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Relationship between uric acid albumin ratio and peripheral artery disease complexity

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Abstract. – OBJECTIVE: Uric acid to albumin ratio (UAR) reflects inflammatory activity and its predictive value has been shown in various diseases. In this study, we sought to evaluate the value of uric acid to albumin ratio in patients with peripheral arterial disease (PAD).

PATIENTS AND METHODS: Two hundred forty-three PAD patients were divided into Trans-Atlantic Inter-Society Consensus-II (TASC-II) A-B and TASC-II C-D groups, according to their TASC-II classification. Biochemical variables of the patients were recorded, and the UAR of each patient was calculated.

RESULTS: Patients who had TASC-II A-B disease were younger than the patients who had TASC-II C-D disease (60.7±8.71 vs. 63.28±8.8 years, p=0.024). Quade ANCOVA results showed that patients with TASC-II C-D disease had higher values of UAR when age was used as a covariate (t=-5.045, p<0.001). Lymphocyte count was significantly lower, and UAR was significantly higher in patients with TASC-II C-D disease (p=0.035 and p<0.001, respectively). UAR and lymphocyte count showed a significant positive correlation and a negative correlation with the TASC-II class of the disease (r=0.403, and r=-0.299, p<0.001 for both). A UAR of 1.54 predicted TASC-II C-D disease with a sensitivity and specificity of 57.9% and 78.8%, respectively. UAR predicted severe PAD with an OR of 3.723.

CONCLUSIONS: UAR was a better tool for predicting TASC-II C-D disease compared to uric acid and albumin levels. UAR is an easily calculable parameter that can be used in clinical practice.

Key Words:
Uric acid, Albumin, Peripheral arterial disease, Ratio.

Introduction

Lower extremity peripheral arterial disease (PAD), a growing health burden caused by the combination of aging and the increasing prevalence of risk factors, has become an issue that needs to be addressed[1]. It has been estimated that approximately 6% of the global population lives with PAD. PAD has a long asymptomatic period, which contributes to difficulties in its detection and treatment. Atherosclerosis is complicit in the pathogenesis of PAD, which leads to complications in other arterial beds, including coronary and cerebrovascular circulation[2]. Patients with PAD have been reported[3] to have up to 6-times increase in cardiovascular morbidity and mortality. Besides sharing traditional risk factors for coronary artery disease (CAD), nontraditional risk factors and various inflammatory markers have also been found[4] to be related to PAD. To date, several studies[5] that have focused on the role of inflammation and the value of inflammatory markers in PAD demonstrated that patients with PAD have increased levels of inflammatory cytokines and markers such as interleukin family, selectins, C-reactive protein, and fibrinogen. Rein et al[6] showed that inflammatory activity in PAD patients was higher than that of coronary artery disease patients. Although the mechanism of intermittent claudication has been classically attributed to critical occlusion in the arteries that supply lower extremities, it might also be affected by factors related to inflammation[7].

Uric acid, the final product of purine catabolism, has been shown[8] to be related to cardiovascular mortality even within the reference values. Although uric acid is associated with traditional risk factors and metabolic syndrome, it is not exactly known whether it has a causal relation with atherosclerosis or plays a role as an intermediary substance for the effects of these risk factors[9]. Activation of the renin-angiotensin system, induction of secretion of pro-inflammatory cytokines, and impairment of endothelial func-

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tions are some of the suggested mechanisms for the adverse effects of uric acid. The net result of these biological alterations is a pathological condition that is represented by chronic low-grade inflammation[10]. Uric acid has been found[11] to be an independent predictor for renal disease in diabetic patients, which can promote the progression and deterioration of renal disease in this group of patients.

Although historically, it has been thought[12] to reflect the nutritional status of the patients, serum albumin level is affected by the inflammatory condition of the body. Serum albumin levels usually decrease in inflammatory diseases, and it can be considered a negative acute phase reactant. It has been suggested[13] that compared to a single parameter, a combination of uric acid and albumin may produce better results in evaluating inflammatory activity. The predictive value of uric acid to albumin ratio (UAR) has been shown[13-15] in acute coronary syndrome, acute kidney injury, and the development of atrial fibrillation. To the best of our knowledge, the value of UAR has not been studied in PAD patients. As such, we investigated UAR in PAD patients and tried to find whether it has a predictive value for the presence of the severity of PAD.

