



Prevalence of metabolic syndrome in gout patients referred to a rheumatology clinic in Kashan from 2013 to 2023

Batol Zamani¹, Hossein Motedayyen^{1*}, Hamidreza Gilasi², Fatemeh Mehdizadeh¹

¹ Autoimmune Diseases Research Center, Kashan University of Medical Sciences, Kashan, Iran

² Social Department of Health Research Center, Faculty of Health, Kashan University of Medical Sciences, Kashan, Iran

* **Corresponding author:** Hossein Motedayyen, Autoimmune Diseases Research Center, Shahid Beheshti Hospital, Kashan University of Medical Sciences, Kashan, Iran. **Email:** hmotedayyen@gmail.com

Received: 24 January 2024 **Revised:** 26 February 2024 **Accepted:** 9 March 2024 **e-Published:** 9 March 2024

Abstract

Background: Gout, characterized by the accumulation of crystals in soft tissues and joints, is caused by elevated uric acid levels in the blood. Diagnosis of gout typically involves evaluating synovial fluid crystals. Metabolic syndrome, a cluster of metabolic disorders, increases the risk of cardiovascular disease.

Objectives: This study was conducted to assess the prevalence of metabolic syndrome in gout patients referred to a rheumatology clinic in Kashan from 2013 to 2023.

Methods: A study was conducted to assess the prevalence of metabolic syndrome in gout patients referred to a rheumatology clinic in Kashan from 2013 to 2023. The study included data from 180 participants aged 21 to 92 years. Demographic data, fasting blood sugar, lipid profile, and physical examination results (blood pressure and weight) were recorded in a questionnaire.

Results: The study revealed that 88.3% of gout patients were male, with a mean age of 58.41 ± 17.22 years. The mean body mass index was 29.6 ± 4.14 , with 49.8% of patients classified as overweight. More than half of the patients (55.6%) had metabolic syndrome, 70% had high blood pressure, 33.9% had diabetes, 39.44% were obese, 32.8% had abdominal obesity, and 40.6% had low HDL levels. Components of metabolic syndrome were present in 90% of individuals. Uric acid levels were not significantly associated with metabolic syndrome ($P=0.814$). Statistically significant correlations were found between metabolic syndrome, obesity, and age (P values <0.001 and 0.020).

Conclusions: Given the high prevalence of metabolic syndrome among gout patients and its significant impact on cardiovascular health, early detection and management of this syndrome in these patients are crucial.

Keywords: Gout, Hyperuricemia, Metabolic Syndrome.

Introduction

Gout is a metabolic disorder that commonly affects middle-aged to elderly men and postmenopausal women. It is characterized by recurrent episodes of acute or chronic arthritis. This condition arises when the body accumulates uric acid in the form of urate. The normal uric acid level in the blood is up to 6.8 mg/dL, with levels above this considered hyperuricemia. The buildup of needle-shaped monosodium urate crystals in soft tissues can trigger gout.^[1]

Uric acid is the end product of purine metabolism in the body, and its levels can be influenced by various factors like genetics, diet, alcohol intake, and kidney function. Elevated uric acid levels have been associated with several

comorbid conditions, including coronary artery disease, stroke, metabolic syndrome, diabetes, and hypertension.^[2] According to a 2007-2008 survey in the United States, the prevalence of gout was 3.9%, with men being three times more affected than women.^[3] The prevalence of gout was 2.7% in 1994, increasing to 3.9% in 2008.^[4] In white men, the prevalence of gout is 4%, rising to 5% in African-American men. The NHANES study reported that African-Americans had a 1.69 times higher risk of gout compared to white men.^[5]

Risk factors for gout include environmental influences such as smoking, alcohol consumption, dietary habits, medications, chronic illnesses, and genetic predisposition.^[6,7] Research has shown a significant

relationship between alcohol and beer consumption and an increased risk of gout.^[8,9] Regular consumption of beverages containing fructose twice a day can elevate the risk of developing gout and hyperuricemia in men. Similarly, the intake of meat and seafood can substantially raise the likelihood of gout development. Conversely, consuming low-fat or fat-free dairy products can help mitigate the risk of gout. It's worth noting that moderate consumption of purine- and protein-rich vegetables can also heighten the risk of gout.

