DOI: https://doi.org/10.53350/pjmhs02024181070

ORIGINAL ARTICLE

Dietary Risk Factors Associated with Gout Flares Among Patients with Gout

NAJIB ULLAH[1], AMJAD SHAHZAD[2], KHUSHAL NADIR HADI[3], AMJAD ALI[4], MARIA ZAFAR[5], IMRAN KHAN[6], ILHAM ULLAH[7], MUHAMMAD ADIL[8], SHERAZ KHAN[9], ISRAR AHMAD[10]

[1]*Specialist Registrar Medicine Department, MTI DHQ, Bannu*
[2]*Assistant Professor Medicine Department, Khyber Teaching Hospital, Peshawar*
[3]*FCPS PGR, Hayatabad Medical Complex, Peshwar*
[4]*Senior Registerar Medical A Ward Saidu Group of Teaching Hospital, Swat*
[5]*Medical specialist PIMS, Islamabad*
[6]*PGR Medicine, Bacha Khan Medical Complex, Swabi*
[7]*PGR Medicine, Khyber Teaching Hospital, Peshawar*
[8]*PGR Medicine, Khyber Teaching Hospital, Peshawar*
[9]*MBBS, RMP, Senior Lecturer, Asian International University*
[10]*PGR Medicine, Khyber Teaching Hospital, Peshawar*
Crosspondence to: Dr Israr Ahmad, Email: jayahmad570@gmail.com

ABSTRACT

Background: Gout is a common inflammatory arthritis caused by deposition of monosodium urate crystals in joints. Dietary habits may influence serum uric acid levels and can act as modifiable triggers for recurrent gout flares.

Objective: To identify dietary risk factors associated with gout flares among patients with gout and assess their relationship with the occurrence of acute attacks.

Material and Methods: This analytical cross-sectional study was conducted in the Department of Medicine, Khyber Teaching Hospital Peshawar from January 2023 to June 2023. Demographic characteristics, body mass index, duration of gout, serum uric acid level, smoking status, comorbidities, medication use, and dietary habits were recorded using a structured questionnaire. Dietary exposures included red meat, organ meat, seafood, sugar-sweetened beverages, high-fat meals, fluid intake, fasting, and low-fat dairy consumption.

Results: The mean age of the 165 participants was 51.6 ± 11.2 years, and 133 (80.6%) were male. Overall, 112 (67.9%) patients experienced at least one gout flare during the preceding 12 months. Patients with flares had significantly higher serum uric acid levels than those without flares (8.7 ± 1.6 vs. 7.6 ± 1.5 mg/dL, p<0.001) and a longer duration of gout (6.7 ± 4.2 vs. 4.9 ± 3.4 years, p=0.007). Frequent red meat intake (adjusted OR=2.41, 95% CI: 1.11–5.22), organ meat consumption (adjusted OR=2.58, 95% CI: 1.02–6.51), sugar-sweetened beverage intake (adjusted OR=2.36, 95% CI: 1.08–5.18), and low fluid intake or dehydration (adjusted OR=2.89, 95% CI: 1.31–6.38) were independently associated with gout flares.

Conclusion: Frequent consumption of red meat, organ meat, sugar-sweetened beverages, and inadequate fluid intake were associated with increased odds of gout flares, while regular low-fat dairy intake showed a possible protective association.

Keywords: gout, gout flare, diet, red meat, organ meat, sugar-sweetened beverages, hydration, uric acid.