Patients and Methods

This was a retrospective cross-sectional study that involved patients diagnosed with PAD in a tertiary hospital. Diagnosis of PAD was made by peripheral angiography. For this purpose, 243 peripheral angiograms, which were performed from January 2018 to January 2022, were re-evaluated. Patients with acute coronary syndrome, non-critical PAD, advanced kidney failure, infectious, inflammatory diseases, previous peripheral stent implantation, and/or bypass operation were excluded. After the application of exclusion criteria, 243 patients were enrolled in the study. Clinical and demographic features of the study population were obtained from the hospital data system. All patients gave informed consent, and the Local Ethical Committee approved the study protocol. The study was conducted in compliance with the ethical principles of the Helsinki Declaration.

Patients were considered hypertensive if they used antihypertensive medications or their blood pressure was greater than 140 and/or 90 mmHg. Diagnosis of diabetes mellitus (DM) was made if the patients used anti-diabetic drugs or their fasting glucose levels were greater than 125 mg/dL. Advanced kidney failure was defined as a glomerular filtration rate lower than 30 mL/min/1.73 m^2. Hyperlipidemia (HL) was defined as fasting total cholesterol (TC) >200 mg/dL or low-density lipoprotein cholesterol (LDL-C) >130 mg/dL or lipid-lowering chronic use of drugs. CAD was defined as the presence of more than 50% stenosis at least in one of the coronary arteries and a history of percutaneous coronary and/or coronary artery bypass operation intervention. After 12 hours of fasting in the sitting position, the blood samples of the patients were collected in tubes containing trisodium citrate (0.109 μM). The collected samples were analyzed for biochemical variables and complete blood count parameters.

Peripheral angiographies of the patients were performed using the Siemens Axiom Artis Zee Cath Lab (Munich, Germany) system. Right common femoral arterial access was preferred, and a 6F pigtail was inserted into the arterial system with the Judkins technique. The catheter was positioned just above the aortic bifurcation, and with the use of a pump injector, angiographic images of the peripheral arteries were taken. Classification of the peripheral arterial lesions was made by using the TransAtlantic Inter-Society Consensus-II (TASC-II) system[3]. Patients were divided into two groups according to their TASC-II classes. Patients who had TASC-II A and TASC-II B type lesions constituted Group 1, whereas patients who had TASC-II C and TASC-II D type lesions constituted Group 2.

Statistical Analysis

The normality assessment of the data was made by assessing the skewness and kurtosis of the data and by use of the Kolmogorov-Smirnov test. Comparisons of the TASC-II A-B group with the TASC-II C-D group were made by using the student *t*-test or Mann-Whitney U test, according to the distribution of the data. The correlation of UAR with the TASC-II group of disease was made by using Spearman's correlation analysis. ROC curve analysis was conducted in order to find the value of UAR that predicted TASC-II C-D disease. Univariate logistic regression was done in order to find predictors of TASC-II C-D disease. Parameters that were found to be meaningful in univariate analysis were put into multivariate logistic regression analysis. Quade ANCOVA was conducted where TASC-II C-D disease and age were accepted as dependent variables and covari-

ates, respectively. All the statistical analyses were done using SPSS version 23.0 (IBM Corp., Armonk, NY, USA), and a *p*-value lower than 0.05 was considered significant.

Results

Patients who had TASC-II A-B disease were younger than the patients who had TASC-IIC-D disease (60.7±8.71 *vs.* 63.28±8.8 years, $p=0.024$). Quade ANCOVA results showed that patients with TASC-II C-D disease had higher values of UAR when age was used as a covariate (t=-5.045, $p<0.001$). We did not find any differences with respect to gender, prevalence of hypertension (HT), DM, cerebrovascular disease, or atrial fibrillation between the two groups of patients. TASC-II C-D patients had a significantly higher rate of CAD compared to that of TASC-II A-B patients. Biochemical variables – except for lymphocyte count and UAR – did not differ between the two groups. Lymphocyte count was significantly lower, and UAR was significantly higher in patients with TASC-II C-D disease ($p=0.035$ and $p<0.001$, respectively). Table I shows the clinical and biochemical parameters of the two groups. UAR and lymphocyte count showed a significant positive correlation and a negative correlation with the TASC-II class of the disease (r=0.403 and r=-0.299, $p<0.001$ for both). ROC curve analysis results showed that a UAR of 1.54 predicted TASC-II C-D disease with a sensitivity and specificity of 57.9% and 78.8%, respectively (AUC: 0.702, $p<0.001$, 95% CI: 0.631-0.774) (Figure 1). Results of ROC curve analyses of uric acid, albumin, and UAR and paired comparisons of ROC curves are shown in Tables II and III, respectively. UAR was a better tool for predicting TASC-II C-D disease compared to uric acid and albumin levels.