Conversely, increasing caffeine consumption (excluding tea) can lower the risk of gout in men. Maintaining a balanced and healthy diet is crucial for preventing the risk of developing gout and hyperuricemia.^[10,11] Laboratory studies have demonstrated that elevated urate levels can impair endothelial function by inhibiting nitric oxide production and vasodilation. This allows urate to enter cells through the URAT-1 receptor expression on endothelial cells, activating the renin-angiotensin system. In rats, urate has been shown to increase glomerular filtration. Recent research has identified monosodium urate crystals and calcification in coronary artery walls and the thoracic aorta in individuals with gout, suggesting urate-induced vessel inflammation.^[12,13]

Recent studies have also suggested that high serum urate levels may raise the risk of cardiovascular morbidity and mortality. However, it remains uncertain whether hyperuricemia is an independent risk factor or a confounding factor for cardiovascular disease (CVD), as many established CVD risk factors are also linked to increased serum urate levels.^[14]

High levels of uric acid react irreversibly with nitric oxide (NO), leading to endothelial dysfunction, which may ultimately result in hypertension and the development of metabolic syndrome.^[15] Additionally, NO plays a role in insulin resistance, and its deficiency can decrease blood supply to tissues and reduce insulin sensitivity, potentially blocking insulin action.^[13] Elevated uric acid levels in the body can induce oxidative stress by inhibiting endothelial NO, resulting in reduced vascular vasodilation, decreased blood flow to target tissues, diminished insulin sensitivity, increased insulin production, and long-term resistance. Uric acid may also trigger inflammation and insulin resistance in fat tissue by affecting the PPAR- γ nuclear receptor.

Endothelial dysfunction increases peripheral resistance, leading to smooth muscle cell growth in the media layer of blood vessels.^[16] Studies in the US have shown an inverse relationship between uric acid levels and diabetes.^[17] Similarly, Japanese men exhibited a similar inverse

relationship between uric acid levels and diabetes, although this association was not observed in Japanese women.^[18]

Uric acid can harm the kidneys and elevate blood pressure, particularly in advanced stages. Several studies have demonstrated that reducing uric acid levels with allopurinol can aid in treating high blood pressure and is effective in the pathogenesis of chronic hypertension. Moreover, individuals with gout have a higher incidence of chronic kidney disease (CKD). A UK study revealed that within a 3-year follow-up period, 78% of gout patients compared to non-gout individuals showed an increased risk of CKD stage 3 or higher due to the direct effects of gout on the kidney and comorbidities such as diabetes, hypertension, kidney stones, and Non-steroidal anti-inflammatory drugs (NSAIDs) overuse.

Elevated uric acid levels contribute to obesity by inhibiting adiponectin production through oxidative stress induction and NADPH oxidase activation. This leads to the generation of oxidized lipids and inflammatory mediators like monocyte chemoattractant protein, associated with body weight. Some studies suggest a higher frequency of gout-related stroke among women.^[20,21]

Metabolic syndrome, also known as syndrome X or insulin resistance syndrome, comprises a cluster of metabolic disorders that increase the risk of CVD and diabetes. Major features include central obesity, hypertriglyceridemia, hyperglycemia, hypertension, and low HDL levels. Diagnosis is based on NCEP-ATP III criteria.^[1]

Uric acid possesses endogenous antioxidant properties that safeguard against diseases caused by oxidants and free radicals.^[22] Very low uric acid levels are linked to endothelial dysfunction and cardiovascular events.^[23]

Gout is a common disease associated with metabolic syndrome. Proper diagnosis and treatment of gout and related conditions can effectively reduce the risk of cardiovascular complications and mortality. Therefore, we conducted this research to enhance follow-up care for these patients.

Objectives

This study was conducted to assess the prevalence of metabolic syndrome in gout patients referred to a rheumatology clinic in Kashan from 2013 to 2023.

Methods

In this cross-sectional study, all patients diagnosed with

gout through joint aspiration and the presence of uric acid crystals within white blood cells were included if they also had at least one-joint rheumatoid arthritis.