INTRODUCTION

Persistent hyperuricemia leads to deposition of monosodium urate crystals in the joints and surrounding tissue, causing gout, a common inflammatory arthritis. Symptoms include a sudden onset of intense pain, swelling, redness, and tenderness, usually affecting the first metatarsophalangeal joint[1]. If the attacks of gout recur, then it can progress to chronic gouty arthritis, tophus formation, damage to the joints, loss of mobility and diminished quality of life. Limitedly, and because of lifestyle changes, dietary changes, obesity, metabolic disorders and population ageing, the burden of gout has grown in many populations. The level of uric acid in the serum is affected by the metabolism of purines in the body, as well as by the influence of other external factors, primarily diet[2]. While certain medications, renal dysfunction, obesity, and hypertension are key contributors to gout, dietary exposures might also be modifiable triggers. Purine-rich foods can help to raise uric acid levels and can help to precipitate the monosodium urate crystals in susceptible people[3]. Eating red meat regularly, organ meat and some seafoods have been linked to an increased risk of gout and attacks[4]. Another key dietary factor is alcohol. Alcohol (beer and spirits) can raise serum uric acid levels through a combination of increasing purine load and decreasing renal urate excretion. Therefore, an acute bout of alcohol may trigger a gout attack in a patient who has already had gout. Likewise, sugary drinks and food items with high fructose content can lead to more uric acid production in the liver by causing a fast metabolism of fructose and causing the depletion of ATP in the liver cell[5]. This has led to some concerns about increased intake of soft drinks, processed foods and high-fructose foods in gout-prone patients. Additional eating habits can also trigger a gout attack. An acute attack is more likely to occur if there is any of the following: eating too much, dehydration, long periods without eating, crash dieting or rapid weight loss. Other dietary factors may be linked to a decreased risk of gout attacks, including low-fat dairy foods, sufficient fluid intake, and a balanced diet[6]. However, effects of each food can differ depending on the presence of comorbidities, medication use, baseline serum uric acid values, and other dietary habits. Dietary modification is an integral component of gout management as it allows the patient to contribute to possibly the elimination of triggers to the gout besides urate-lowering therapy. Many patients still don't know which foods are most strongly linked to getting a gout attack; however, what people eat varies widely from one population to the next due to various cultural, socioeconomic, and regional food habits[7]. Dietary factors play a role in gout, too, as many people suffer flares even after medical treatment. Patients frequently complain of attacks occurring after eating a large meal, during holiday seasons, after eating more meat, dehydration, or eating certain drinks. Based on these observations, short-term exposures to certain foods may cause acute flares even if long-term serum uric acid control is being managed[8]. Hence, dietary assessment, based on recent gout attacks, rather than only during the early development of gout, may be more practical. The association between diet and gout gets further complicated considering the common

Received on 12-10-2023
Accepted on 29-12-2023

comorbidities of obesity, diabetes mellitus, hypertension, dyslipidaemia, chronic kidney disease and cardiovascular disease[9]. Dietary preferences may be affected by these conditions as well as uric acid metabolism. Additionally, medications like diuretics, aspirin and urate-lowering therapy can also affect the likelihood of flares and can be confounding factors in the study of dietary associations[10,11].

Objective: To determine the dietary risk factors for gout flares in gout patients and their relationship with the rate of acute gout attacks.

MATERIAL AND METHODS

This analytical cross-sectional study was conducted in the Department of Medicine, Khyber Teaching Hospital Peshawar from January 2023 to June 2023. A total of 165 patients with gout were studied. The participants who met the eligibility criteria were selected using a non-probability consecutive sampling technique. Patients, both male and female, over the age of 18, who had a documented clinical diagnosis and/or accepted classification criteria for gout, were included. The patients who were capable of giving some information about their food intake, medication intake and gout flares history were eligible to be included. Other inflammatory arthritides (rheumatoid arthritis or septic arthritis), patients with acute critical illness, patients with missing clinical or dietary information, incomplete history taking due to cognitive impairment, and patients with malignancy under active chemotherapy were excluded. Wherever there was reliable information on the patient's habit dietary intakes, patients who had recently changed their diet with a specific purpose of managing a gout flare were excluded.