Results of univariate binary logistic regression analysis showed that age, presence of CAD, levels of hemoglobin, uric acid, albumin and UAR were the independent predictors of TASC-II C-D disease (Table IV). Parameters that showed predictive value in univariate logistic regression were put into multivariate logistic regression analysis (Table V). We conducted two models of multivariate logistic regression analysis. In model A, uric acid and albumin were put into the model, whereas in model B, UAR was put into the model. Results of the models showed that compared to uric acid and albumin, UAR predicted severe PAD with an OR of 3.723.

Discussion

Our study showed that patients with severe PAD had higher UAR. Moreover, UAR had a pos-

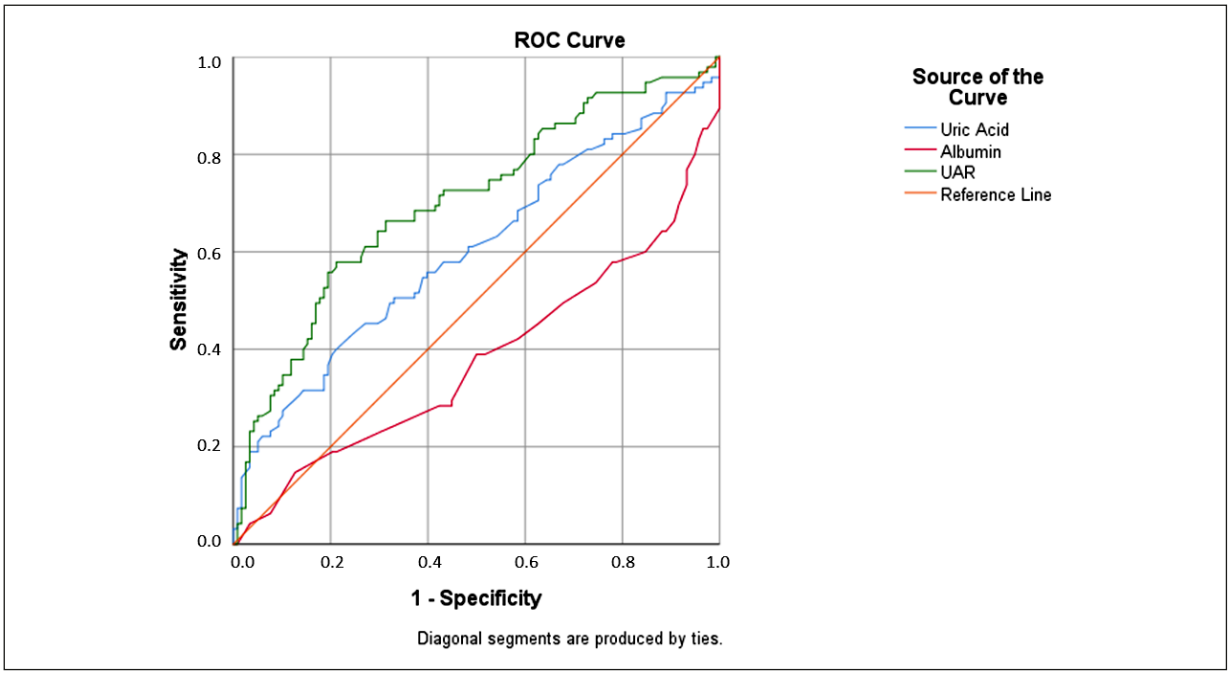

Figure 1. ROC curves of uric acid, albumin and UAR.

Table I. Clinical and biochemical variables of two groups.

