The Relationship Between Microalbuminuria and Serum Uric Acid Levels in Patients With Type 2 Diabetes Mellitus

Nazire Aladağ,¹ Funda Müşerref Türkmen²

¹Department of Internal Medicine, Kartal Dr. Lütfi Kırdar City Hospital, İstanbul, Türkiye ²Department of Internal Medicine, Haydarpaşa Numune Educational and Research Hospital, İstanbul, Türkiye

> Submitted: 27.11.2023 Revised: 24.01.2024 Accepted: 02.02.2024

Correspondence: Nazire Aladağ, Kartal Dr. Lütfi Kırdar City Hospital, İstanbul, Türkiye

E-mail: drnazirealadag@yahoo.com



Keywords: Albuminuria; diabetic nephropathy; proteinuria; type 2 diabetes mellitus; uric acid.



This work is licensed under a Creative Commons

ABSTRACT

Objective: Increasing evidence suggests that oxidants from uric acid synthesis may cause renal dysfunction and cardiovascular diseases by inducing inflammation and endothelial dysfunction. Variations in the correlations between inflammation and albuminuria due to race and ethnicity have been noted in diabetic patients. However, studies on this topic among Turkish patients with type 2 diabetes remain limited. This study aimed to investigate the relationship between serum uric acid levels and microalbuminuria in patients with type 2 DM.

Methods: This prospective cross-sectional study included 80 patients with T2DM, divided into two groups: 40 with microalbuminuria (30–300 mg in 24-hour urine proteinuria) and 40 without (<30 mg in 24-hour urine proteinuria). Biochemical parameters were assessed through the analysis of venous blood samples, which were collected following a 12-hour fasting period during outpatient evaluations.

Results: The study population had a mean age of 57.8 ± 11.6 years. The mean uric acid levels $(4.7\pm1.5 \text{ vs. } 4.0\pm1.1, \text{ P=0.036})$ and the mean creatinine clearance (114.6 $\pm5.8 \text{ vs. } 98.1\pm3.4$, P=0.050) were higher in the group with microalbuminuria compared to the group without. The 24-hour urinary protein levels were positively correlated with HbA1C (r=0.305, P=0.036), uric acid (r=0.308, P=0.032), and creatinine clearance (r=0.294, P=0.050).

Conclusion: Individuals with T2DM and microalbuminuria tend to display elevated levels of uric acid. Considering the potential effects of increased uric acid levels on diabetic nephropathy, end-stage renal disease, and the development of cardiovascular disease, routine monitoring of uric acid levels in T2DM patients may be important from a prognostic perspective.

INTRODUCTION

Diabetes mellitus (DM), a major health concern, is projected to impact 578 million individuals worldwide by 2030 and escalate to 700 million by 2045.^[1] Diabetes is a significant metabolic disease known for causing macrovascular complications such as cardiovascular disease (CVD) and atherosclerosis, as well as microvascular complications such as nephropathy.^[2] These complications are well-established factors for the increased risk of mortality in patients with type-2 DM (T2DM).^[3,4] Therefore, studies concerning the pathogenesis of diabetes and its complications are gaining increased importance.

Uric acid, a product of purine metabolism, is generated through the enzymatic actions of xanthine oxidase. This process also results in the production of oxidants, which have been implicated in cardiovascular diseases and renal dysfunction.^[5] This can also induce an inflammatory re-

sponse, leading to organ dysfunction. Therefore, in clinical practice, uric acid can serve as a significant marker for predicting elevated levels of oxidative stress.^[6] Oxidative stress can increase reactive oxygen species (ROS), leading to renal vascular endothelial damage and the production of proteinuria.^[7,8] Albuminuria, defined as the presence of an excessive amount of serum protein in the urine. is a significant indicator of kidney damage, including impaired reabsorption and filtration by the kidneys.[9] On the other hand, it has been suggested that race and ethnicity could cause significant variations in the relationship between inflammation and albuminuria in patients with diabetic diseases.^[10] Globally, recent studies in patients with cardiovascular or diabetic diseases have demonstrated a significant association between uric acid levels and albuminuria levels.[11-14] However, studies on this topic among Turkish patients with T2DM remain limited.

