Acta Medica Alanya

RESEARCH ARTICLE

ARAŞTIRMA

Acta Medica Alanya

2019;3(1):54-58

DOI:10.30565/medalanya.505058

Prevalence of Chronic Kidney Disease and Hyperuricemia In Gout Arthritis Patients

Gut Artritli Hastalarda Hiperürisemi ve Kronik Böbrek Hastalığının Prevalansı

Can Hüzmeli[1*], Meryem Timucin[2], Murat Güllü[3], Kazım Öztürk[3], Eylem Yetimoğlu[4], Ferhan Candan[2]

1. *Kahramanmaraş Necip Fazıl City Hospital, Department of Nefrology, Kahramanmaraş, Turkey*
2. *Cumhuriyet Universty, faculty of Medicine, Deperment of Nefrology, Sivas, Turkey*
3. *Hatay State Hspital, Depermant of İnternal Medicine, Hatay, Turkey*
4. *Kahramanmaraş Necip Fazıl City Hospital, Department of Romatology, Kahramanmaraş, Turkey*

ABSTRACT

Aim: The aim of this study is to determine the prevalence of Chronic renal disease (CKD) in the patients known with gout arthritis.

Method:A total of 162 patients with gout arthritis diagnosed between 2014 and 2017 were included in the study. Our work is a retrospective study. Glomerular filtration rate was calculated by Modification of Renal Disease (MDRD) method.

Results: The mean age of the patients was found as 59,64 ± 14,54 (18-93). The majority of patients are male. Mean uric acid levels of the patients were found to be 9.07 mg / dL ± 1.75 (4-14.7). Among these individuals with gout arthritis, % 39,5 (64) had CKD stage 3-5, %46,9 (76) had hypertension, %18,5 (30) had diabetes mellitus, %14,2 (23) had coronary artery disease. 33.3% of the patients had CKD stage 2. Nephrolithiasis was detected in 22 of 85 ultrasound patients. The use of diuretics was detected in 36 of the patients. The consultation rate requested by the dietician was 41,4%.

Conclusions: Hyperuricemia and hypertension are high in CKD. Diuretics used in the treatment of hypertension and edema may trigger gout arthritis. Similar to other studies in our study, the prevalence of CKD in gout arthritis was found high.

Key word;Gout arthritis, uric acid, chronic kidney disease

ÖZ

Amaç: Bu çalışmanın amacı, gut artriti tanısı konan hastalarda kronik böbrek hastalığı (KBH) prevalansını saptamaktır.

Yöntem: Bu çalışmaya 2014 ile 2017 tarihleri arasında gut artirti tanısı konan toplam 162 hasta alındı. Çalışmamız retrospektif bir çalışmadır. Glomeruler filtrasyon hızı Modification of Renal Disease (MDRD) yöntemiyle hesaplandı.

Bulgular: Hastaların yaş ortalaması 59,64±14,54 (18-93) olarak bulundu. Hastaların çoğunluğu erkekti. Hastaların ortalama ürik asit seviyeleri 9,07mg/dl±1,75(4-14,7) olarak bulundu. Gut artritli bireylerin % 39,5'i (64) KBH evre 3-5, % 46.9'u (46) hipertansiyon, % 18,5 (30) diabetes mellitus, % 14,2'sinde (23) koroner arter hastalığı mevcuttu. Hastaların %33,3'ü CKD evre 2 olarak saptandı. Ultrasonu yapılmış 85 hastanın 22'sinde nefrolitiyazis vardı. Hastaların 36'sında diüretik kullanımı tespit edildi. Diyetisyenden istenilen konsültasyon oranı %41,4 idi.

Sonuçlar: KBH'de hiperürisemi ve hipertansiyon sıklığı yüksektir. Hipertansiyon ve ödem tedavisinde kullanılan diüretikler gut hastalığını tetikleyebilir. Yaptığımız çalışmada diğer çalışmalara benzer şekilde, gut artritli hastalarda CKD prevalansı yüksek oranda saptandı.

