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Role of the uric acid/albumin ratio in predicting mortality of patients who underwent transcatheter aortic valve implantation

Fatih Levent[1], İlker Gül[2], Fatih Koca[1], Oktay Şenöz[3], Süleyman Anıl Sarıca[2]

[1]Department of Cardiology, University of Health Sciences, Bursa Yüksek Ihtisas Training Research Hospital, Bursa, Türkiye
[2]Department of Cardiology, University of Kyrenia Faculty of Medicine, Girne, The Turkish Republic of Northern Cyprus
[3]Department of Cardiology, Bakırçay University Çiğli Training and Research Hospital, Izmir, Türkiye

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ABSTRACT

Objectives: The aim of this study was to investigate whether baseline uric acid/albumin ratio (UAR) was a predictor for mortality in patients who underwent transcatheter aortic valve implantation (TAVI) due to severe aortic stenosis.

Patients and methods: The retrospective study included 240 patients (121 females, 119 males; mean age 77.5±7.6 years; range, 52 to 95 years) who underwent TAVI between January 2015 and January 2020 in two centers. Patient characteristics were compared between two groups according to mortality during follow-up (the mortality group and the survival group). The value of the UAR in predicting mortality was evaluated with receiver operating characteristic curve analysis. Predictors of mortality after TAVI were investigated with Cox regression analysis.

Results: In-hospital mortality developed in 16 (6.7%) patients, and postdischarge all-cause mortality was observed in 41 (17.1%). The two-year mortality rate was determined to be 15%. The rate of systolic heart failure, systolic pulmonary artery pressure, and UAR were found to be significantly higher in the mortality group (p=0.007, p=0.036, and p<0.001, respectively). The diagnostic power of UAR in predicting mortality was poor (the area under the curve=0.671, confidence interval [CI]: 0.589-0.753, p<0.001). Independent predictors of mortality after TAVI were UAR >2.03 (hazard ratio=2.958, CI: 1.623-5.393, p<0.001) and platelet count (hazard ratio=0.996, CI: 0.992-1.000, p=0.05).

Conclusion: Uric acid/albumin ratio was found to be an independent predictor for short-and long-term all-cause mortality in patients who underwent TAVI.

Keywords: Albumin, mortality, severe aortic stenosis, transcatheter aortic valve implantation, uric acid.

Transcatheter aortic valve implantation (TAVI) is an alternative treatment method in patients with severe aortic stenosis, particularly those at high risk for surgical aortic valve replacement. In addition to markers such as the logistic EuroSCORE (European system for cardiac operative risk evaluation), the New York Heart Association functional classification, the Society of Thoracic Surgeons Predicted Risk of Mortality algorithm, and postoperative acute renal failure, baseline albumin levels has also been shown to be a predictor for mortality after TAVI.[1-4]

Albumin not only provides intravascular oncotic pressure and functions as a hormone, drug, and free fatty acid transporter but also plays an important role as an anti-inflammatory and antiapoptotic factor and provides protection against oxidative stress.[5-7] Serum levels of albumin may be reduced due to reduced synthesis in the liver, malnutrition, increased catabolism, reduced absorption from the gastrointestinal system, and inflammation-related capillary leakage.[8] Low levels of serum albumin are associated with coronary artery disease, acute renal failure, and all-cause mortality.[9,10]

Uric acid is the end-product of the purine metabolism. Two-thirds of uric acid is excreted by the kidneys, while the remaining one-third is excreted by intestinal uricolysis.[11,12] An increased level of uric acid is associated with endothelial dysfunction, increased inflammation and oxidative stress, and activation of the renin angiotensin aldosterone system.[13,14]

Corresponding author: Fatih Levent, MD. SBÜ Bursa Yüksek İhtisas Eğitim ve Araştırma Hastanesi Kardiyoloji Kliniği, 16310 Yıldırım, Bursa, Türkiye.
E-mail: fatihlevent85@hotmail.com

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An increased level of uric acid is also an independent predictor of cardiovascular disease, aortic dilatation, hypertension, cardiovascular event, kidney failure, and all-cause mortality.[15-18]

A previous study showed that uric acid/albumin ratio (UAR) was shown to be associated with short-term mortality in acute renal failure patients.[19] Similarly, Wang et al.[20] found that UAR was an independent risk factor for postoperative long-term mortality in type A aortic dissection patients.

