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Makale Türü Özgün Araştırma

The predictive value of neutrophil to lymphocyte ratio and mean platelet volume on acute kidney injury after TAVI

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ABSTRACT

Objective: Transcatheter aortic valve implantation (TAVI) is an effective treatment for severe aortic stenosis. Acute kidney injury (AKI) is a common complication after TAVI. The development of AKI after TAVI has been linked to the thromboinflammatory response as evaluated by the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV). We sought to determine the predictive value of NLR and MPV on AKI after TAVI in this study. **Method:** This is a single-center retrospective study. Baseline, peri-procedural, and post-procedural demographic, laboratory, and clinical characteristics were assessed for AKI. The optimal NLR and MPV cut-off values for predicting AKI were determined using receiver operating characteristic (ROC) curve analysis. **Results:** A total of 184 consecutive patients (age 79.4 ± 7.8 years, 61.4% female) who underwent TAVI were included in this study. The optimal cut-off points for the NLR and MPV were 5.70 (AUC: 0.790; 95% CI: 0.711-0.869, p<0.001 and sensitivity= 80.0%, specificity= 65.2%) and 9.15 fL (AUC:0.713; 95% CI: 0.629-0.997, p<0.001 and sensitivity=70.7%, specificity=63.6%), respectively. AKI occurred in 41 (22.3%) patients included in the study and 6 (3.3%) required dialysis. Baseline GFR (OR: 0.98; 95% CI: 0.94-0.99, p = 0.001), post-procedural WBC count (OR: 1.17; 95% CI: 1.03-1.33, p= 0.02), post-procedural NLR≥ 5.7 (OR: 5.16; 95% CI: 1.84-14.48, p= 0.002), and post-procedural MPV ≥ 9.15 fL (OR: 3.70; 95% CI: 1.54–8.91, p= 0.004) were found to be the independent predictors of AKI after TAVI in multivariable analysis. **Conclusion:** Post-procedural NLR and MPV were found to be independent predictors of AKI after TAVI.

Keywords: Acute kidney injury, transcatheter aortic valve replacement, neutrophil, lymphocyte, mean platelet volume

TAVI sonrası akut böbrek hasarında nötrofil/lenfosit oranı ve ortalama trombosit hacminin öngördürücü değeri

ÖZET

Amaç: Transkateter aort kapak implantasyonu (TAVI), ciddi aort darlığı için etkili bir tedavidir. Akut böbrek hasarı (ABH), TAVI sonrası sık görülen bir komplikasyondur. TAVI sonrası ABH gelişimi, nötrofil-lenfosit oranı (NLR) ve ortalama trombosit hacmi (MPV) ile değerlendirilen tromboinflamatuar yanıtla ilişkilendirilmiştir. Bu çalışmanın amacı, TAVI sonrası gelişen ABH'de NLR ve MPV'nin öngördürücü değerini belirlemektir. **Yöntem:** Bu çalışma tek merkezli retrospektif bir çalışmadır. ABH için bazal, işlem öncesi ve işlem sonrası demografik, laboratuvar ve klinik özellikler değerlendirildi. ABH'yi en iyi öngördüren NLR ve MPV kesme değerleri, alıcı işletim karakteristik (ROC) eğrisi analizi kullanılarak belirlendi. **Bulgular:** Bu çalışmaya TAVI yapılan ardışık 184 hasta (yaş 79.4 ± 7.8 yıl, %61.4 kadın) dahil edildi. NLR ve MPV için optimal kesme değerleri sırasıyla 5.70 (EAA: 0.790; %95 GA: 0.711-0.869, p<0,001 ve duyarlılık= %80.0, özgüllük= %65.2) ve 9.15 fL (EAA: 0.713; %95 GA: 0.629-0.997, p<0.001 ve duyarlılık= %70.7, özgüllük=%63.6) olarak saptandı. Çalışmaya dahil edilen 41 (%22.3) hastada ABH meydana geldi ve 6 (%3.3) hastada diyaliz uygulanması gerekti. Çok değişkenli analizde bazal GFR (OR: 0.98; %95 GA: 0.94-0.99, p = 0.001), prosedür sonrası WBC sayısı (OR: 1.17; %95 GA: 1.03-1.33, p= 0.02), prosedür sonrası NLR ≥5.7 (OR: 5.16; %95 GA: 1.84-14.48, p= 0.002) ve işlem sonrası MPV ≥9.15 fL (OR: 3.70; %95 GA: 1.54–8.91, p= 0.004) TAVI sonrası gelişen ABH'nin bağımsız öngördürücüleri olarak bulundu. **Sonuç:** İşlem sonrası NLR ve MPV, TAVI sonrası ABH'nin bağımsız öngördürücüleri olarak bulundu.

