

- A clear history of at least two episodes of painful joint swelling that resolved completely within two weeks;
- A clear history or observation of podagra, the presence of tophus;
- Rapid response to colchicine within 48 hours of starting treatment [21].

Definitive diagnosis: the presence of monosodium urate crystals in the serum, in the joint fluid, or in the tissues.

The primary pathophysiologic events defining the clinical state of gout are the deposition of urate crystals in tissue and the ensuing inflammatory and potentially harmful effects. A gout flare is caused by monosodium urate (MSU) crystals, which can be seen when polarized light microscopy is used to identify these crystals in synovial fluid taken from joints or bursas [21]. While the joint spaces are typically intact and periarticular osteopenia is absent, unlike the radiographic alterations typical of rheumatoid arthritis, plain radiography may not detect early disease that does not show any abnormality. Bone erosions indicative of advanced gout frequently have an overhanging edge and sclerotic rim. In the event of a difficult diagnostic situation and for early detection, ultrasonography can be very helpful. Hyperechoic linear density (double contour sign) on the surface of hyaline cartilage or tophaceous deposits in joints or tendons are described as hyperechoic and encircled by a small anechoic rim, are crucial diagnostic findings [22].

Without joint fluid analysis, the primary care environment uses clinical diagnostic criteria to predict the likelihood of gout. The first metatarsal phalangeal joint is involved in the model along with seven other factors, including male gender (2 points), previous patient-reported arthritis (2 points), onset within a day (0.5 points), joint redness (1 point), hypertension or at least one cardiovascular disease (1.5 points), and serum urate level  $>5.88$  mg/dL or 350 micromol/L (3.5 points) [22]. Patients can be classified as having a low (4 points), intermediate ( $>4$  or 8 points), or greater (8 points) chance of gout based on their overall score. Patients in the intermediate category gain the most from additional assessment by synovial fluid analysis [22]. This clinical diagnostic criterion was tested on 390 Dutch primary care patients with acute monoarthritis, outperforming a doctor's diagnosis and providing a positive result in the derivation study [23]. Primary care physicians should be aware that acute calcium pyrophosphate crystal arthritis or septic arthritis may occasionally occur with gout flares (pseudogout). When a joint is aspirated, an orthopedic surgeon or rheumatologist will help determine the diagnosis by analyzing the synovial fluid [23]. In our routine clinical practice, we currently observe the three traditional stages of gout: acute gout flare, inter-critical gout,

chronic gouty arthritis, and tophaceous gout).

#### *Risk Factors*

Factors that raise hyperuricemia are risk factors for gout. A baseline gender-associated 1 mg/dL greater uric acid in men increases the risk for gout compared to premenopausal women. The difference between the premenopausal and postmenopausal states is gone, and estrogen effects on uric acid clearance are most likely the cause [24]. The greater risk of hyperuricemia observed in the elderly is expected to be caused by comorbid renal illness in advanced age [24]. African Americans have higher serum uric acid levels than Caucasians do at baseline, which is one of the ethnic factors that affect risk [25]. Hyperuricemia and gout are linked to rare X-linked inborn metabolic abnormalities, such as Lesch-Nyhan syndrome (hypoxanthine-guanine phosphoribosyl transferase enzyme deficiency). Hyperuricemia and gout are also linked to metabolic comorbidities such as obesity, hypertension, hyperlipidemia, and metabolic syndrome [26]. Although it is recognized that endocrine disorders, such as hyperparathyroidism and hypoparathyroidism, and hypothyroidism, can affect kidney function, it is less apparent how these conditions affect blood urate levels and the risk of developing clinical gout [27].

#### *Treatment*

Treatment for acute gout should begin within 24 hours after the beginning of symptoms to obtain rapid and complete symptom relief [28]. Colchicine, NSAIDs, intravenous corticosteroids, and oral corticosteroids all work well to treat acute gout flares-ups [28]. The initial line of treatment is NSAIDs. Although Indocin (indomethacin) has traditionally been the drug of choice, there is no proof that it is more efficacious than other NSAIDs. It appears that intramuscular ketorolac works similarly [29]. After symptoms have subsided, any oral NSAID may be used at the maximum dosage for one to two days. For people who cannot tolerate NSAIDs or colchicine, corticosteroids are a suitable substitute [30]. Corticosteroids can be administered to patients with diabetes mellitus for brief periods with the proper monitoring for hyperglycemia. Due to their lower risk of side effects, intra-articular corticosteroid injections may be preferable to systemic corticosteroids when gout is restricted to a single joint [31].