	TASC A-B group (n = 138)	TASC C-D group (n = 105)	p
Age (years)	60.7 ± 8.71	63.28 ± 8.8	**0.024**
Gender (n, %)			0.490
Male	121 (87.7)	95 (90.5)	
Female	17 (12.3)	10 (9.5)	
Hypertension (n, %)	59 (42.8)	45 (42.9)	0.987
Diabetes mellitus (n, %)	43 (31.2)	41 (39)	0.200
Cerebrovascular disease (n, %)	6 (4.3)	8 (7.6)	0.281
Coronary artery disease (n, %)	39 (28.5)	44 (41.9)	**0.029**
Atrial fibrillation (n, %)	8 (5.8)	6 (5.7)	0.978
WBC (10^3/Ul)	9.04 (7.9-11.32)	9.09 (7.65-11.3)	0.687
Hemoglobin (g/dL)	13.7 (12.4-15.1)	13.4 (11.8-14.6)	0.070
Platelet (10^3/uL)	257.5 (198-309.75)	249 (210.5-302.5)	0.960
RDW (%)	13.5 (12.9-14.45)	13.6 (13.1-14.5)	0.315
Lymphocyte (10^3/uL)	2.51 (2.03-3.3)	2.05 (1.60-2.80)	**< 0.001**
Monocyte (10^3/uL)	0.7 (0.56-0.88)	0.69 (0.54-0.89)	0.776
Glucose (mg/dL)	123 (98.5-179)	127 (99-172)	0.886
Urea (mg/dL)	0.85 (0.75-1)	0.90 (0.78-1.08)	0.608
GFR (mL/min/1.73 m^2)	87.68 ± 17.33	83.35 ± 21.6	0.094
Uric acid (mg/dL)	4.7 (3.6-5.54)	5.18 (4.08-6.67)	**0.003**
Albumin (g/dL)	3.99 (3.4-4.2)	3.6 (2.7-4.1)	**0.002**
UAR	1.29 (1.05-1.51)	1.57 (1.21-1.98)	**< 0.001**
Total cholesterol (mg/dL)	207.23 ± 50.01	199.31 ± 59.12	0.289
HDL-C (mg/dL)	39 (34-47)	38.35 (33-45.02)	0.342
Triglyceride (mg/dL)	168 (128.25-239.25)	151.5 (110-206.5)	0.069
LDL-C (mg/dL)	129.9 ± 40.32	126.04 ± 53.31	0.564

WBC: White blood cell, RDW: Red cell distribution width, GFR: Glomerular filtration rate, UAR: Uric acid albumin ratio, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol.

Table II. Clinical and biochemical variables of two groups.

	AUC	p	95% CI	Value	Sensitivity	Specificity
Uric Acid	0.603	0.003	0.540-0.684	5.65	39.4	79.1
Albumin	0.380	0.040	0.302-0.457	4.35	14.7	86.7
UAR	0.702	< 0.001	0.631-0.774	1.54	57.9	78.8

AUC: area under the curve; UAR: Uric acid albumin ratio.

Table III. Pairwise comparisons of ROC curves of uric acid, albumin, and UAR.

	z	p	AUC difference	95% CI
Uric acid/albumin	4.668	< 0.001	0.223	0.129 – 0.316
Uric acid/UAR	-3.365	0.001	-0.099	-0.157 – -0.042
Albumin/UAR	-5.188	< 0.001	-0.322	-0.444 – -0.200

AUC: area under the curve; UAR: Uric acid albumin ratio.

itive correlation with the TASC-II classification of PAD. Among the variables that were examined in our study, only UAR was found to be an independent predictor of severe PAD (TASC-II C-D disease).

Inflammation has been complicit in the pathophysiology of atherosclerosis, which is the most prevalent cause of PAD. It is now clear that functional disturbances in endothelial cells with the expression of cell adhesion molecules, migration

Table IV. Binary logistic regression analysis for the prediction of TASC-II C-D disease.

	p	OR	95% CI
Age	0.026	1.034	1.004-1.066
Gender	0.522	0.763	0.334-1.744
Coronary artery disease	**0.046**	1.727	1.010
Lymphocyte	0.489	1.010	0.981-1.040
Hemoglobin	**0.045**	0.880	0.777-0.997
Glucose	0.862	1.000	0.997-1.004
Uric acid	**0.002**	1.197	1.071-1.338
Albumin	< **0.001**	0.482	0.324-0.717
UAR	< **0.001**	3.78	2.069-6.916
LDL-C	0.645	0.999	0.993-1.005
HDL-C	0.119	0.982	0.960-1.005
GFR	0.073	0.998	0.975-1.001