Anahtar Kelimeler: Akut böbrek hasarı, transkateter aort kapağının değiştirilmesi, nötrofil, lenfosit, ortalama trombosit hacmi

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INTRODUCTION

Aortic stenosis (AS) frequently identified valve lesion in Europe.[1] Transcatheter aortic valve implantation (TAVI) is effective and minimally invasive modality.[2] Acute kidney injury (AKI) is a significant complication after TAVI and occurs approximately at 10.7%.[3-5] AKI following TAVI leads to increased morbidity and 1-year mortality.[5-6]

Age, female gender, diabetes mellitus, chronic kidney disease, hypertension, peripheral vascular disease, hemodynamic instability, major bleeding, abnormal leukocyte count, and thrombocytopenia have all been identified as risk factors for AKI after TAVI.[7-12] Thromboinflammatory response after TAVI has been linked to poor outcomes in numerous studies.[13-14] To assess the thromboinflammatory response, basic whole blood parameters such as the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume were used (MPV). The NLR has been identified as a predictive marker of systemic inflammation in a number of cardiovascular and non-cardiovascular diseases.[15-17] Following TAVI, the NLR has been linked to the development of AKI.[18-20] The average platelet volume (MPV) is a computerized measurement of platelet size. As a result of increased platelet size, an increased MPV indicates larger, more reactive platelets.[21] It has been proposed that it could be used as a prothrombotic indicator in a variety of metabolic and cardiovascular disorders.[22] MPV has been shown to be higher in AS and lower after TAVI.[23-24] There is, however, little information in the literature about how MPV affects AKI after TAVI.

We aimed to investigate the predictive value of NLR and MPV on AKI after TAVI in this study.

METHODS

Patient population

The study was carried out retrospectively at a single cardiovascular center. We looked at 196 patients who had TAVI for severe AS between October 2010 and December 2016. This research adheres to the Helsinki Declaration's ethical principles. The hospital's institutional review board granted ethics committee approval (IRB date and number: 26.04.2017-2017/06).

Patient selection

Inclusion criteria: 1-Severe AS, 2-Symptomatic patients (NYHA class III-IV), 3-High risk of surgery (EuroSCORE >20% or an STS score >10%).[25-26]

Exclusion criteria: 1-Severe left ventricular systolic dysfunction (LVEF < 20%) 2- AMI, 3- Coronary artery disease necessitating revascularization, 4- Chronic dialysis prior to TAVI, 5-Active infection, 6-Expected life <12 months.

Procedural details

The severity of AS, the structure of the aortic valve, and the aortic root were evaluated using transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE). Aortic root calcification, femoral and iliac artery diameters, as well as calcifications and tortuosities, were evaluated using multislice computed tomography (CT) and angiography. The coronary anatomy was assessed using traditional coronary angiography.

Under general anesthesia, TAVI was carried out in the catheterization laboratory. Patients who required anticoagulation due to atrial fibrillation or other conditions were given aspirin and warfarin, but not clopidogrel.

Data collection

Data on baseline and post-procedural features, co-morbidities, laboratory results (CBC and biochemistry), and echocardiographic findings pre and post procedure were collected by reviewing patient medical records. CBC and chemistry blood samples were obtained 24 hours prior to the procedure and then once daily for up to 7 days while the patient was hospitalized.

Definitions and study endpoints

AKI was defined according to the valve academic research consortium-3 (VARC-3) criteria.[26] Procedure was deemed successful if the valve expanded in the correct location and without significant aortic regurgitation. The estimated glomerular filtration rate (eGFR) was calculated by the Modification of Diet in Renal Disease (MDRD) equation.[27] CKD was defined as eGFR of <60 mL/min/1.73m². Atrial fibrillation was identified by an ECG performed during index admission and/or from medical. "Anemia is hemoglobin level of <13 g/dL in men and <12 g/dL in women".[28] A platelet count of less than $140x10^9$/L was considered to be thrombocytopenia. The words "peri-procedural" and "post-procedural" refer to the 24 and 72 hours following the beginning of the procedure, respectively, throughout this article.