Following the cessation of corticosteroid therapy for acute gout, rebound flares are frequent. After symptoms have subsided, preventative treatment and the start of a tapering course of corticosteroids over 10 to 14 days are advised to lower the likelihood of a return flare. Another alternative for acute gout treatment is colchicine. The US Food and Drug Administration (FDA) did not formally assess generic colchicine, which has been used for decades, for this indication until 2009, when branded colchicine (Colcrys) was approved. However, Colcrys is pricey, and there is no longer a generic version of colchicine.

Furthermore, when administered more than 72 to 96 hours after the onset of symptoms, colchicine may be less helpful at treating acute flares because it lacks analgesic qualities. Typical side effects include vomiting, diarrhea, and nausea [29]. Patients with renal or hepatic impairment should use colchicine with caution.

#### **Conclusion**

The condition of gout is easily treatable and can even be cured. The goal of care during an acute flare aims to relieve pain while preventing impairment and initiating treatment early. Although ULTs are widely accessible and efficient, allopurinol is still the first-line therapy [32]. Primary care practitioners should know the rationale for starting ULT, setting serum uric acid targets with patients, treating potential adverse effects of ULTs, and educating patients about dietary and lifestyle changes. Testing for hypertension, diabetes, hypercholesterolemia, and keeping an eye on weight is essential because gout is commonly a part of the metabolic phenomenon. Treatment should be used when necessary.

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#### **References**

1. Kuo CF, Grainge MJ, Mallen C, Zhang W, Doherty M. Rising burden of gout in the UK but continuing suboptimal management: a nationwide population study. *Ann Rheum Dis.* 2015;74(4):661-7.
2. Lawrence RC, Felson DT, Helmick CG, Arnold LM, Choi H, Deyo RA, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. *Arthritis Rheum.* 2008;58(1):26-35.
3. Winnard D, Wright C, Taylor WJ, Jackson G, Te Karu L, Gow PJ, et al. National prevalence of gout derived from administrative health data in Aotearoa New Zealand. *Rheumatology (Oxford).* 2012;51(5):901-9.
4. Roddy E, Muller S, Rome K, Chandratre P, Hider SL, Richardson J, et al. Foot problems in people with gout in primary care: baseline findings from a prospective cohort study. *J Foot Ankle Res.* 2015;8:31.