Anahtar kelimeler; Gut artriti, ürik asit, kronik böbrek hastalığı

Received Date: 28.12.2018 Accepted Date: 26.03.2019 Published Date:24.04.2019

* Coresponding Authors: Can huzmeli. Kahramanmaras Necip Fazil City Hospital, Department of Nephrology, Kahramanmaras, Turkey.Phone:+905067159443, mail: chuzmeli@hotmail.com

ORCİD:0000-0002-5455-4886

Introduction

Gout arthritis is common in the population.The prevalence of gout arthritis varies according to the geographical region. In studies conducted, the prevalence of gout arthritis was 3.9% in United States, 2.4% in England, %6,8 in Australian and 0.51% in Japan [1-4]. Gout arthritis is the most common form of inflammatory arthropathy. Gout is a disease characterized by arthritis, hyperuricemia, recurrent attacks of arthritis, and accumulation of monosodium urate crystals (MSU) in the joint and soft tissues. Attacks of gout arthritis are usually present in the form of lower extremities mono and oligoarthritis, which rarely develops on the upper extremity and polyarticular. Gout arthritis is a chronic disorder of crystal accumulation. MSU can cause chronic arthritis, tophus, urolithiasis and renal disease, as well as recurrent acute arthritis and bursitis. Gout arthritis and tophus cause chronic disability and deterioration of health-related quality of life [5-7].

Chronic kidney disease (CKD) is common. The prevalence of CKD in the world is found as 8-13%. In our country, the prevalence of CKD was found to be 15.7% in Chronic Renal Disease In Turkey study. CKD was defined based on the presence of kidney damage (structural abnormalities or functional abnormalities other than decreased glomerular filtration rate) or glomerular filtration rate <60 ml/min per 1.73 m2 for ≥3 months [6-7]. Two-thirds of human uric acid excretion occurs through the kidneys, and the remaining one-third occurs through the gastrointestinal tract. It is associated with impaired kidney function and hyperuricemia. In addition, hyperuricemia has been associated with progression and development of CKD and hypertension in studies conducted [8].

Methods

A total of 162 patients diagnosed with Gout arthritis were included in the study. The study was retrospectively conducted between April 2014 and July 2017. The study was conducted in Kahramanmaras and Sivas. Patients were included for gout disease by scanning from the International Classification of Diseases code. Patients' ages, sex, glucose, serum creatinine, serum uric acid level, and additional diagnoses were recorded. Patients with asymptomatic hyperuricemia and patients under the age of 18 were not included in the study. Biochemical values of the patients (blood urea nitrogen, creatinine, and uric acid), C - reactive protein, sedimentation and hemogram were studied. The additional diagnoses of the patients and the treatments they received were noted. Glomerular filtration rate was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) and Modification of Renal Disease (MDRD). Glomerular filtration rateis known as ≥90ml / min is known as stage 1, stage 2 at 60-89 ml / min, stage 3 at 30-59 ml / min, stage 4 at 15-29 ml / min and stage 5 at <15 ml / min. Compliance with Ethical Standards Ethical approval: The Ethics Committee of Cumhuriyet University, Faculty of Medicine approved the present study.

Statistics: For statistical analysis, Windows 20.0 Statistical Package for Social Sciences (SPSS) will be used. Student t test (mean ± standard deviation) for normal distribution data, Mann Whitney U test (median (minimum-maximum) for non-normal distribution groups, square analysis for nominal data were used. Nonparametric correlation test was used for correlation between uric acid and other parameters. ≤0.05 p was considered statistically significant.

Results

A total of 162 patients diagnosed with gout arthritis were included in the study. 135 of these patients were male and 27 were female. The average uric acid levels of the patients were found to be 9.07 ± 1.75 mg / dL (4-14.7). The demographic and laboratory findings of the patients were presented in table 1. There was a statistically significant relationship between uric acid level and age, creatinine, blood urea nitrogen and leucocyte counts (p <0,05).

Glomerular filtration rate was 69.15 ± 26.12 ml / min (11-130) with MDRD and 68.93 ± 26.50 ml / min (11-130) with CKD EPI. There was no significant difference between the two. When glomerular filtration rate was calculated by both CKD EPI and MDRD, CKD stage 3-5 was detected in 39.5% of the patients. When glomerular filtration rate was calculated by MDRD, 33,3% of patients were stage 2 CKD, 34% were stage 3 CKD, 4,9 % were stage 4 CKD, 0,6 % were stage 5 CKD. When glomerular filtration rate was calculated by CKD EPI,

34.6% of patients were stage 2 CKD, 35.8% were stage 3 CKD, 2.5% were stage 4 CKD, 1.2% were stage 5 CKD. There was a statistically significant correlation between uric acid increase and glomerular filtration rate (p <0.05). Uric acid level was 8.63 mg/dL in CKD stage 2 and 9.76 mg / dL in stage 3 patients. CKD stage 3 uric acid were statistically significantly higher.