The primary endpoint was the incidence of AKI following TAVI. Secondary endpoints included determining the predictive value of NLR and MPV on acute kidney injury following TAVI. For predicting AKI, the optimal cut-off value for NLR (post-procedural day 3) was determined to be 9.15 and for MPV (post-procedural day 3) was 5.70 using ROC curve analysis.

Statistical analysis

The normality of the continuous variables was assessed by Shapiro-Wilk The Student t-test was used for continuous variables and the Chi-square test for categorical variables to compare the groups. A ROC curve was used to identify the optimal cut-off points for the NLR and MPV values. AUC was calculated to evaluate the discrimination ability of NLR and MPV for AKI. Logistic regression was used to for univariable and multivariable analyses. Statistical Package for the Social Sciences (SPSS) software program (version 20.0, SPSS, Chicago, Illinois, USA) was used for statistical analyses.

RESULTS

One hundred ninety-six TAVI patients were screened and 184 patients included in the study.

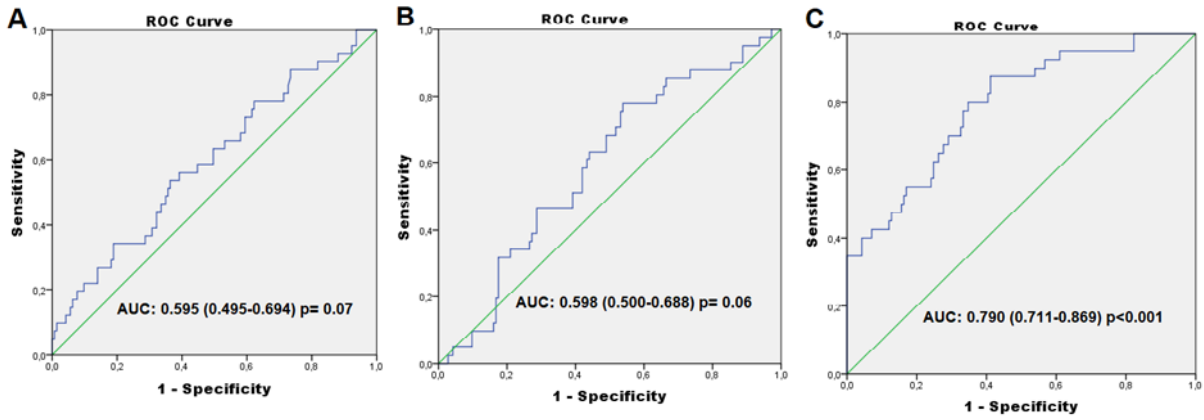

Figure 1. ROC curves of (A) baseline, (B) peri-procedural, and (C) post-procedural 3. day NLR for predicting AKI after TAVI

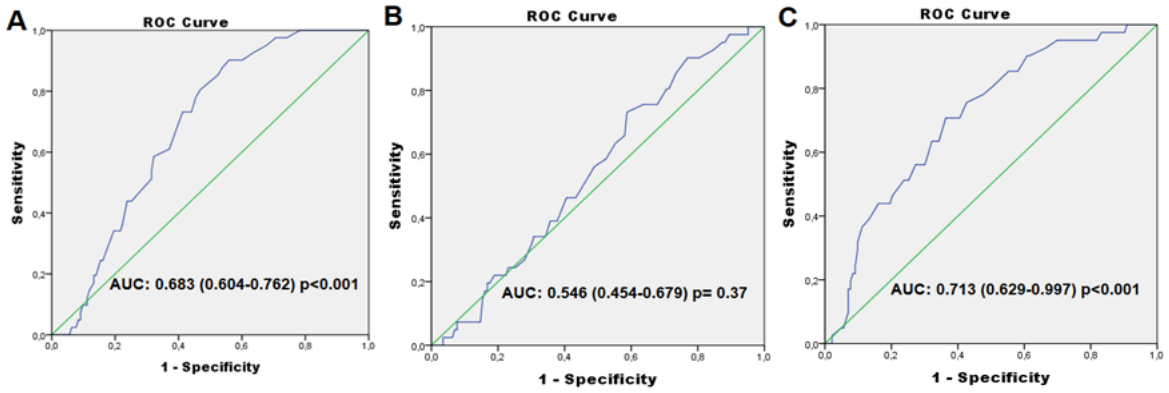

Figure 2. ROC curves of (A) baseline, (B) peri-procedural, and (C) post-procedural 3. day MPV for predicting AKI after TAVI