Data Collection: Demographic and clinical data were obtained with informed consent and a structured questionnaire. Data consisted of age, sex, BMI, gout duration, serum uric acid level, smoking, and other medical conditions and medications which may affect serum uric acid. A history of gout flares in the previous year was taken, along with the number of attacks, location of the affected joints, length of time the symptoms lasted before medical treatment was required. A gout flare was considered clinically if the patient had a sudden onset of joint pain with swelling, warmth and/or redness of the joint that met the criteria for acute gout. Patients were classified on the basis of the occurrence and occurrence rate of the gout flares during the study period. Dietary exposure was estimated using a structured, food-frequency section. The participants were asked to report the frequency of consumption and approximate amounts of red meat, organ meat, seafood, poultry, pulses, sugar-sweetened beverages, fructose-rich drinks, high fat foods, dairy products, and other common foods. Data about fast, dehydration, excess food and drinks and alcohol (where applicable) was also gathered. Consumption was classified into frequency categories (rarely or never, 1–2 times per week, 3–4 times per week, ≥5 times per week) according to prespecified categories. The main outcome measured was the number of gout flares in the last year. Secondary outcomes were number of flares and relationship between flares and individual dietary factors. Other clinical variables, such as medication and comorbidities, BMI, and serum uric acid level, were also assessed as potential confounders.

Statistical Analysis: IBM SPSS Statistics 21.0 was used to enter and analyze data. All quantitative variables were presented as mean ± standard deviation or as a median and interquartile range, depending on the distribution of the data. Categorical variables (gender, dietary exposures, smoking, medications, and gout flares) were reported as frequencies and percentages. The chi-square test or Fisher's exact test, as appropriate, was used to determine associations between categorical dietary factors and gout flares. Odds ratios with 95% confidence intervals are presented. The value of $p < 0.05$ considered as statistically significant.

RESULTS

A total of 165 patients with gout were included, with a mean age of 51.6 ± 11.2 years. Most participants were male, 133 (80.6%), while 32 (19.4%) were female. Mean BMI was 28.1 ± 4.2 kg/m², mean duration of gout was 6.1 ± 4.0 years, and mean serum uric acid level was 8.3 ± 1.7 mg/dL. Current smoking was reported by 48 (29.1%) patients, hypertension by 79 (47.9%), diabetes mellitus by 41 (24.8%), and chronic kidney disease by 21 (12.7%). Urate-lowering therapy was being received by 101 (61.2%) patients.

Patients with gout flares had a mean age of 52.0 ± 11.0 years compared with 50.8 ± 11.7 years in those without flares (p=0.523), while mean BMI was 28.5 ± 4.3 versus 27.3 ± 3.9 kg/m² (p=0.086). Mean duration of gout was significantly longer among patients with flares, 6.7 ± 4.2 versus 4.9 ± 3.4 years (p=0.007), and mean serum uric acid was also significantly higher, 8.7 ± 1.6 versus 7.6 ± 1.5 mg/dL (p<0.001).

Red meat intake ≥3 times/week was reported by 58 (51.8%) patients with flares compared with 14 (26.4%) without flares (p=0.002), while organ meat intake ≥1 time/week was reported by 35 (31.3%) versus 7 (13.2%) patients (p=0.013). Sugar-sweetened beverage consumption was higher in the flare group, 55 (49.1%) versus 14 (26.4%) (p=0.006), as were frequent high-fat or heavy meals, 49 (43.8%) versus 14 (26.4%) (p=0.033), low fluid intake or dehydration, 60 (53.6%) versus 14 (26.4%) (p=0.001), and frequent fasting or skipping meals, 38 (33.9%) versus 10 (18.9%) (p=0.047).

Table 1: Demographic and Clinical Characteristics of Patients

Variable	Overall (n=165)
Age, years, Mean ± SD	51.6 ± 11.2
Male, n (%)	133 (80.6%)
Female, n (%)	32 (19.4%)
BMI, kg/m², Mean ± SD	28.1 ± 4.2
Duration of gout, years, Mean ± SD	6.1 ± 4.0
Serum uric acid, mg/dL, Mean ± SD	8.3 ± 1.7
Current smoker, n (%)	48 (29.1%)
Hypertension, n (%)	79 (47.9%)
Diabetes mellitus, n (%)	41 (24.8%)
Chronic kidney disease, n (%)	21 (12.7%)
Receiving urate-lowering therapy, n (%)	101 (61.2%)
At least one gout flare in previous 12 months, n (%)	112 (67.9%)
No gout flare, n (%)	53 (32.1%)