To assess the presence of metabolic syndrome, blood pressure measurements were taken in the morning after a 10-minute rest in a quiet room while seated. Blood pressure was measured twice with a 5-minute interval, and the average value was recorded. A systolic blood pressure above 130 mm Hg or diastolic blood pressure above 85 mm Hg was considered the initial criterion for metabolic syndrome.^[1]

Blood samples were collected from fasting patients to evaluate potential comorbidities and risk factors associated with gout. Tests included measurements of fasting blood sugar, total cholesterol, HDL, LDL, uric acid, and creatinine levels. The diagnosis of metabolic syndrome was based on five criteria: 1) systolic blood pressure >130 mm Hg or diastolic blood pressure >85 mm Hg; 2) triglyceride levels >150; 3) HDL levels <40 in men or <50 in women; 4) blood sugar >100 mg/dL or existing type 2 diabetes; and 5) waist circumference >102 cm in men or >88 cm in women. Meeting at least three of these criteria classified a gout patient as having metabolic syndrome.^[1]

Hyperuricemia was defined as a uric acid level exceeding 6.8 mg/dL. Obesity was determined by calculating the Body Mass Index (BMI) based on height and weight measurements, with a BMI \geq 30 indicating obesity. Demographic information, such as age, sex, gout duration, history of kidney stones, CVD, and family history of gout, was recorded in the questionnaire.^[1]

Statistical analysis was performed by an expert on the collected data, with 180 gout patients randomly selected using online random number generation. Data were entered into SPSS 22 for analysis. Frequency distribution tables and graphs were used to describe qualitative variables, while central dispersion indices were employed for quantitative variables. Correlations between quantitative variables were assessed using Pearson's correlation coefficient and Spearman's correlation coefficient for ranks. The relationship between qualitative variables was evaluated using Chi-square or Fisher's exact test.

Statistical analysis

The continuous variables were expressed as the mean \pm SD, and the categorical variables were presented as a percentage and frequency. Because the data showed a non-normal distribution, the Mann-Whitney test was used to compare the parameters between patients and health

groups. The relations between parameters were evaluated using the Pearson correlation coefficient. All statistical analyses were performed with SPSS (version 16.0, SPSS Inc, Chicago, IL, USA). A "P-value" less than 0.05 was considered significant.

Ethical considerations

The study was conducted in accordance with the Declaration of Helsinki. Institutional Review Board approval was obtained. The present study did not interfere with the process of diagnosis and treatment of patients and all participants signed an informed consent form.

Results

Out of the 180 patients diagnosed with gout, 88.3% (159) were male. The average BMI of the patients was 29.6 ± 4.14 , indicating overweight, and the average age was 58.41 ± 17.22 years. The demographic characteristics of the patients are presented in Table 1.

Among the 180 gout patients, 100 (55.6%) had a diagnosis of metabolic syndrome. Blood pressure, with a frequency of 70% (100 individuals), was the most prevalent component of metabolic syndrome. The frequency of metabolic syndrome and its components is illustrated in Table 2.

Table 3 demonstrates that 90% of patients had at least one component of metabolic syndrome. The highest frequency was related to the presence of three components, which included 30% of patients. Among those with metabolic syndrome, more than half (54 individuals) exhibited three components of the syndrome.

The study revealed that 59.1% of participants over the age of 40 were diagnosed with metabolic syndrome, compared to 34.6% among those aged 40 or younger. The relationship between age and metabolic syndrome was statistically significant with a P-value of 0.020. Among male subjects, 54.1% were found to have metabolic syndrome; however, no significant relationship was observed between metabolic syndrome and gender in gout patients ($P=0.276$).

Regarding BMI, 80.3% of obese patients (BMI >30) had metabolic syndrome. The relationship between metabolic syndrome and obesity (higher BMI) was statistically significant ($P<0.001$).

The average uric acid levels in patients with and without metabolic syndrome did not show a significant difference. There was no significant relationship between uric acid levels and metabolic syndrome ($P=0.814$).

Table 1. Demographic information of the patients

Variable	Data
Total number of patients	180
Number of males (percentage)	159 (88.3%)
Number of females (percentage)	21 (11.7%)
Average age (year)	58.41 ± 17.22
Average body mass index (BMI)	29.6 ± 4.14
Uric acid level (mg/dL)	8.26 ± 1.93
Males waist circumference	96.13 ± 6.81
Females waist circumference	86.04 ± 4.55
Triglyceride level (mg/dL)	170.81 ± 81.7
Fasting blood sugar (mg/dL)	109.23 ± 35.41
Systolic blood pressure (mmHg)	136.41 ± 11.02
Diastolic blood pressure (mmHg)	88.12 ± 8.26
Males HDL levels (mg/dL)	45.5 ± 8.5
Females HDL levels (mg/dL)	49.5 ± 8.5