The objective of this research was to explore the associ-

86 South. Clin. Ist. Euras.

ation between microalbuminuria and serum uric acid concentrations in patients with T2DM.

MATERIALS AND METHODS

This prospective cross-sectional investigation, carried out at the DM Clinic of HaydarpaşaNumune Training and Research Hospital between January 2006 and May 2007, was in alignment with the Helsinki Declaration and Good Clinical Practice Guidelines. This study is a specialization thesis, it was conducted with institutional approval before 2020, the ethics committee decision was not taken at that time

The study was conducted 80 patients with T2DM, aged between 28 and 77 years, all of whom presented to the DM outpatient clinic. Each patient was undergoing treatment with oral antidiabetic medication. Patients with secondary and type-I DM, suspected pregnancy, and an estimated glomerular filtration rate (eGFR) less than 60 mg/dL/1.173 m² were excluded from the study. Those suspected of pregnancy were tested for beta human chorionic gonadotropin. Patients with hypertension who were using antihypertensive drugs known to affect uric acid levels (such as losartan, various diuretics including hydrochlorothiazide, furosemide, and ethacrynic acid) were also excluded from the study.

Operational definitions

Patients with a significant increase in urine albumin-creatinine ratio (ACR) in the range of 30-299 mg/g of creatinine or with a urine albumin excretion rate (UAE) in the range of 30-299 mg/24 hours were considered to have microal-buminuria.

Study protocol

The demographic and clinical data of all patients were recorded during the outpatient examination. Biochemical parameters were assessed through the analysis of venous blood samples, which were collected following a 12-hour fasting period during outpatient evaluations. The analysis of all samples was conducted in a single laboratory, utilizing a uniform methodology detailed later in the text. Patients with T2DM were categorized into two distinct groups: those with microalbuminuria (n=40) and those without (n=40). Measurements of fat mass and body fat percentage were conducted using the Tanita Body Composition Analyzer TBF-300, following a 30-minute rest period for the subjects.

Biochemical analysis

The Hitachi Modular autoanalyzer (Roche Diagnostics Corp., Indianapolis, IN, USA) was used for the analysis of patients' venous blood and 24-hour urine samples. Levels of 24-hour urine protein (microalbumin turbidimetric method), hemoglobin AIC (HbAIC) (turbidimetric inhibition immunoassay, TINIA), fasting blood glucose (FBG), and lipid parameters (enzymatic colorimetric method) were measured. The determination of low-density lipoprotein cholesterol (LDL-C) was carried out employing

the Friedewald formula.^[15] Creatinine clearance was determined using the Cockcroft-Gault equation. For female patients, the obtained value was adjusted by multiplying by a factor of 0.85. The formula for creatinine clearance (mL/min) is: (140 - age) × weight / (72 × serum creatinine level).^[16]

Statistical Analysis

All data were analyzed with MINITAB RELEASE 14 (Minitab Inc., Pennsylvania, USA). The assessment of normal distribution was conducted through the Anderson-Darling test. Numerical data were represented in terms of mean ± standard deviation. Depending on the distribution's normality, the comparison of numerical data between the two groups was conducted using either the Student's T-test or the Mann-Whitney U test. The correlations among numerical data sets were assessed through Pearson or Spearman correlation analysis, depending on the normality of distribution. Categorical variables were displayed in the form of numbers and percentages, with group comparisons conducted through the Chi-square test. A p-value of less than 0.05 (*) was considered statistically significant for all analyses.

RESULTS

The study population consisted of 80 cases including 49 women (61.3%) and 31 men (38.7%), with a mean age of 57.8±11.6 years. The mean weight was higher in the group with microalbuminuria compared to the group without (89.5±12.0 vs. 75.0±12.6, P=0.002), while the mean BMI was lower (35.1±9.6 vs. 40.0±9.6, P=0.024). The mean high-density lipoprotein cholesterol (HDL-C) was lower in the group with microalbuminuria compared to the group without (40.6±8.6 vs. 47.3±9.3, P=0.001), while the mean uric acid (4.7±1.5 vs. 4.0±1.1, P=0.036) (Figure 1) and the mean creatinine clearance (114.6±5.8 vs. 98.1±3.4, P=0.050) were higher. There were no significant variances in other demographic and clinical findings between the groups with and without microalbuminuria (Table 1).