Table 2: Clinical Characteristics According to Presence of Gout Flares

Variable	Gout Flare (n=112)	No Gout Flare (n=53)	p-value
Age, years, Mean ± SD	52.0 ± 11.0	50.8 ± 11.7	0.523
BMI, kg/m², Mean ± SD	28.5 ± 4.3	27.3 ± 3.9	0.086
Duration of gout, years, Mean ± SD	6.7 ± 4.2	4.9 ± 3.4	0.007
Serum uric acid, mg/dL, Mean ± SD	8.7 ± 1.6	7.6 ± 1.5	<0.001
Male, n (%)	92 (82.1%)	41 (77.4%)	0.471
Current smoker, n (%)	36 (32.1%)	12 (22.6%)	0.210
Receiving urate-lowering therapy, n (%)	64 (57.1%)	37 (69.8%)	0.119

Table 3: Association of Dietary Factors With Gout Flares

Dietary Factor	Gout Flare (n=112), n (%)	No Gout Flare (n=53), n (%)	p-value
Red meat ≥3 times/week	58 (51.8%)	14 (26.4%)	0.002
Organ meat ≥1 time/week	35 (31.3%)	7 (13.2%)	0.013
Seafood ≥2 times/week	30 (26.8%)	8 (15.1%)	0.096
Sugar-sweetened beverages ≥3 times/week	55 (49.1%)	14 (26.4%)	0.006
Frequent high-fat/heavy meals	49 (43.8%)	14 (26.4%)	0.033
Low fluid intake/dehydration	60 (53.6%)	14 (26.4%)	0.001
Frequent fasting/skipping meals	38 (33.9%)	10 (18.9%)	0.047
Low-fat dairy ≥4 times/week	29 (25.9%)	25 (47.2%)	0.006

Table 4: Multivariable Logistic Regression for Dietary Predictors of Gout Flares

Predictor	Adjusted OR	95% CI	p-value
Red meat ≥3 times/week	2.41	1.11–5.22	0.026
Organ meat ≥1 time/week	2.58	1.02–6.51	0.045
Seafood ≥2 times/week	1.70	0.76–3.82	0.198
Sugar-sweetened beverages ≥3 times/week	2.36	1.08–5.18	0.032
Frequent high-fat/heavy meals	1.73	0.80–3.74	0.164
Low fluid intake/dehydration	2.89	1.31–6.38	0.009
Frequent fasting/skipping meals	1.64	0.74–3.64	0.224
Low-fat dairy ≥4 times/week	0.48	0.23–0.99	0.047

On multivariable logistic regression, red meat intake ≥3 times/week was independently associated with 2.41 times higher odds of gout flares (95% CI: 1.11–5.22, p=0.026). Organ meat intake ≥1 time/week increased the odds by 2.58 times (95% CI: 1.02–6.51, p=0.045), while sugar-sweetened beverages ≥3 times/week increased the odds by 2.36 times (95% CI: 1.08–5.18, p=0.032).

DISCUSSION

The current study evaluated the dietary risk factors for gout attacks in 165 gout patients. In total, 112 (67.9%) patients had an acute attack of gout at least once in the last 12 months suggesting frequent recurrence in this cohort. The duration of the disease and serum uric acid was significantly higher in patients with gout flares than in those without flares. A number of dietary factors, including regular consumption of red meat, organ meat, sugar-sweetened drinks and not drinking enough water, were linked to a higher risk of gout flare-ups. The mean serum uric acid level was significantly higher among patients with gout flares than among those without flares (8.7 ± 1.6 mg/dL vs. 7.6 ± 1.5 mg/dL, p<0.001). This is clinically predicted since the presence of a high uric acid level in the blood (hyperuricemia) facilitates the accumulation of urate crystals in and around the joints and these crystals can subsequently be a stimulus to recurrent inflammatory attacks. Patients with flares also had a significantly longer duration of gout (6.7 ± 4.2 years) than those without flares (4.9 ± 3.4 years, p=0.007). A lack of long-term control of serum uric acid also has been demonstrated to be related to a higher rate of recurrent gout attacks, as well as a longer duration of the disease, as noted in previous studies[12-14].