5. Baker D, Mead N, Campbell S. Inequalities in morbidity and consulting behaviour for socially vulnerable groups. *Br J Gen Pract.* 2002;52(475):124-30.
6. Jaffe DH, Eisenbach Z, Neumark YD, Manor O. Individual, household and neighborhood socioeconomic status and mortality: a study of absolute and relative deprivation. *Soc Sci Med.* 2005;60(5):989-97.
7. Hayward RA, Rathod T, Roddy E, Muller S, Hider SL, Mallen CD. The association of gout with socioeconomic status in primary care: a cross-sectional observational study. *Rheumatology (Oxford).* 2013;52(11):2004-8.
8. Robinson PC, Taylor WJ, Dalbeth N. An Observational Study of Gout Prevalence and Quality of Care in a National Australian General Practice Population. *J Rheumatol.* 2015;42(9):1702-7.
9. Ting K, Gill TK, Keen H, Tucker GR, Hill CL. Prevalence and associations of gout and hyperuricaemia: results from an Australian population-based study. *Intern Med J.* 2016;46(5):566-73.
10. Helmert U, Shea S. Social inequalities and health status in western Germany. *Public Health.* 1994;108(5):341-56.
11. Gardner MJ, Power C, Barker DJ, Padday R. The prevalence of gout in three English towns. *Int J Epidemiol.* 1982;11(1):71-5.
12. Vázquez-Mellado J, Cruz J, Guzmán S, Casasola-Vargas J, Lino L, Burgos-Vargas R. Severe tophaceous gout. Characterization of low socioeconomic level patients from México. *Clin Exp Rheumatol.* 2006;24(3):233-8.
13. Tausche AK, Panzner I, Aust D, Wunderlich C. Disabling gout. *Lancet.* 2010;376(9746):1093.
14. Singh JA. The impact of gout on patient's lives: a study of African-American and Caucasian men and women with gout. *Arthritis Res Ther.* 2014;16(3):R132.
15. Kleinman NL, Brook RA, Patel PA, Melkonian AK, Brizee TJ, Smeeding JE, et al. The impact of gout on work absence and productivity. *Value Health.* 2007;10(4):231-7.
16. Lynch W, Chan W, Kleinman N, Andrews LM, Yadao AM. Economic burden of gouty arthritis attacks for employees with frequent and infrequent attacks. *Popul Health Manag.* 2013;16(2):138-45.
17. Edwards NL, Sundy JS, Forsythe A, Blume S, Pan F, Becker MA. Work productivity loss due to flares in patients with chronic gout refractory to conventional therapy. *J Med Econ.* 2011;14(1):10-5.
18. Wood R, Fermer S, Ramachandran S, Baumgartner S, Morlock R. Patients with Gout Treated with Conventional Urate-lowering Therapy: Association with Disease Control, Health-related Quality of Life, and Work Productivity. *J Rheumatol.* 2016;43(10):1897-903.
19. Khanna PP, Nuki G, Bardin T, Tausche AK, Forsythe A, Goren A, et al. Tophi and frequent gout flares are associated with impairments to quality of life, productivity, and increased healthcare resource use: Results from a cross-sectional survey. *Health Qual Life Outcomes.* 2012;10:117.
20. FitzGerald JD, Dalbeth N, Mikuls T, Brignardello-Petersen R, Guyatt G, Abeles AM, et al. American College of Rheumatology guideline for the management of gout. *Arthritis Care Res.* 2020;72(6):744-60. doi:10.1002/art.41247
21. Steinberg J. Clinical Diagnosis of Gout Without Joint Aspiration. *Am Fam Physician.* 2016;94(6):505-6.
22. Kienhorst LB, Janssens HJ, Franssen J, Janssen M. The validation of a diagnostic rule for gout without joint fluid analysis: a prospective study. *Rheumatology (Oxford).* 2015;54(4):609-14.
23. Hak AE, Choi HK. Menopause, postmenopausal hormone use and serum uric acid levels in US women--the Third National Health and Nutrition Examination Survey. *Arthritis Res Ther.* 2008;10(5):R116.
24. Fang J, Alderman MH. Serum uric acid and cardiovascular mortality the NHANES I epidemiologic follow-up study, 1971-1992. *National Health and Nutrition Examination Survey. JAMA.* 2000;283(18):2404-10.
25. Chen JH, Yeh WT, Chuang SY, Wu YY, Pan WH. Gender-specific risk factors for incident gout: a prospective cohort study. *Clin Rheumatol.* 2012;31(2):239-45.
26. Hui JY, Choi JW, Mount DB, Zhu Y, Zhang Y, Choi HK. The independent association between parathyroid hormone levels and hyperuricemia: a national population study. *Arthritis Res Ther.* 2012;14(2):R56.
27. Janssens HJ, Janssen M, van de Lisdonk EH, van Riel PL, van Weel C. Use of oral prednisolone or naproxen for the treatment of gout arthritis: a double-blind, randomised equivalence trial. *Lancet.* 2008;371(9627):1854-60.
28. Khanna D, Khanna PP, Fitzgerald JD, Singh MK, Bae S, Neogi T, et al. 2012 American College of Rheumatology guidelines for management of gout. Part 2: therapy and antiinflammatory prophylaxis of acute gouty arthritis. *Arthritis Care Res (Hoboken).* 2012;64(10):1447-61.
29. Burns CM, Wortmann RL. Latest evidence on gout management: what the clinician needs to know. *Ther Adv Chronic Dis.* 2012;3(6):271-86.
30. Zhang W, Doherty M, Bardin T, Pascual E, Barskova V, Conaghan P, et al. EULAR evidence based recommendations for gout. Part II: Management. Report of a task force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCIIT). *Ann Rheum Dis.* 2006;65(10):1312-24.
31. Richette P, Doherty M, Pascual E, Barskova V, Becce F, Castañeda-Sanabria J, et al. 2016 updated EULAR evidence-based recommendations for the management of gout. *Ann Rheum Dis.* 2017;76(1):29-42. doi:10.1136/annrheumdis-2016-209707
32. Khanna D, Fitzgerald JD, Khanna PP, Bae S, Singh MK, Neogi T, et al. 2012 American College of Rheumatology guidelines for management of gout. Part 1: systematic nonpharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care Res (Hoboken).* 2012;64(10):1431-46.