GFR: Glomerular filtration rate, UAR: Uric acid albumin ratio, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol.

of leukocytes into subendothelial space, and secretion of inflammatory mediators are active drivers of plaque development[16]. Various studies[17-19] examined the value of inflammatory markers in both the extent and prognosis of PAD. The results of these studies showed the utility of circulatory markers in these patients. Identification of novel risk markers might enable us to detect the disease earlier, improve our understanding of the disease pathology, and develop new treatment strategies. Several biomarkers, including neutrophil to lymphocyte ratio, C-reactive protein, interleukin-6, cellular adhesion molecules, and beta-2 microglobin, have been found to be related to severity and adverse outcomes in patients with PAD[20].

Although the exact mechanism has not been fully elucidated, the relationship between hyperuricemia and cardiovascular disease has been shown in epidemiological studies[21]. Crystallization of uric acid in the form of monosodium urate in tissues may aggravate inflammatory response. A remarkable amount of uric acid has been found[22] in atherosclerotic plaques. In contrast, the beneficial effects of serum albumin include anti-inflammation and protective effects against oxidative stress[23]. UAR is a recently introduced marker that reflects the inflammatory and oxidative conditions of the body. Its use in the prediction of new-onset atrial fibrillation has been shown[15] in ST-elevation myocardial infarction patients treated with percutaneous intervention. Çakmak et al[14] showed that compared to other biomarkers, such as neutrophil to lymphocyte ratio and C-reactive protein, UAR exhibited better predictive performance for the assessment of coronary artery disease severity in non-ST elevation myocardial infarction patients. UAR has also been shown[13,24,25] to have a value in the estimation of mortality in patients with acute kidney injury, type an acute aortic dissection, and unstable angina treated with percutaneous intervention.

Several pieces of evidence[26-28] supported the value of uric acid in PAD, independent of other traditional risk factors. Studies[26-28] demonstrated that elevated levels of uric acid were independently associated with PAD after adjustment for potential confounders. In addition, PAD patients with low levels of albumin have been found[29,30] to have more prevalent disease and worse outcomes after surgical intervention. However, the value of UAR has not been sought in any study so far. According to our results, patients with severe PAD had higher levels of UAR compared to patients who had less severe forms of the disease. UAR had higher sensitivity compared to uric acid and albumin in predicting more severe disease. We performed two models of regression in order to find the predictive values of UAR, uric acid and albumin for the presence of TASC-II CD disease. Our results showed that UAR had a higher value of OR compared to uric acid and albumin and might be a better tool for the discrimination of severe PAD disease.

Limitations

Our study was a single-center study, and the sample size was small. It had a retrospective study design. We did not assess the effect of treatment modalities that decrease UAR on disease severity. Follow-up of the study population was not done, and the prognostic value of UAR was not evaluated.

Conclusions

Uric acid and albumin are two parameters that can be easily obtained in clinical practice. Compared to a single parameter, a combination of the two could give us more information about disease severity. There is a need for further studies to evaluate the prognostic value of UAR.

Conflict of Interest
The authors declare that they have no conflict of interests.

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Ethics Approval
Ethical approval of the study was obtained from Bakırkoy Dr. Sadi Konuk Training and Research Hospital Ethical Committee (date: 04.04.2022, approval number: 2022-07-18).

Availability of Data and Materials
Data supporting the results reported in the article can be reached from the corresponding author.

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Authors' Contribution
E.O., C.Y., A.K., B.M., D.K., F.N.T.Ç had critical roles during the study conception. Study design was planned by E.O., C.Y., A.K., B.M . Supervision of the study was conducted by D.K., F.N.T.Ç., A.A.K., S.T. Statistical analysis was performed by CY. Each author also participated in patient enrollment or referral. A literature search was conducted by all the authors. The main writers were E.O., C.Y. and B.M., D.K., and F.N.T.Ç helped with the spelling. All authors have read and approved and edited the final manuscript.

Informed Consent
All the patients gave informed consent for participation in this study.

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