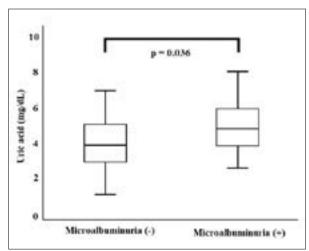


Figure 1. Distribution of uric acid levels in patients with type 2 diabetes mellitus based on the presence of microalbuminuria.

Table I.	Demographic and laborator	y findings of patients w	vith type 2 diabetes mellitus

Variables	Type 2 DM		P-value
	With MA n = 40	Without MA n = 40	
Age, years	57.0±11.8	58.0±12.6	0.833
Gender, n (%)			
Male	16 (40.0)	15 (37.5)	0.820
Female	24 (60.0)	25 (62.5)	
Weight, kg	89.5±12.0	75.0±12.6	0.002*
Height, m	1.7±3.5	1.6±2.8	0.131
Waist circumference, cm	104.1±9.6	99.4±14.0	0.079
BMI, kg/m2	35.1±9.6	40.0±9.6	0.024*
Smoker, n (%)	6 (15.0)	8 (20.0)	0.559
Duration of DM, years	7.8 ±1.0	9.5±1.3	0.279
Hypertension, n (%)	21 (52.5)	18 (45.0)	0.505
Cholesterol, mg/dL	207.0±57.7	196.0±51.0	0.353
LDL-C, mg/dL	122.0±39.8	I I 8.0±37.0	0.634
HDL-C, mg/dL	40.6±8.6	47.3±9.3	0.001*
Triglyceride, mg/dL	229.0±60.3	161.0±40.4	0.197
HbA1C, %	8.5±2.4	10.6±3.0	0.409
Uric acid, mg/dL	4.7±1.5	4.0±1.1	0.036*
Creatinine clearance, mL/min	114.6±5.8	98.1±3.4	0.050*

Data are mean ± standard deviation or median (IQR), or number (%). *p<0.05 indicates statistical significance. Abbreviations: BMI: Body mass index; DM: Diabetes mellitus; HbA1C: Hemoglobin A1C; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; MA: Microalbuminuria.

Table 2. Parameters associated with 24-hour urinary protein levels in patients with type 2 diabetes mellitus

Variables	r	P-value
Age	0.102	0.862
Weight	0.258	0.301
Waist circumference	0.271	0.109
BMI	0.282	0.126
Duration of DM	0.125	0.354
Cholesterol	0.233	0.422
LDL-C	0.105	0.711
HDL-C	-0.318	0.011*
Triglyceride	0.251	0.258
HbAIC	-0.305	0.036*
Uric acid	0.308	0.032*
Creatinine clearance	0.294	0.050*

*p<0.05 indicates statistical significance. Abbreviations: BMI: Body mass index; DM: Diabetes mellitus; HbA1C: Hemoglobin A1C; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol.

The 24-hour urinary protein levels were positively correlated with HbA1C (r=0.305, P=0.036), uric acid (r=0.308, P=0.032), and creatinine clearance (r=0.294, P=0.050), whereas they showed a negative correlation with HDL-C levels (r=-0.318, P=0.011) (Table 2).

DISCUSSION

Even with the latest advancements in the treatment of diabetes, nephropathy continues to be a leading cause of end-stage renal disease. Increasing evidence indicates that oxidants generated in the process of uric acid synthesis contribute to the pathogenesis of renal dysfunction and the development of cardiovascular diseases by triggering inflammation and leading to endothelial dysfunction. [17] Moreover, studies propose that endothelial dysfunction resulting from hyperuricemia promotes the proliferation of afferent vascular smooth muscle cells and diminishes renal perfusion. This suggests that increased levels of uric acid may play a crucial role in the progression of renal damage. [18]

Observations indicated that patients with microalbuminuria had increased uric acid levels. A study by Behradmanesh et al.^[19] suggested that higher levels of serum uric acid might significantly contribute to the development of diabetic nephropathy. In another study conducted in patients with T2DM, a positive correlation was reported between serum uric acid concentration and urinary albumin excretion.^[20] A study involving healthy Korean men demonstrated that elevated serum uric acid levels independently increased the risk of microalbuminuria development over a period of five years.^[21] Studies have shown that even mild hyperuricemia has an independent relationship with microalbumin.^[22] Furthermore, in patients with T2DM who