Red meat was strongly associated with gout flares, with a higher frequency of consumption. Flares were reported by 51.8% of patients who consumed red meat ≥3 times per week, versus 26.4% of those without flares (p=0.002). Other factors, however, were adjusted for, and frequent red meat consumption was found to be an independent risk factor for gout flares, with an adjusted odds ratio of 2.41. A relatively high purine content is found in red meat, where it is broken down to uric acid and can lead to temporary elevations of serum urate. Previous studies have also linked high intake of meat to the risk of gout and recurrent attacks, suggesting the need for caution of high consumption of purine-rich animal food in patients with existing gout. An important association was found for use of organ meat[15]. Nearly 31.3% of gout patients with flares ate organ meat once a week or more often, while 13.2% of the non-gout patients ate organ meat at least once a week (p=0.013). Univariate and multivariable analyses both showed that organ meat intake was independently associated with flares (adjusted OR=2.58, 95% CI: 1.02–6.51). Purines are especially abundant in organ meats and can have a significant effect on the exogenous purine load. This discovery lends support to current dietary advice for patients with gout to restrict or avoid consuming liver, kidney and other organ meats often[16].

Sugar-sweetened beverages were also significantly associated with gout attacks. Flares were observed in 49.1% of patients who consumed frequently versus 26.4% who did not experience flares (p=0.006). The odds of a gout attack rose about 2.4-fold with frequent intake that was adjusted. Fructose may raise uric acid levels via accelerated metabolic rate in the liver and enhanced degradation of ATP. Earlier studies also showed that high consumption of sugar-sweetened and fructose drinks were associated with hyperuricemia and gout, indicating sugar-sweetened and fructose drinks can be a key component of dietary counseling. An interesting finding in the current study was the link between fluid intake and fluid restriction to the likelihood of gout flares. 53.6% of patients with flares reported low fluid intake or dehydration, while 26.4% reported no flares (p=0.001)[17]. The adjusted odds ratio was 2.89 and it continued to be the strongest dietary predictor. Dehydration can contribute to decreased excretion of uric acid from the kidneys, lead to high levels of urate in body fluids, and contribute to the formation of urate crystals and acute inflammation. Thus, proper hydration of the body, which is easy and practical, should be emphasized as a preventive measure especially for people working outdoors and in hot climates and those who have long fasting periods. Patients with gout flares did not significantly differ in age, gender, BMI, smoking or use of urate-lowering therapy[18]. Despite most of the population (61.2%) being on urate-lowering therapy, recurrent flares were still seen. This could be due to inaccurate dose titration, sub-optimal treatment compliance, inability to reach a therapeutic target serum urate range, and/or the need to continue dietary and lifestyle triggers. So, dietary counselling should be used in addition to and not as a substitute for adequate urate-lowering therapy with drugs.

Limitations: There were several limitations of this study. The cross-sectional design did not allow for a causal relationship to be drawn between dietary factors and gout flares. Food intake and flare history were based primarily on self-report and were open to recall and reporting bias. Dietary subgroups were relatively small, and some had fewer participants, which decreased the statistical power for some of the associations. Dietary intake was not assessed in detail, and there was no detailed information on the quantity of purines served nor on total daily purine intake. In addition, no detailed information was obtained regarding short-term dietary changes before individual flares. Other lifestyle factors might also have confounded the results, as well as medication adherence, urate-lowering therapy dose, and renal function.

CONCLUSION

Frequent intake of red meat, organ meat, and sugar-sweetened beverages, along with inadequate fluid intake, was associated with a higher likelihood of gout flares among patients with gout. Regular low-fat dairy consumption showed a possible protective association. These findings highlight the importance of dietary counseling, adequate hydration, and appropriate urate-lowering therapy in reducing recurrent gout attacks. Dietary modification should therefore be considered an important supportive component of long-term gout management.