Table 1 Demographic and laboratory values

		Average ± standard deviation
Age		59,64±14,54 (18-93)
Gender	Male	135
	Female	27
glucose (mg/dl)		108,32±30,11(62-275)
creatinine (mg/dl)		1,25±0,55(0,5-5,2)
BUN (mg/dl)		21,78±14,07(6-92)
Uric acid (mg/dl)		9,07±1,75(4-14,7)
CRP (mg/L)		18,47±24,66(3-143)
Sedimentation rate (min/hr)		21,96±16,61(2-87)
Leukocyte		8,77±2,34(4,5-16)
Haemoglobin		14,41±1,54(10-20)

When the additional diseases of the patients were evaluated, there were cases of 26.5% isolated hypertension, 4,9% isolated diabetes mellitus (DM), 1,9% coronary artery disease (CAD), 9,3% hypertension and DM, 1% hypertension, DM, CAD, 1.2% DM and CAD. In our study, heart failure was not detected in patients with gout arthritis. Twenty-two of the 85 ultrasound patients had nephrolithiasis. Atrophy in a kidney was detected in 5.5% of these patients. 119 patients with complete urine analysis had proteinuria of 8.8% and hematuria of 6.0%. For the treatment of hypertension or CAD, the use of diuretics (8% furasemide and 15.4% thiazide use) was found in 22.2% of the patients. For gout arthritis treatment, 41.4% of dietician consultations were requested for gout arthritis diet, the use of colchicine was found as 92%, nonsteroidal anti-inflammatory drugs was found as 37.7%, and corticosteroids was found as 19.8%.

Discussion

Normal serum uric acid level is known as 3-7mg / dl. Hyperuricemia serum uric acid level is defined as > 7mg / dl in males and > 6mg / dl in females. Continuous hyperuricemia increases the risk of gout arthritis. In a study conducted in Taiwan, a total of 223 individuals with hyperuricemia were followed for 5 years. During this period, a new gout was found in 18.83% [11]. In a study conducted in France, uric acid level <6 mg/dl gout frequency was found to be 1.3%, while in the case of 6-7,9 mg / dl it was 3.2% and > 8 mg / dl was 17.6% [12].

As renal function decreases, uric acid excretion decreases and as a result hyperuricemia develops.In the study conducted, the prevalence of gout arthritis was 2.9% in patients with normal renal function, while 24% was found in patients with glomerular filtration rate <60ml /min. As a result, it was emphasized that the prevalence of hyperuricemia and gout arthritis increased as the renal impairment increased in this study. In addition, in the presence of severe renal impairment, the prevalence of gout arthritis was found to be 6 fold and the prevalence of hyperuricemia 20 fold [13]. In another study, the incidence of gout was detected 2-3% in non-CKD, 4% in CKD stage 1, 6-10% in CKD stage 2, 11-13% in CKD stage 3, approximately 30 % in CKD stage 4 [14]. A total of 493 (169 female, 324 male) dialysis patients were enrolled in the study, the incidence of gout arthritis more than 2 years before dialysis was 4.1% in women and 15.4% in men. The incidence of gout arthritis less than 2 years before dialysis was 0.6% in women and 7.7% in men. No cases were found in women with gout arthritis after dialysis for males, 3.4% for the first two years and 1.2% for the first two years [15].

In a study conducted, 20% of individuals with gout arthritis had CKD stage 3-5, 5% of non-gout subjects had CKD stage 3-5. It was found that 15% of patients with hyperuricemia had CKD stage 3-5 and 3% of patients with non-hyperuricemia had CKD stage 3-5 [16]. In another study, 39% CKD (eGRF <90 ml / min) was detected in patients with gout arthritis [17]. A recent study found that the risk of CKD in gout arthritis patients was asso-ciated with a 3-fold increase [18]. In our study, CKD stage 3-5 was found to be 39.5%. In addition, when glomerular flilltration rate was assessed with CKD EPI, CKD stage 2 was found to be 34.6%, CKD stage 3 35.8%, CKD stage 4 2.5% and CKD stage 5 1.2%.