88 South. Clin. Ist. Euras.

have preserved renal function, an elevation in serum uric acid levels by one standard deviation is associated with a 21% increased risk of initiating chronic kidney disease.[23] These findings are supported by a meta-analysis involving patients with T2DM, which also revealed a link between high uric acid levels and an increased risk of diabetic kidney disease.[24] On the other hand, a Japanese two-year cohort study indicated that higher levels of serum uric acid in diabetic patients correlate with a greater risk of diabetic nephropathy worsening, characterized by a transition from microalbuminuria to albuminuria. However, the study also revealed no association between uric acid levels and the probability of progression either from normoalbuminuria to microalbuminuria or from microalbuminuria to albuminuria.[25] An Italian 4-year cohort study identified a significant correlation between the levels of serum uric acid and the onset risk of albuminuria, specifically in instances where the estimated GFR was under 60 mL/min per 1.73 m^2.[26] These differences between studies may be attributable to potential variations in patients' age ranges, follow-up durations, race, and ethnic background.[27]

A significant finding of this study was the negative correlation between serum uric acid and HDL-C levels. Several studies have demonstrated a significant relationship between serum uric acid and the lipid profile, particularly indicating a negative correlation with HDL.[28-30] These findings are also consistent with previous studies that indicate an overlap in the pathogenesis between hyperuricemia and dyslipidemia.[31,32] Furthermore, studies have indicated that in patients with T2DM, higher HDL-C levels are associated with a lower likelihood of microalbuminuria.[33,34] Similarly, in this study, HDL-C levels were found to be lower in T2DM patients with microalbuminuria. The hypothesized mechanisms by which HDL-C contributes to the pathogenesis of microalbuminuria encompass the facilitation of glomerular sclerosis and renal tubular interstitial damage, in addition to the exacerbation of systemic oxidative stress consequent to augmented oxidized LDL concentrations.[35,36]

Numerous studies have indicated that HbAIC is an independent predictor in the development of microalbuminuria [37-39] In this study, the study observed a tendency towards lower HbAIC levels in T2DM patients with microalbuminuria. This may be associated with the elevated levels of uric acid observed in these patients. Previous studies have indicated that there is a negative correlation between serum uric acid and HbAIC levels. [40,41] The inverse transport of uric acid and glucose in the renal tubules could be responsible for these correlations. [40]

There are notable limitations to this study. Firstly, the study was single-center with a small sample size. Secondly, the collection of uric acid levels was limited to outpatient examinations, and there was no longitudinal monitoring of uric acid levels. Therefore, the influence of uric acid on the development of diabetic nephropathy over an extended period could not be evaluated in this study. Thirdly, medications including urate-lowering, anti-diabetic, and antihypertensive drugs were not accounted for. This might limit

the generalizability of the study.

Conclusion

In patients with T2DM, a positive correlation exists between levels of uric acid and 24-hour urinary protein. Individuals with T2DM and microalbuminuria tend to display elevated levels of uric acid. Considering the potential effects of increased uric acid levels on diabetic nephropathy, end-stage renal failure, and the development of cardiovascular disease, routine monitoring of uric acid levels in T2DM patients may be important from a prognostic perspective.

Informed Consent

Retrospective study.

Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept: N.A., F.M.T.; Design: N.A.; Supervision: N.A.; Fundings: N.A.; Materials: N.A.; Data: N.A.; Analysis: N.A.; Literature search: N.A.; Writing: N.A.; Critical revision: N.A., F.M.T.

Conflict of Interest

None declared.