To the best of our knowledge, no study has been conducted to investigate whether UAR is an independent predictor for mortality following TAVI. Therefore, this study aimed to evaluate the prognostic value of UAR for mortality in TAVI patients.

PATIENTS AND METHODS

The retrospective study was conducted at two centers with 240 patients (121 females, 119 males; mean age 77.5±7.6 years; range, 52 to 95 years) who underwent TAVI between January 2015 and January 2020. The exclusion criteria were defined as follows: *(i)* having a comorbid congenital disease, *(ii)* a history of malignancy, *(iii)* a history of metabolic or hormonal disease other than diabetes or thyroid disease, *(iv)* a history of liver disease other than Grade 1 or 2 hepatic steatosis or a history of chronic gastrointestinal disease, *(v)* a diagnosis of sepsis, decompensated heart failure, or cardiogenic shock, *(vi)* having Grade ≥4 renal failure (creatine clearance <30 mL/min/1.73m^2) before the procedure, and *(vii)* a history of gout. The patients were evaluated in two groups according to mortality during follow-up (the mortality group and the survival group).

The demographic, clinical, laboratory, and procedural characteristics of the patients were recorded. The laboratory results were obtained from fasting blood samples taken between 9:00 am and 12:00 am before the procedure. The follow-up periods and mortality status of the patients were obtained from the national healthcare system. All patients underwent two-dimensional echocardiography within 24 h before the procedure. Left ventricular ejection fraction was calculated using the modified Simpson method. Patients with a fasting blood glucose >126 mg/dL measured on two separate occasions during hospitalization and those receiving antidiabetic treatment were accepted as diabetic. Patients with brachial blood pressure >140/90 mmHg measured on two separate occasions during hospitalization and those receiving anti-hypertensive treatment were accepted as hypertensive. A left ventricular ejection fraction <40% was accepted as systolic heart failure. The glomerular filtration rate was calculated using the Modification of Diet in Renal Disease formula.[21]

Statistical analysis

Statistical analyses were performed using the MedCalc version 20.014 software (MedCalc Software Ltd., Ostend, Belgium). Continuous variables with normal distribution according to the Kolmogorov-Smirnov test were expressed as mean ± standard deviation, and those not showing normal distribution were presented as median (interquartile range [IQR]). Categorical variables were expressed as number (n) and percentage (%). Comparisons of the variables between the two groups were performed with the paired samples t-test, Mann-Whitney U test, and the chi-square test. The receiver operating characteristic curve analysis was used to determine the diagnostic power of UAR and albumin level and the cutoff value of UAR for the optimal sensitivity and specificity in predicting mortality. The Kaplan-Meier survival analysis and the log-rank test were used to determine and compare the survival periods of the two groups separated by the cutoff UAR value. To determine independent predictors of mortality after TAVI, univariate and multivariate Cox regression analyses were used. Parameters with statistical significance in the univariate analysis were included in the subsequent multivariate analysis. A p value <0.05 was accepted as statistically significant.

RESULTS

The median follow-up period was 36 months (IQR: 18.75-47 months). The median logistic EuroSCORE was 28.25 (IQR: 5-86). A bicuspid aorta was determined in 11 (4.6%) patients.

Permanent pacemaker implantation due to new-onset atrioventricular complete block after the TAVI was performed in 25 (10.4%) patients. The rate of baseline right bundle branch block was significantly higher among the patients who developed new-onset atrioventricular complete block than among the patients who did not (34.8% *vs.* 12.9%, p=0.01). Valve-in-valve implantation due to severe aortic regurgitation was performed in eight (3.3%) patients.