REFERENCES

1. Singh JA, Reddy SG, Kundukulam J. Risk factors for gout and prevention: a systematic review of the literature. Curr Opin Rheumatol. 2011 Mar;23(2):192-202. doi: 10.1097/BOR.0b013e3283438e13. PMID: 21285714; PMCID: PMC4104583.
2. Li R, Yu K, Li C. Dietary factors and risk of gout and hyperuricemia: a meta-analysis and systematic review. Asia Pac

J Clin Nutr. 2018;27(6):1344-1356. doi: 10.6133/apjcn.201811_27(6).0022. PMID: 30485934.
3. Glovaci D, Fan W, Wong ND. Epidemiology of diabetes mellitus and cardiovascular disease. Curr Cardiol Rep. 2019;21:21. doi:10.1007/s11886-019-1107-y.
4. Viigimaa M, Sachinidis A, Toumpourleka M, Koutsampasopoulos K, Alliksoo S, Titma T. Macrovascular complications of type 2 diabetes mellitus. Curr Vasc Pharmacol. 2020;18:110-116. doi:10.2174/1570161117666190405165151.
5. Kolb H, Martin S. Environmental/lifestyle factors in the pathogenesis and prevention of type 2 diabetes. BMC Med. 2017;15:131. doi:10.1186/s12916-017-0901-x.
6. Wallace KL, Riedel AA, Joseph-Ridge N, Wortmann R. Increasing prevalence of gout and hyperuricemia over 10 years among older adults in a managed care population. J Rheumatol. 2004;31:1582-1587.
7. Menon A, Roddy E. Update on factors predisposing to gout. Int J Clin Rheumatol. 2009;4:75-87. doi:10.2217/17584272.4.1.75.
8. Singh JA, Reddy SG, Kundukulam J. Risk factors for gout and prevention: a systematic review of the literature. Curr Opin Rheumatol. 2011;23:192-202. doi:10.1097/BOR.0b013e3283438e13.
9. Rho YH, Lu N, Peloquin CE, Man A, Zhu Y, Zhang Y. Independent impact of gout on the risk of diabetes mellitus among women and men: a population-based, BMI-matched cohort study. Ann Rheum Dis. 2016;75:91-95. doi:10.1136/annrheumdis-2014-205827.
10. Li C, Hsieh MC, Chang SJ. Metabolic syndrome, diabetes, and hyperuricemia. Curr Opin Rheumatol. 2013;25:210-216. doi:10.1097/BOR.0b013e32835d951e.
11. Bardin T, Richette P. Impact of comorbidities on gout and hyperuricaemia: an update on prevalence and treatment options. BMC Med. 2017;15:123. doi:10.1186/s12916-017-0890-9.
12. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes. 1988;37:1595-1607. doi:10.2337/diab.37.12.1595.
13. Vuorinen-Markkola H, Yki-Järvinen H. Hyperuricemia and insulin resistance. J Clin Endocrinol Metab. 1994;78:25-29. doi:10.1210/jcem.78.1.8288709.
14. King C, Lanaspa MA, Jensen T, Tolan DR, Sánchez-Lozada LG, Johnson RJ. Uric acid as a cause of the metabolic syndrome. Contrib Nephrol. 2018;192:88-102. doi:10.1159/000484283.
15. van der Schaft N, Brahimaj A, Wen KX, Franco OH, Dehghan A. The association between serum uric acid and the incidence of prediabetes and type 2 diabetes mellitus: the Rotterdam Study. PLoS One. 2017;12:e0179482. doi:10.1371/journal.pone.0179482.
16. Kodama S, Saito K, Yachi Y, Asumi M, Sugawara A, Totsuka K, et al. Association between serum uric acid and development of type 2 diabetes. Diabetes Care. 2009;32:1737-1742. doi:10.2337/dc09-0288.
17. Wallace SL, Robinson H, Masi AT, Decker JL, McCarty DJ, Yü TF. Preliminary criteria for the classification of the acute arthritis of primary gout. Arthritis Rheum. 1977;20:895-900. doi:10.1002/art.1780200320.
18. Alberti G. A desktop guide to type 2 diabetes mellitus. European Diabetes Policy Group 1998-1999, International Diabetes Federation European Region. Exp Clin Endocrinol Diabetes. 1999;107:390-420.

This article may be cited as: Ullah N., Shahzad A., Hadi K. N., Ali A., Zafar M., Khan I., Ullah I., Adil M., Khan S., Ahmad I., Dietary Risk Factors Associated with Gout Flares Among Patients with Gout. Pak J Med Health Sci, 2023; 18(1): 1070-1073.