REFERENCES

- Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. Diabetes Res Clin Pract 2019;157:107843. [CrossRef]
- Chawla A, Chawla R, Jaggi S. Microvasular and macrovascular complications in diabetes mellitus: Distinct or continuum? Indian J Endocrinol Metab 2016;20:546–51. [CrossRef]
- Hirakawa Y, Ninomiya T, Kiyohara Y, Murakami Y, Saitoh S, Nakagawa H, et al. Age-specific impact of diabetes mellitus on the risk of cardiovascular mortality: An overview from the evidence for Cardiovascular Prevention from Observational Cohorts in the Japan Research Group (EPOCH-JAPAN). J Epidemiol 2017;27:123–9. [CrossRef]
- Salinero-Fort MÁ, San Andrés-Rebollo FJ, de Burgos-Lunar C, Abánades-Herranz JC, Carrillo-de-Santa-Pau E, Chico-Moraleja RM, et al. Cardiovascular and all-cause mortality in patients with type 2 diabetes mellitus in the MADIABETES Cohort Study: Association with chronic kidney disease. J Diabetes Complications 2016;30:227–36. [CrossRef]
- Sekizuka H. Uric acid, xanthine oxidase, and vascular damage: Potential of xanthine oxidoreductase inhibitors to prevent cardiovascular diseases. Hypertens Res 2022;45:772–4. [CrossRef]
- Kurajoh M, Fukumoto S, Yoshida S, Akari S, Murase T, Nakamura T, et al. Uric acid shown to contribute to increased oxidative stress level independent of xanthine oxidoreductase activity in MedCity21 health examination registry. Sci Rep 2021;11:7378. [CrossRef]
- Duni A, Liakopoulos V, Roumeliotis S, Peschos D, Dounousi E.
 Oxidative stress in the pathogenesis and evolution of chronic kidney disease: Untangling Ariadne's thread. Int J Mol Sci 2019;20:3711.

 [CrossRef]
- 8. Schei J, Fuskevåg OM, Stefansson VTN, Solbu MD, Jenssen TG,

- Eriksen BO, et al. Urinary markers of oxidative stress are associated with albuminuria but not GFR decline. Kidney Int Rep 2018;3:573–82. [CrossRef]
- Gonzalez Suarez ML, Thomas DB, Barisoni L, Fornoni A. Diabetic nephropathy: Is it time yet for routine kidney biopsy? World J Diabetes 2013;4:245–55. [CrossRef]
- Sinha SK, Shaheen M, Rajavashisth TB, Pan D, Norris KC, Nicholas SB. Association of race/ethnicity, inflammation, and albuminuria in patients with diabetes and early chronic kidney disease. Diabetes Care 2014;37:1060–8. [CrossRef]
- Lai YJ, Chen YY, Ku PW, Chen LJ, Yen YF. Association between uric acid level and incidence of albuminuria in patients with type 2 diabetes mellitus: A 4.5-year cohort study. Medicine Baltimore 2021;100:e27496. [CrossRef]
- Kocak MZ, Aktas G, Duman TT, Atak BM, Savli H. Is uric acid elevation a random finding or a causative agent of diabetic nephropathy? Rev Assoc Med Bras 2019;65:1155–60. [CrossRef]
- Chuengsamarn S, Rattanamongkolgul S, Jirawatnotai S. Association between serum uric acid level and microalbuminuria to chronic vascular complications in Thai patients with type 2 diabetes. J Diabetes Complications 2014;28:124–9. [CrossRef]
- Latif H, Iqbal A, Rathore R, Butt NF. Correlation between serum uric acid level and microalbuminuria in type-2 diabetic nephropathy. Pak J Med Sci 2017;33:1371–5. [CrossRef]
- Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499–502. [CrossRef]
- Matz R. Cockcroft-Gault equation and estimation of creatinine clearance. Am J Med 2002;112:684. [CrossRef]
- Gherghina ME, Peride I, Tiglis M, Neagu TP, Niculae A, Checherita IA. Uric acid and oxidative stress-relationship with cardiovascular, metabolic, and renal impairment. Int J Mol Sci 2022;23:3188. [Cross-Ref]
- Park JH, Jo YI, Lee JH. Renal effects of uric acid: Hyperuricemia and hypouricemia. Korean J Intern Med 2020;35:1291–304. [CrossRef]
- Behradmanesh S, Horestani MK, Baradaran A, Nasri H. Association of serum uric acid with proteinuria in type 2 diabetic patients. J Res Med Sci 2013;18:44–6. [CrossRef]
- Neupane S, Dubey RK, Gautam N, Agrawal KK, Jayan A, Shrestha S, et al. Association between serum uric acid, urinary albumin excretion, and glycated hemoglobin in Type 2 diabetic patient. Niger Med J 2016;57:119–23. [CrossRef]
- Oh CM, Park SK, Ryoo JH. Serum uric acid level is associated with the development of microalbuminuria in Korean men. Eur J Clin Invest 2014;44:4–12. [CrossRef]
- Viazzi F, Leoncini G, Ratto E, Falqui V, Parodi A, Conti N, et al. Mild hyperuricemia and subclinical renal damage in untreated primary hypertension. Am J Hypertens 2007;20:1276–82. [CrossRef]
- Zoppini G, Targher G, Chonchol M, Ortalda V, Abaterusso C, Pichiri I, et al. Serum uric acid levels and incident chronic kidney disease in patients with type 2 diabetes and preserved kidney function. Diabetes Care 2012;35:99–104. [CrossRef]
- Ji P, Zhu J, Feng J, et al. Serum uric acid levels and diabetic kidney disease in patients with type 2 diabetes mellitus: A dose-response meta-analysis. Prim Care Diabetes 2022;16:457–465. [CrossRef]
- Hayashino Y, Okamura S, Tsujii S, Ishii H. Association of serum uric acid levels with the risk of development or progression of albuminuria among Japanese patients with type 2 diabetes: A prospective cohort