Table 1
Demographic and clinic characteristics of the patients

	Survival group (n=183)					Mortality group (n=57)					
Variables	n	%	Mean±SD	Median	Min-Max	n	%	Mean±SD	Median	Min-Max	*p*
Age (year)			77.9±7.6					77.6±7.8			0.797
Sex											
Male	92	50.3				27	47.4				0.702
Body mass index (kg/m^2)			26.8±4.7					26.3±3.2			0.470
Diabetes	71	38.8				15	26.3				0.086
Coronary artery disease	74	40.4				25	43.9				0.647
Hypertension	132	72.1				42	73.7				0.819
Systolic heart failure	46	25.1				25	43.9				0.007
Low-gradient aortic stenosis	37	20.2				15	26.3				0.329
Stroke	10	5.5				3	5.3				0.953
Hypoalbuminemia (<3.5 g/dL)	52	28.4				36	63.2				<0.001
COPD	30	16.4				12	21.1				0.419
Cancer	5	2.7				5	8.8				0.060
Atrial fibrillation	46	25.1				20	35.1				0.142
RBBB	29	15.8				7	12.3				0.510
Bicuspid aortic valve	10	5.5				1	1.8				0.242
Beta-blocker	114	62.3				33	57.9				0.552
ACE inh/ARB	139	76				36	63.2				0.058
Diuretic	116	63.4				30	52.6				0.146
Mineralocorticoid receptor antagonist	53	29.0				16	28.1				0.897
Valve type											0.357
SAPIEN XT	29	15.8				12	21.1				
ACURATE neo	48	26.2				9	15.8				
St. Jude Medical Portico	53	29.0				16	28.1				
CoreValve Evolut R	53	29.0				20	35.1				
Log. EuroSCORE (%)				28.00	5.00-86.00				30.08	9.00-85.90	0.095

SD: Standard deviation; COPD: Chronic obstructive pulmonary disease; RBBB: Right bundle branch block; ACE inh: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; Log. EuroSCORE: Logistic European system for cardiac operative risk evaluation.

Table 2
The laboratory and echocardiographic characteristics of the patients

Variables	Survival group (n=183)			Mortality group (n=57)			*p*
	Mean±SD	Median	25th-75th percentile	Mean±SD	Median	25th-75th percentile	
Hemoglobin (g/dL)	11.5±1.7			11.2±1.8			0.243
CrCl (mL/min)		62.27	43.84-76.70		58.76	40.23-77.91	0.546
Sodium (mEq/L)	137.2±3.9			137.6±3.6			0.447
Potassium (mmol/L)	4.6±3.0			4.3±0.7			0.374
White blood cell ($\times10^9$/L)	7.9±2.8			7.6±2.3			0.548
hs-CRP (mg/L)		2.86	0.65-4.10		3.11	1.05-5.96	0.145
ALT (IU/L)	23.1±36.8			20.7±29.1			0.653
AST (IU/L)	27.6±28.5			26.7±30.9			0.837
TSH (mIU/L)		1.37	0.90-2.30		1.45	0.75-3.27	0.736
Platelet ($\times10^3$/L)	235.0±76.4			212.4±72.4			0.050
Triglyceride (mg/dL)	136.3±83.7			128.9±56.3			0.533
LDL (mg/dL)	108.0±38.0			108.4±38.3			0.938
BNP (pg/mL)		1481	636- 4347		2336	952-3942	0.125
Albumin (g/dL)	3.7±0.5			3.4±0.6			<0.001
Uric acid (mg/dL)	7.1±2.1			7.8±2.0			0.054
UAR	2.0±0.6			2.3±0.7			<0.001
Pre-op (LVEF %)	46.0±12.0			41.9±13.1			0.027
PASP (mmHg)	49.8±13.4			54.2±15.2			0.036
AVA (cm^2)	0.7±0.1			0.7±0.1			0.579
TAPSE (mm)	20.6±2.7			20.4±2.8			0.541

SD: Standard deviation; CrCI: Creatinine Clearance; hs-CRP: High sensitivity c-reactive protein; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; TSH: Thyroid-stimulating hormone; LDL: Low-density lipoprotein; BNP: Brain natriuretic peptide; UAR: Uric acid/albumin ratio; LVEF: Left ventricular ejection fraction; PASP: Pulmonary artery systolic pressure; AVA: Aortic valve area; TAPSE: Tricuspid annular plane systolic excursion.