Co-morbidities are an important condition in patients with gout arthritis. Cardiovascular disease,

DM, hypertension, obesity and CKD are common in patients with gout arthritis. In the study conducted, hypertension frequency was found to be 67.9% in women with gout arthritis and 11.9% in women without gout arthritis, was found to be 54.4% in man with gout arthritis and 10.5% in man without gout arthritis, DM and insu-lin resistance frequency was found to be 28,1%in women with gout arthritis and 3.3% in women without gouty arthritis, was found to be 19.9% in man with gout arthritis and 4.61% in man without gout arthritis, the frequency of chronic heart failure was found to be 25,4% in women with gout arthritis and 1.21% in women without gouty arthritis, was found to be 14.9% in man with gout arthritis and 1.28% in man without gout arthritis. In another study, hypertension frequency was found to be 58.4% in individuals with gout arthritis and 33% in individuals without gout arthritis, DM frequency was found to be 18.6% in individuals with gout arthritis and 9,2% in individuals without gout arthritis, ischemic heart disease frequency was found to be 26.6% in individuals with gout arthritis and 13,5% in individuals without gout arthritis [19-21]. In our study, hypertension was frequently found in individuals with gouty arthritis.

Kidney stones in cases of gout arthritis is correlated with hyperuricemia, hyperuricosuria and low urinary pH. Lower urinary pH in gout arthritis can increase the frequency of both uric acid and other type stones. In studies conducted, the prevalence of nephrolithiasis in patients with gout arthritis ranged from 8.8% to 14% [22-23].

Gout arthritis treatment consists of lifestyle modification, diet and pharmacological treatment. Suggestions should be made about the lifestyle of everyone who gets caught in gout disease. Weight loss should be avoided in excess of meat and seafood, heavy meals, sugary drinks, alcohol (especially beer and alcoholic beverages). Regular exercise should be recommended. Nonsteroidal anti-inflammatory drugs (NSAID), colchicine, prednisone, and interleukin-1 antagonist are recommended in gout treatment. Colchicine and steroid therapy may be used as an alternative because NSAID use in CKD is objectionable. Urate lowering therapy should be started, after the benefit of the treatment and the damage should be assessed with the patient if 2 or more attacks per year, gout-associated tophus, CKD (CKD stage ≥2 for American College of Rheumatology and stage ≥3 for European League Against Rheumatism) and urolithiasis are present. Besides, in cases European League Against Rheumatism young patient (under 40 years), high serum uric acid levels (> 8mg / dl, cardiovascular comorbidities (Hypertension, ischemic heart disease, heart failure) urate lowering therapy should be considered. Both American College of Rheumatology and European League Against Rheumatism target uric acid levels are recommended to be <6 mg / dl, <5 mg / dl in the presence of tophus [24-27]. The first recommended drug in urate lowering therapy is allopurinol. In patients contraindicated to allopurinol, febuxostat is recommended [28].

As a result, individuals with gout arthritis were found to have a high prevalence of CKD. There was a significant relationship between uric acid highness and CKD stage 3. It was found that the rate of glomerular filtration rate decreased significantly with increasing uric acid level. This suggests that the highness of uric acid may increase the CKD progression. It also causes progression of CKD in comorbidities associated with gout arthritis.

Conflict of interests: The authors declare that there is no conflict of interests

Funding sources: There is no source of funding or financial interest in this study.

REFERENCES

1. Kuo CF, Grainge MJ, Mallen C, et al. Eligibility for and prescrip¬tion of urate lowering treatment in patients with incident gout in England. JAMA. 2014; 312: 2684-6. PMID: 25536262
2. 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. Arthritis Rheum. 2011;63:3136–41. PMID: 21800283
3. Kawasaki T, Shichikawa K. Epidemiology survey of gout using residents' health checks. Gout Nucleic Acid Metabo, 2006;30:66.
4. Pisaniello HL, Lester S, Gonzalez-Chia D et al. Gout prevalence and predictors of urate-lowering therapy use: results from a popula¬tion-based study. Research & Therapy 2018;20:143. PMID: 29996922
5. Chuang SY, Chen JH, Yeh WT, Wu CC, Pan WH. Hyperuricemia and increased risk of ischemic heart disease in a large Chinese co¬hort. Int J Cardiol. 2012;154(3):316–21. PMID: 21862159
6. Chandratre P, Roddy E, Clarson L et al. Health related quality of life in gout: a systematic review. Rheumatology (Oxford). 2013;52(11):2031-40. PMID: 23934311
7. Punzi L, Scanu A, Spinella P, Galozzi P, Oliviero F. One year in review 2018: gout. Clin Exp Rheumatol. 2019;37(1):1-11. PMID: 30620275
8. Delanaye T, Glassock RJ, De Broe ME. Epidemiology of chron¬ic kidney disease:think (at least) twice Clinical Kidney Journal. 2017;10(3): 370–4. https://doi.org/10.1093/ckj/sfw154
9. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease Kidney International Supplements. 2013;3: 91–111. doi:10.1038/kisup.2012.67