Table 2. Frequency of metabolic syndrome and its components

Variable	Percentage
Metabolic syndrome	55.6%
Blood pressure (mmHg)	70%
Hypertriglyceridemia	48.3%
low HDL (mg/dL)	40.6%
Diabetes	33.9%
Abdominal obesity	32.8%
Obesity	39.44%

Table 3. Frequency of metabolic syndrome components in patients

Component (n)	Patient (n)	Percentage
0	18	10%
1	45	25%
2	20	11.1%
3	54	30%
4	33	18.3%
5	10	5.6%
Total	108	100%

Discussion

The study aimed to determine the frequency of metabolic syndrome in gout patients who visited the rheumatology clinic in Kashan, Iran, between 2013 and September 2014. The study included 180 participants aged between 19 and 92 years, with an average age of 58.31±17.69.

Out of the 180 participants, 100 were found to have metabolic syndrome, indicating a frequency of 55.6%, which is consistent with studies by Jae Hyun Jung, Marie Doualla-Bija, and Aguilar-Andrad.^[24-26] However, some studies have reported lower frequencies of metabolic

syndrome.^[27,28]

Gout is more prevalent in males than females, typically affecting middle-aged or older males and postmenopausal females. In our study, 88.3% of the participants were male, aligning with Ismailzadeh's study and previous research.^[24, 29-30]

The mean age of the patients in our study was 58.41±17.22, falling within the middle age range, consistent with other studies by Ismailzadeh and Saghafi.^[25,29,31,32] Our study found no statistically significant association between metabolic syndrome and gender.

We investigated the relationship between metabolic syndrome and age, revealing a significant correlation with increasing age ($P=0.020$). Additionally, we explored the link between obesity and the risk of developing gout and metabolic syndrome. Our findings showed that 39.44% of the study population was obese, in line with previous publications. We also found a statistically significant relationship between metabolic syndrome and obesity, as higher BMI was associated with a higher prevalence of metabolic syndrome ($P\leq 0.001$).

Our study on the frequency of components of metabolic syndrome in patients indicated that hypertension was the most common component, with a frequency of 70%, consistent with several previous studies.^[24,25,31,33-35] The frequencies of hypertriglyceridemia, low HDL, diabetes, and abdominal obesity were 48.3%, 40.6%, 33.9%, and 32.8%, respectively.

This study examined the prevalence of metabolic syndrome components among gout patients. It was observed that 90% of the patients exhibited at least one component of metabolic syndrome. Among those with metabolic syndrome, the most common number of components present was three, seen in 30% of the patients. One component was identified in 25% of the patients, four components in 18.3%, two components in 11.1%, and five components in 5.6% of the patients. Only 10% of the patients showed no signs of metabolic syndrome. Therefore, out of the patients with metabolic syndrome, 54 individuals had three components, representing over half of all patients with metabolic syndrome.

Our study aimed to explore the potential role of uric acid in the development of metabolic syndrome by comparing uric acid levels in patients with and without the condition. Given that hyperuricemia is a known factor in gout development, we aimed to investigate its impact on metabolic syndrome as well. The average uric acid level in patients with metabolic syndrome was 8.51±1.87, while those without metabolic syndrome had an average uric

acid level of 7.95 ± 1.94 . However, subsequent statistical analysis revealed no significant difference between metabolic syndrome and uric acid levels ($P=0.814$). These results are consistent with Fraile's study, which also reported no statistically significant variance in mean serum uric acid levels between individuals with and without metabolic syndrome.^[31]

Conclusions

The study revealed a higher prevalence of metabolic syndrome in gout patients (55.6%) compared to the general population (25-30%). This finding is consistent with similar studies conducted in various countries. Therefore, it is crucial for clinicians to be mindful of the elevated prevalence of gout and its significant association with metabolic syndrome and CVD. Consequently, clinicians should prioritize assessing for the presence of metabolic syndrome in gout patients to effectively prevent, diagnose, and manage any cardiovascular risk factors in this patient population.

Acknowledgment

The authors would like to thank the Clinical Research Development Unit of Kashan Shahid Beheshti hospital.

Competing interests

The authors declare that they have no competing interests.