Following TAVI, acute renal failure was determined in 28 (11.7%) patients, peripheral vascular damage (e.g., dissection, hematoma) in 32 (13.3%), ischemic stroke in six (2.5%), myocardial infarction in 10 (4.2%), and ascending aorta dissection in two (0.8%). In-hospital mortality occurred in 16 (6.7%) patients, whereas postdischarge all-cause mortality occurred in 41 (17.1%). Of 16 in-hospital deceased patients, two patients died due to stroke, two patients due to ascending aorta dissection, three patients due to myocardial infarction, three patients due to peripheral vascular damage, one patient due to nosocomial pneumonia, three patients due to decompensated heart failure/cardiogenic shock, and two patients due to an unknown cause. The two-year mortality rate was determined to be 15% (n=36).

When the demographic and clinical characteristics of the patients were compared, the rate of systolic heart failure was found to be higher in the mortality group compared to the survival group (43.9% *vs.* 25.1%, p=0.007). The demographic and clinical characteristics of the patients are shown in Table 1.

When the laboratory and echocardiographic data of the patients were compared, the albumin level was significantly lower and the UAR was significantly higher in the mortality group (p<0.001 and p=0.001, respectively). The laboratory and echocardiographic data of the patients are shown in Table 2.

The area under the curve (AUC), evaluated for determining the diagnostic power of UAR in

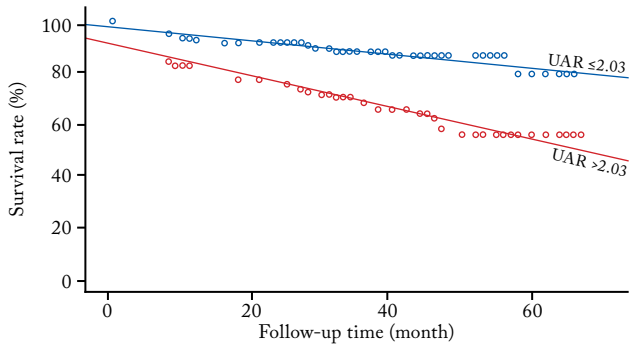

Figure 1. Kaplan-Meier analysis comparing the survival periods in patients above and below the cut off value of UAR.
UAR: Uric acid/albumin ratio.

Table 3
Cox regression analysis for identifying predictors of mortality following TAVI

Variables	HR	95% CI	*p*
Univariate analysis			
SHF	1.956	1.159-3.301	**0.012**
Platelet count	0.996	0.992-1.000	**0.039**
UAR >2.03	3.139	1.741-5.662	**<0.001**
PASP	1.016	0.998-1.035	0.086
Multivariate analysis			
SHF	1.457	0.850-2.497	0.171
Platelet count	0.996	0.992-1.000	**0.05**
UAR >2.03	2.958	1.623-5.393	**<0.001**

TAVI: Transcatheter aortic valve implantation; HR: Hazard ratio; CI: Confidence interval; SHF: Ssystolic heart failure; UAR: Uric acid/albumin ratio; PASP: Pulmonary artery systolic pressure.

predicting mortality, was 0.671 (confidence interval [CI]: 0.589-0.753, p<0.001). No significant difference was found between the AUC of albumin and the AUC of UAR (0.675 *vs.* 0.671, p=0.93). A UAR value >2.03 had 70.2% sensitivity and 60.1% specificity in predicting all-cause mortality.

In the Kaplan-Meier survival analysis, the survival period was significantly shorter in the group with a UAR >2.03 (46.9 months *vs.* 58.6 months, p<0.001, Figure 1).

In the univariate Cox regression analysis, systolic heart failure, platelet count, and a UAR >2.03 were found to be associated with mortality after TAVI. In the multivariate regression analysis, UAR >2.03 (hazard ratio=2.958, CI: 1.623-5.393, p<0.001) and platelet count (hazard ratio=0.996, CI: 0.992-1.000, p=0.05) remained as independent predictors for mortality after TAVI (Table 3).

DISCUSSION

This study investigated the prognostic value of UAR for mortality in TAVI patients. The results showed the in-hospital mortality rate to be 6.7% and the postdischarge all-cause mortality to be 17.1%. Although the predictive power of UAR for mortality was poor in the receiver operating characteristic analysis, a UAR above the cutoff value of 2.03 was an independent predictor of increased risk of mortality following TAVI. A decreased platelet count was also an independent predictor of mortality.