- study [Diabetes Distress and Care Registry at Tenri (DDCRT 10)]. Acta Diabetol. 2016;53:599–607. [CrossRef]
- De Cosmo S, Viazzi F, Pacilli A, Giorda C, Ceriello A, Gentile S, et al. Serum uric acid and risk of CKD in type 2 diabetes. Clin J Am Soc Nephrol 2015;10:1921–9. [CrossRef]
- Bhalla V, Zhao B, Azar KM, Wang EJ, Choi S, Wong EC, et al. Racial/ethnic differences in the prevalence of proteinuric and nonproteinuric diabetic kidney disease. Diabetes Care 2013;36:1215–21. [CrossRef]
- Ali N, Rahman S, Islam S, Haque T, Molla NH, Sumon AH, et al. The relationship between serum uric acid and lipid profile in Bangladeshi adults. BMC Cardiovasc Disord 2019;19:42. [CrossRef]
- Peng TC, Wang CC, Kao TW, Chan JY, Yang YH, Chang YW, et al. Relationship between hyperuricemia and lipid profiles in US adults. Biomed Res Int 2015;2015:127596. [CrossRef]
- Maloberti A, Vanoli J, Finotto A, Bombelli M, Facchetti R, Redon P, et al. Uric acid relationships with lipid profile and adiposity indices: Impact of different hyperuricemic thresholds. J Clin Hypertens Greenwich 2023;25:78–85. [CrossRef]
- Lu W, Song K, Wang Y, Zhang Q, Li W, Jiao H, et al. Relationship between serum uric acid and metabolic syndrome: An analysis by structural equation modeling. J Clin Lipidol 2012;6:159–67. [Cross-Ref]
- Conen D, Wietlisbach V, Bovet P, Shamlaye C, Riesen W, Paccaud F, et al. Prevalence of hyperuricemia and relation of serum uric acid with cardiovascular risk factors in a developing country. BMC Public Health 2004;4:9. [CrossRef]
- Sun X, Xiao Y, Li PM, Ma XY, Sun XJ, Lv WS, et al. Association of serum high-density lipoprotein cholesterol with microalbuminuria in type 2 diabetes patients. Lipids Health Dis 2018;17:229. [CrossRef]
- Zoppini G, Targher G, Chonchol M, Perrone F, Lippi G, Muggeo M. Higher HDL cholesterol levels are associated with a lower incidence of chronic kidney disease in patients with type 2 diabetes. Nutr Metab Cardiovasc Dis 2009;19:580–6. [CrossRef]
- Vaziri ND. Lipotoxicity and impaired high density lipoprotein-mediated reverse cholesterol transport in chronic kidney disease. J Ren Nutr 2010;20:S35–43. [CrossRef]
- Li C, Gu Q. Protective effect of paraoxonase 1 of high-density lipoprotein in type 2 diabetic patients with nephropathy. Nephrology Carlton 2009;14:514–20. [CrossRef]
- Chiu WC, Lai YR, Cheng BC, Huang CC, Chen JF, Lu CH. HbA1C variability is strongly associated with development of macroalbuminuria in normal or microalbuminuria in patients with type 2 diabetes mellitus: A six-year follow-up study. Biomed Res Int 2020;2020:7462158. [CrossRef]
- Lian H, Wu H, Ning J, Lin D, Huang C, Li F, et al. The risk threshold for hemoglobin A1c associated with albuminuria: A population-based study in China. Front Endocrinol Lausanne 2021;12:673976. [Cross-Ref]
- Hsu CC, Chang HY, Huang MC, Hwang SJ, Yang YC, Lee YS, et al. HbA1c variability is associated with microalbuminuria development in type 2 diabetes: A 7-year prospective cohort study. Diabetologia 2012;55:3163–72. [CrossRef]
- Wei F, Chang B, Yang X, Wang Y, Chen L, Li WD. Serum uric acid levels were dynamically coupled with hemoglobin A1c in the development of type 2 diabetes. Sci Rep 2016;6:28549. [CrossRef]
- Cui Y, Bu H, Ma X, Zhao S, Li X, Lu S. The relation between serum uric acid and HbA1c Is dependent upon hyperinsulinemia in patients with newly diagnosed type 2 diabetes mellitus. J Diabetes Res 2016;2016:7184123. [CrossRef]