Abbreviations

Cardiovascular disease: CVD; Nitric oxide: NO;
Chronic kidney disease: CKD; Body Mass Index: BMI;
Non-steroidal anti-inflammatory drugs: NSAIDs.

Authors' contributions

All authors read and approved the final manuscript. All authors take responsibility for the integrity of the data and the accuracy of the data analysis.

Funding

None.

Role of the funding source

None.

Availability of data and materials

The data used in this study are available from the corresponding author on request.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. Institutional Review Board approval was obtained. The present study did not interfere with the

process of diagnosis and treatment of patients and all participants signed an informed consent form.

Consent for publication

By submitting this document, the authors declare their consent for the final accepted version of the manuscript to be considered for publication.

References

- Weidong Z, Bin L, Huaiping Z. A study on the correlation between tumor escape in ovarian carcinoma and the expression of transcription factors T-bet/GATA3. *Acta Universitatis Medicinalis Nahui*. 2004; 3(009).
- Ford ES, Li C, Cook S, Choi HK. Serum concentrations of uric acid and the metabolic syndrome among US children and adolescents. *Circulation*. 2007; 115(19):2526-32. doi:10.1161/CIRCULATIONAHA.106.657627 PMID:17470699
- Singh JA, Gaffo A. Gout epidemiology and comorbidities. *Semin Arthrit Rheum*. 2020; 50(3s):S11-s6. doi:10.1016/j.semarthrit.2020.04.008 PMID:32620196
- Zhu Y, Pandya BJ, Choi HK. Prevalence of gout and hyperuricemia in the US general population: the National Health and Nutrition Examination Survey 2007-2008. *Arthrit Rheum*. 2011; 63(10):3136-41. doi:10.1002/art.30520 PMID:21800283
- Chen-Xu M, Yokose C, Rai SK, Pillinger MH, Choi HK. Contemporary Prevalence of Gout and Hyperuricemia in the United States and Decadal Trends: The National Health and Nutrition Examination Survey, 2007-2016. *Arthrit Rheum*. 2019; 71(6):991-9 doi:10.1002/art.40807 PMID:30618180 PMCID:PMC6536335
- Singh JA, Reddy SG, Kundukulam J. Risk factors for gout and prevention: a systematic review of the literature. *Curr Opin Rheumatol*. 2011; 23(2):192-202 doi:10.1097/BOR.0b013e3283438e13 PMID:21285714 PMCID:PMC4104583
- McCormick N, Rai SK, Lu N, Yokose C, Curhan GC, Choi HK. Estimation of Primary Prevention of Gout in Men through Modification of Obesity and Other Key Lifestyle Factors. *JAMA Netw Open*. 2020; 3(11):e2027421 doi:10.1001/jamanetworkopen.2020.27421 PMID:33231639 PMCID:PMC7686865
- Choi HK, Atkinson K, Karlson EW, Willett W, Curhan G. Alcohol intake and risk of incident gout in men: a prospective study. *Lancet*. 2004; 363(9417):1277-81 doi:10.1016/S0140-6736(04)16000-5 PMID:15094272
- Neogi T, Chen C, Niu J, Chaisson C, Hunter DJ, Zhang Y. Alcohol quantity and type on risk of recurrent gout attacks: an internet-based case-crossover study. *Am J Med*. 2014; 127(4): 311-8. doi:10.1016/j.amjmed.2013.12.019 PMID:24440541 PMCID:PMC3991555
- Choi HK, Atkinson K, Karlson EW, Willett W, Curhan G. Purine-rich foods, dairy and protein intake, and the risk of gout in men. *N Engl J Med*. 2004; 350(11):1093-103. doi:10.1056/NEJMoa035700 PMID:15014182
- Choi HK, Willett W, Curhan G. Coffee consumption and risk of incident gout in men: a prospective study. *Arthritis Rheum*. 2007; 56(6):2049-55. doi:10.1002/art.22712 PMID:17530645