In a study in 2016, Gaede et al.[22] evaluated the results of 15,050 patients who had undergone TAVI and reported an in-hospital mortality rate of 2.6% and a permanent pacemaker implantation rate of 11.4%. Additionally, in this study, a cerebrovascular event was observed in 2.2%, vascular complications in 7.1%, myocardial infarction in 0.2%, and acute renal damage in 3%. Compared to the results of the current study, the lower rates of vascular complications, in-hospital mortality, and acute renal damage can be considered to be due to a difference in the experience of the operators and different study populations. In a 2013 study by Finkelstein et al.,[23] the outcomes of 300 TAVI patients were reported and in-hospital mortality, stroke, minor vascular complication, and permanent peacemaker implantation rates were 2.3%, 1.6%, 10.7%, and 22%, respectively. Although the in-hospital mortality rate was low compared to the rate in the current study, the high rate of permanent pacemaker implantation could have been due to the use of older generation bioprosthetic valves. Gleason et al.[3] evaluated patients implanted with a self-expandable valve, and found the five-year mortality rate after TAVI to be 55.3%. The significantly higher mortality could be attributed to the difference in the study populations and the shorter follow-up period in that study.

The current study results showed a low platelet count to be an independent predictor of mortality following TAVI. In parallel to the current study results, Kalińczuk et al.[24] also reported that a reduced platelet count after TAVI was associated with increased mortality. In another study, the development of thrombocytopenia after TAVI was found to be a predictor of 30-day

mortality, and platelet count before the TAVI was the only predictor of thrombocytopenia.[25] Similarly, it can be considered that a lower thrombocyte count in the current study may have increased the risk of thrombocytopenia development after TAVI. However, there is a need for further studies on this subject.

In a previous meta-analysis, the baseline albumin level was found to be an independent predictor of 30-day and one-year mortality after TAVI.[4] This finding is compatible with the finding of the current study that the albumin level was significantly lower in the mortality group. The mounting evidence indicating that low albumin level is related to endothelial dysfunction, subclinical systemic inflammation, and renal and liver dysfunction could explain the increased mortality in TAVI patients. Moreover, a decreased albumin level causes decreased intravascular osmotic colloidal pressure, which may accelerate the development of acute renal damage, pulmonary edema, or decompensated heart failure in TAVI patients.[5,26,27]

In a study by Sokmen et al.,[1] although the baseline uric acid level was significantly higher in patients who deceased after TAVI, it was not found to be an independent predictor of mortality. It has been previously stated that a high uric acid level is associated with increased inflammation, renin-angiotensin aldosterone activation, endothelial dysfunction, and aorta dilatation.[13,14,28]

To minimize the effect of the confounding factors on uric acid and albumin levels, these two markers were combined in the current study. Evidence related to the prognostic importance of uric acid and albumin levels may explain why the ratio of these two markers is a predictor of mortality following TAVI.

This study has several limitations. This study was retrospective in design, was conducted in only two centers, and the number of patients included was relatively limited. The scores other than the logistic EuroSCORE were not evaluated. Only four types of bioprosthetic valves were preferred so there was no experience with other bioprosthetic valves (e.g., Myval). When evaluating UAR and platelet count, only preprocedural blood results were used. Therefore, the effects of change in UAR and platelet count could not be investigated.

In conclusion, UAR, an easy-to-obtain and practical marker, was found to be an independent predictor of mortality after TAVI. Therefore, it would be appropriate to investigate the value of this marker with more evidence-based research methods to determine the risk of death in TAVI patients.

Ethics Committee Approval: The study protocol was approved by the University of Health Sciences, Bursa Yüksek Ihtisas Training and Research Hospital Ethics Committee (date: 02.11.2022, no: 2011-KAEK-25 2022/11-15). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patient Consent for Publication: A written informed consent was obtained from each patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept, design, data collection and processing, analysis and interpretation, writing the article, materials: F.L.; Idea/concept, control /supervision, data collection and processing, literature review, writing the article, critical review, references and fundings: İ.G.; Design, writing the article, materials: F.K.; Control/supervision, literature review, references and fundings: O.Ş.; Analysis and interpretation, critical review; S.A.S.

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