A ROC curve analysis was used to identify the optimal cut-off points for the NLR and MPV for predicting AKI. ROC curves for NLR and MPV were drawn at the baseline, peri-procedural, and post-procedural time points (Figure 1 and Figure 2). The AUC was the highest at the post-procedural time point for both NLR and MPV. A cut-off value of 5.70 for the NLR (AUC: 0.790; 0.711-0.869, p<0.001 and sensitivity= 80.0%, specificity= 65.2%) and 9.15 fL for the MPV (AUC:0.713; 0.629-0.997, p<0.001 and sensitivity=70.7%, specificity=63.6%).

Baseline characteristics of the patients are given in Table 1 and Table 2. The mean age was 79.4 ± 7.8 years, and 113 (61.4%) of the patients were female. The prevalence of CKD, higher WBC count, and thrombocytopenia were significantly higher in the AKI group. Also, the mean NLR and MPV levels at post-procedural time points were significantly higher in the AKI group.

Acute kidney injury

AKI happened in 41 (22.3%) of the patients included in the study. Twenty-two (12%) patients developed AKI stage 1, 11 (5.9%) developed stage 2, 2 (1.1%) developed stage 3, and 6 (3.3%) developed stage 4 (requiring dialysis following the TAVI procedure).

Table 1. Baseline characteristics of the patients

Variables	All Patients (n=184)	AKI (n=41, 22.3%)	Non-AKI (n=143, 77.7%)	P-value
Age (years)	79.4 ± 7.8	79.3 ± 8.3	79.4 ± 7.8	0.92
Female gender	113 (61.4%)	23 (56.1%)	90 (62.9%)	1.00
Body mass index (kg/m2)	27.5 ± 5.5	26.6 ± 5.3	28.3 ± 5.7	0.38
Logistic EuroSCORE (%)	32.7 ± 15.4	30.3 ± 14.6	33.4 ± 15.6	0.26
STS score (%)	12.0 ± 5.0	11.6 ± 6.2	12.2 ± 4.7	0.55
NYHA Class III	119 (64.7%)	25 (61.0%)	94 (65.7%)	0.58
Class IV	65 (35.30%)	16 (39.0%)	49 (34.3%)	
Coronary artery disease	114 (62.0%)	24 (58.5%)	90 (62.9%)	0.61
Coronary artery bypass graft surgery	44 (23.9%)	10 (24.4%)	34 (23.8%)	0.93
Peripheral vascular disease	68 (37%)	12 (29.3%)	56 (39.2%)	0.25
Cerebrovascular disease	8 (4.3%)	3 (7.3%)	5 (3.5%)	0.29
Pulmonary hypertension	124 (67.4%)	27 (65.9%)	97 (67.8%)	0.82
Atrial Fibrillation	30 (16.3%)	6 (14.6%)	24 (16.8%)	0.74
Hypertension	151 (82.1%)	31 (75.6%)	120 (83.9%)	0.2
Diabetes Mellitus	74 (40.2%)	19 (46.3%)	55 (38.5%)	0.36
Chronic obstructive pulmonary disease	112 (60.9%)	25 (61.0)	87 (60.8%)	0.99
Chronic kidney disease	54 (29.3%)	19 (46.3%)	35 (24.5%)	**0.007**
Serum creatinine (mg/dL)	1.07 ± 0.60	1.26 ± 0.80	1.01± 0.53	**0.02**
Glomerular filtration rate (mL/min/1.73m²)	69.0 ± 24.7	58.5 ± 22.3	72.0 ± 24.6	**0.002**
Hemoglobin, (g/dL)	11.5 ± 1.7	11.3 ± 1.8	11.6 ± 1.70	0.30
Platelet (×10^9/L)	215.2 ± 83.5	216.3 ± 123.1	214.9 ± 68.7	0.92
Thrombocytopenia (<140×10^9/L)	27 (14.7%)	5 (12.2%)	22 (15.4%)	0.61
MPV, fL	8.72 ± 1.63	9.23 ± 1.14	8.58 ± 1.73	**0.02**
White blood cell (× 10^9/L)	7.10 ± 1.90	7.01 ± 1.90	7.12 ± 1.89	0.73
Neutrophil (× 10^9/L)	4.48 ± 1.73	4.64 ± 1.94	4.43 ± 1.67	0.50
Lymphocyte (× 10^9/L)	1.74 ± 0.58	1.56 ± 0.64	1.79 ± 0.55	0.02
NLR	2.98 ± 2.28	3.80 ± 3.74	2.74 ± 1.58	0.08
Echocardiographic data				
Baseline aortic valve area (cm²)	0.74 ± 0.14	0.75 ± 0.15	0.74 ± 0.14	0.74
Baseline peak gradient (mm Hg)	83.2 ± 18.6	86.4 ± 19.4	82.2 ± 18.3	0.20
Baseline mean gradient (mm Hg)	51.5 ± 12.7	53.6 ± 12.6	50.9 ± 12.9	0.24
Moderate or severe mitral regurgitation	54 (29.3%)	11 (29.3%)	43 (30.1%)	0.68
Left ventricular ejection fraction, %	53.7 ± 12.2	54.1 ± 11.0	53.3 ± 12.5	0.70
Left ventricular ejection fraction ≤%40	37 (20.1%)	6 (14.6%)	31 (21.7%)	0.32