12. Mazzali M, Hughes J, Kim YG, Jefferson JA, Kang DH, Gordon KL, et al. Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. *Hypertension*. 2001; 38(5):1101-6 [doi:10.1161/hy.1101.092839](https://doi.org/10.1161/hy.1101.092839) PMID:11711505
13. Klauser AS, Halpern EJ, Strobl S, Gruber J, Feuchtner G, Bellmann-Weiler R, et al. Dual-Energy Computed Tomography Detection of Cardiovascular Monosodium Urate Deposits in Patients With Gout. *JAMA Cardiol*. 2019; 4(10):1019-28. [doi:10.1001/jamacardio.2019.3201](https://doi.org/10.1001/jamacardio.2019.3201) PMID:31509156 PMID:PMC6739730
14. Cicero AFG, Fogacci F, Giovannini M, Grandi E, Rosticci M, D'Addato S, et al. Serum uric acid predicts incident metabolic syndrome in the elderly in an analysis of the Brisighella Heart Study. *Sci Rep*. 2018; 8(1):11529. [doi:10.1038/s41598-018-29955-w](https://doi.org/10.1038/s41598-018-29955-w) PMID:30068918 PMID:PMC6070523
15. Gersch C, Pali SP, Kim KM, Angerhofer A, Johnson RJ, Henderson GN. Inactivation of nitric oxide by uric acid. *Nucleosides Nucleotides Nucleic Acids*. 2008; 27(8):967-78. [doi:10.1080/15257770802257952](https://doi.org/10.1080/15257770802257952) PMID:18696365 PMID:PMC2701227
16. Hwu CM, Lin KH. Uric acid and the development of hypertension. *Med Sci Monit*. 2010; 16(10):Ra224-30.
17. Bandaru P, Shankar A. Association between Serum Uric Acid Levels and Diabetes Mellitus. *Int J Endocrinol*. 2011; 2011:604715. [doi:10.1155/2011/604715](https://doi.org/10.1155/2011/604715) PMID:22114591 PMID:PMC3216382
18. Oda E, Kawai R, Sukumaran V, Watanabe K. Uric acid is positively associated with metabolic syndrome but negatively associated with diabetes in Japanese men. *Intern Med (Tokyo, Japan)*. 2009; 48(20):1785-91. [doi:10.2169/internalmedicine.48.2426](https://doi.org/10.2169/internalmedicine.48.2426) PMID:19834269
19. Feig DI, Soletsky B, Johnson RJ. Effect of allopurinol on blood pressure of adolescents with newly diagnosed essential hypertension: a randomized trial. *JAMA*. 2008; 300(8):924-32. [doi:10.1001/jama.300.8.924](https://doi.org/10.1001/jama.300.8.924) PMID:18728266 PMID:PMC2684336
20. Wen CP, David Cheng TY, Chan HT, Tsai MK, Chung WS, Tsai SP, et al. Is high serum uric acid a risk marker or a target for treatment? Examination of its independent effect in a large cohort with low cardiovascular risk. *Am J Kidney Dis*. 2010; 56(2):273-88. [doi:10.1053/j.ajkd.2010.01.024](https://doi.org/10.1053/j.ajkd.2010.01.024) PMID:20605302
21. Vedder D, Walrabenstein W, Heslinga M, de Vries R, Nurmohamed M, van Schaardenburg D, et al. Dietary Interventions for Gout and Effect on Cardiovascular Risk Factors: A Systematic Review. *Nutrients*. 2019; 11(12). [doi:10.3390/nu11122955](https://doi.org/10.3390/nu11122955) PMID:31817107 PMID:PMC6950134
22. Ames BN, Cathcart R, Schwiers E, Hochstein P. Uric acid provides an antioxidant defense in humans against oxidant- and radical-caused aging and cancer: a hypothesis. *Proceedings of the National Academy of Sciences of the United States of America*. 1981; 78(11):6858-62. [doi:10.1073/pnas.78.11.6858](https://doi.org/10.1073/pnas.78.11.6858) PMID:6947260 PMID:PMC349151
23. Iso T, Kurabayashi M. Extremely low levels of serum uric acid are associated with endothelial dysfunction in humans. *Circulat J* 2015; 79(5):978-80. [doi:10.1253/circj.CJ-15-0232](https://doi.org/10.1253/circj.CJ-15-0232) PMID:25787674