90 South, Clin, Ist, Euras,

Tip 2 Diyabetli Hastalarda Mikroalbuminüri ile Serum Ürik Asit Seviyeleri Arasındaki İlişki

Amaç: Giderek artan kanıtlar, ürik asit sentezindeki oksidanların, inflamasyonu ve endotel disfonksiyonunu indükleyerek böbrek fonksiyon bozukluğuna ve kardiyovasküler hastalıklara neden olabileceğini düşündürmektedir. Diyabetik hastalarda ırk ve etnik kökene bağlı olarak inflamasyon ve albüminüri korelasyonlarında farklılıklar kaydedilmiştir. Ancak tip 2 diyabetli Türk hastalarda bu konuda yapılan çalışmalar sınırlı kalmaktadır. Bu çalışmada tip 2 DM hastalarında serum ürik asit düzeyleri ile mikroalbüminüri arasındaki ilişkinin araştırılması amaçlandı

Gereç ve Yöntem: Bu prospektif kesitsel çalışmaya T2DM'li 80 hasta dahil edildi ve iki gruba ayrıldı: mikroalbüminürisi olan 40 hasta (24 saatlik idrar proteinürisinde 30-300 mg) ve olmayan 40 hasta (24 saatlik idrar proteinürisinde <30 mg). 12 saatlik açlık sonrası hastalardan alınan venöz kan örneklerinin analizi ile biyokimyasal parametreler değerlendirildi.

Bulgular: Çalışma popülasyonunun yaş ortalaması 57.8±11.6 yıldı. Ortalama ürik asit (4.7±1.5 vs. 4.0±1.1, P=0.036) ve ortalama kreatinin klirensi (114.6±5.8 vs. 98.1±3.4, P=0,050) mikroalbüminürisi olmayan gruba göre mikroalbüminürisi olan grupta daha yüksekti. 24 saatlik idrar protein düzeyleri HbA1C (r=0.305, P=0.036), ürik asit (r=0.308, P=0.032) ve kreatinin klirensi (r= 0,294, P=0.050) ile pozitif korelasyon gösterdi.

Sonuç: Mikroalbuminürisi olan Tip2 DM hastaların ürik asit seviyesi daha yüksek değerdeydi. Artan ürik asit düzeylerinin diyabetik nefropati, son dönem böbrek yetmezliği ve kardiyovasküler hastalık gelişimi üzerindeki potansiyel etkileri göz önüne alındığında, T2DM hastalarında ürik asit düzeylerinin rutin olarak izlenmesi prognostik açıdan önemli olabilir.

Anahtar Sözcükler: Albuminüri; diyabetik nefropati; proteinüri; ürik asit; tip 2 diyabetes mellitus.