10. Johnson RJ. Why focus on uric acid? Curr Med Res Opin.2015;31(suppl 2):3-7. PMID: 26414730
11. Lin KC, Lin H Y & Chou P. The interaction between uric acid level and other risk factors on the development of gout among asymptomatic hyperuricemic men in a prospective study. J. Rheumatol. 2000;27:1501–5. PMID: 10852278
12. Zalokar J, Lellouch J, Claude JR. & Kuntz D. Serum uric acid in 23,923 men and gout in a subsample of 4,257 men in France. J Chronic Dis. 1972;25,:305–12. PMID: 4639928
13. Krishnan E. Reduced glomerular function and prevalence of gout: NHANES 2009–10. PLoS One. 2012;7:e50046. PMID: 23209642
14. Juraschek SP, Kovell LC, Miller ER 3rd, Gelber AC. Association of kidney disease with prevalent gout in the United States in 1988–1994 and 2007–2010.Semin Arthritis Rheum. 2013;42(6):551-61. PMID: 23312548
15. Ohno I, Ichida K, Okabe H, Hikita M, Uetake D, Kimura H, Saikawa H, Hosoya T. Frequency of gouty arthritis in patients with endstage renal disease in Japan. Intern Med. 2005;44(7):706-9. PMID: 16093591
16. Zhu Y, Pandya BJ, Choi HK. Comorbidities of gout and hyperuricemia in the US general population:NHANES 2007-2008. Am J Med. 2012;125:679-687e1. PMID: 22626509
17. Fuldeore MJ, Riedel AA, Zarotsky V, Pandya BJ, Dabbous O, Krishnan E Chronic kidney disease in gout in a managed care setting. BMC Nephrol. 2011;12:36. PMID: 21812963
18. Singh JA, Cleveland JD. Gout is associated with a higher risk of chronic renal disease in older adults: a retrospective cohort study of U. S. Medicare population. BMC Nephrol. 2019;20(1):93. PMID: 30876398
19. Wändell P, Carlsson AC, Ljunggren G. Gout and its comorbidities in the total population of Stockholm. Prev Med. 2015;81:387-91. PMID: 26500085
20. Roughley MJ, Belcher J, Mallen CD, & Roddy E. Gout and risk of chronic kidney disease and nephrolithiasis: meta-analysis of observational studies. Arthritis Res Ther. 2015;17:90. PMID: 25889144
21. Robinson PC. Gout- An update of aetiology, genetics, co-morbidities and management. Maturitas. 2018;118:67-73. PMID: 30415758
22. Scales C, Smith A, Hanley J, Saigal C. Prevalence of kidney stones in the United States. Eur Urol. 2012;62:160–5.
23. Pascart T, Richette P. Current and future therapies for gout. Expert Opin Pharmacother. 2017;18(12):1201-11. PMID: 28689430
24. Richette P, Doherty M, Pascual E, et al. 2016 updated EULAR evidence-based recommendations for the management of gout. Ann Rheum Dis. 2017;76(1):29–42. PMID: 27457514
25. Nuki G, Doherty M, Richette P. Current management of gout: practical messages from 2016 EULAR guidelines. Pol Arch Intern Med. 2017;127(4):267-77. PMID: 28430170
26. Slot O. Gout in a rheumatology clinic: results of EULAR/ACR guidelines-compliant treatment. Scand J Rheumatol. 2018;47(3): 194-7. PMID: 28891365
27. Bardin T, Richette P. The role of febuxostat in gout. Curr Opin Rheumatol. 2019;31(2): 152-8. PMID: 30601228

How to cite this article/Bu makaleye atıf için:
Hüzmeli C, Timucin M, Güllü M, Öztürk K, Yetimoğlu E, Candan F. Prevalence of Chronic Kidney Disease and Hyperuricemia In Gout Arthritis Patients. Acta Medica Alanya 2019;3(1):54-58. DOI:10.30565/medalanya.505058