24. Jung JH, Song GG, Ji JD, Lee YH, Kim J, Seo Y, et al. Metabolic syndrome: prevalence and risk factors in Korean gout patients. *Korean J Inter Med*. 2018;33(4):815. [doi:10.3904/kjim.2016.062](https://doi.org/10.3904/kjim.2016.062) PMID:27729624 PMID:PMC6030414
25. Doualla-Bija M, Batchama YL, Moutchia-Suh J, Moor VJA, Kamdem F, Lekpa FK, et al. Prevalence and characteristics of metabolic syndrome in gout patients in a hospital setting in sub-Saharan Africa. *Diabetes Metab Syndr: Cli Res Rev*. 2018; 12(6):1007-11. [doi:10.1016/j.dsx.2018.06.015](https://doi.org/10.1016/j.dsx.2018.06.015) PMID:29980454
26. Aguilar-Andrade G, Álvarez-Bañuelos T, Morales-Romero J, Fonseca-Reyes S, Margozzini-Maira P, Carvajal-Moreno M. Relationship between uric acid and metabolic syndrome among users of a primary care clinic in Mexico. *Metabolomics (Los Angel)*. 2018; 8(1):197 [doi:10.4172/2153-0769.1000197](https://doi.org/10.4172/2153-0769.1000197)
27. Dao HH, Harun-Or-Rashid M, Sakamoto J. Body composition and metabolic syndrome in patients with primary gout in Vietnam. *Rheumatology*. 2010; 49(12):2400-7 [doi:10.1093/rheumatology/keq274](https://doi.org/10.1093/rheumatology/keq274) PMID:20817682
28. Bevis M, Blagojevic-Bucknall M, Mallen C, Hider S, Roddy E. Comorbidity clusters in people with gout: an observational cohort study with linked medical record review. *Rheumatology*. 2018; 57(8):1358-63 [doi:10.1093/rheumatology/key096](https://doi.org/10.1093/rheumatology/key096) PMID:29672754
29. Esmaeilzadeh M, Bashiri SA, Tahmasbirad M, Khalvat A, Rostamian A. Epidemiology and clinical manifestation of GOUT in Imam Khomeini Hospital in Tehran, Iran. 2009.
30. Fraile J, Torres R, de Miguel ME, Martinez P, Lundelin K, Vazquez J, et al. Metabolic syndrome characteristics in gout patients. *Nucleosides Nucleotides Nucleic Acids*. 2010; 29(4-6):325-9. [doi:10.1080/15257771003738709](https://doi.org/10.1080/15257771003738709) PMID:20544515
31. Richette P, Clerson P, Périssin L, Flipo R-M, Bardin T. Revisiting comorbidities in gout: a cluster analysis. *Ann Rheum Dis*. 2015; 74(1):142-7 [doi:10.1136/annrheumdis-2013-203779](https://doi.org/10.1136/annrheumdis-2013-203779) PMID:24107981
32. Saghafi M, Sahebari M, Elahi H. Evaluation of patients with chronic gouty arthritis. *J Inflamm Dis*. 2012; 14(2):76-82
33. Choi HK, Ford ES, Li C, Curhan G. Prevalence of the metabolic syndrome in patients with gout: the Third National Health and Nutrition Examination Survey. *Arthrit Care Res: Official J Am College Rheumatol*. 2007; 57(1):109-15. [doi:10.1002/art.23245](https://doi.org/10.1002/art.23245) [doi:10.1002/art.22466](https://doi.org/10.1002/art.22466) PMID:17266099
34. Rho YH, Choi SJ, Lee YH, Ji JD, Choi KM, Baik SH, et al. The prevalence of metabolic syndrome in patients with gout: a multicenter study. *J Korean Med* 20(6):1029-33. [doi:10.3346/jkms.2005.20.6.1029](https://doi.org/10.3346/jkms.2005.20.6.1029) PMID:16361817 PMID:PMC2779304
35. González-Senac NM, Bailén R, Torres RJ, de Miguel E, Puig JG. Metabolic syndrome in primary gout. *Nucleosides, Nucleotides Nucleic Acids*. 2014;33 (4-6):185-91. [doi:10.1080/15257770.2013.853785](https://doi.org/10.1080/15257770.2013.853785) PMID:24940668

How to Cite this Article:

Zamani B, Motedayyen H, Gilasi H, Mehdizadeh F. Prevalence of metabolic syndrome in gout patients referred to a rheumatology clinic in Kashan from 2013 to 2023. *Basic Clin Biochem Nutr*. 2024;1(1):24-29. [doi:10.48307/bcbn.2024.445493.1006](https://doi.org/10.48307/bcbn.2024.445493.1006)