EuroSCORE: european system for cardiac operative risk evaluation, **NYHA:** New York Heart Association, **STS:** Society of Thoracic Surgeons,

Table 2. Peri-procedural and post-procedural clinical, and laboratory characteristics of the patients

Variables	All Patients (n=184)	AKI (n=41, 22.3%)	Non-AKI (n=143, 77.7%)	P-value
Peri-procedural data				
Contrast amount (mL)	178.8 ± 26.9	182.4 ± 32.5	177.8 ± 25.1	0.33
Implanted valve size (mm)	25.5 ± 2.1	26.0 ± 2.4	25.4 ± 2.0	0.11
Valve type				0.59
Edwards Sapien	147 (79.9%)	32 (78.0%)	115 (80.4%)	
Lotus	26 (14.1%)	5 (12.2%)	21 (14.7%)	
Medtronic CoreValve	7 (3.8%)	3 (7.3%)	4 (2.8%)	
Portico	4 (2.2%)	1 (2.4%)	3 (2.1%)	
Blood transfusion (%)	42 (22.8%)	13 (31.7%)	29 (20.3%)	0.12
Red blood cell (U)	2.76 ± 2.36	2.87 ± 2.58	2.51 ± 1.81	0.68
Peripheral vascular complication	23 (12.5%)	11 (26.8%)	12 (8.4%)	0.22
Platelet count (×10^9/L)	170.2 ± 87.0	169.1 ± 144.5	170.6 ± 62.4	0.93
Mean platelet volume, fL	8.98 ± 1.77	9.02 ± 1.42	8.87 ± 1.87	0.87
Thrombocytopenia (<140×10^9/L)	64 (34.8%)	18 (43.9%)	46 (32.2%)	0.17
White blood cell (× 10^9/L)	12.18 ± 4.60	15.04 ± 5.92	11.36 ± 3.80	**<0.001**
Neutrophil-to-lymphocyte ratio	12.92 ± 8.32	14.18 ± 7.47	12.56 ± 8.54	0.27
Post-procedural data				
Platelet count (×10^9/L)	18.5 ± 63.9	117.9 ± 82.8	131.5 ± 57.5	0.24
Thrombocytopenia (≤ 140×10^9/L)	120 (65.2%)	32 (80.0%)	88 (62.4%)	**0.04**
MPV, fL	9.15 ± 1.56	9.94 ± 1.40	8.93 ± 1.54	**<0.001**
MPV ≥ 9.15 fL	81 (44.0%)	29 (70.7%)	52 (36.4%)	**<0.001**
White blood cell (× 10^9/L)	9.87 ± 4.35	13.30 ± 6.58	8.89 ± 2.81	**<0.001**
NLR	7.29 ± 5.83	12.72 ± 8.50	5.75 ± 3.58	**<0.001**
NLR ≥ 5.7	82 (44.6%)	32 (80.0%)	50 (35.5%)	**<0.001**
Postoperative AVA, (cm²)	1.94 ± 0.33	1.89 ± 0.39	1.97 ± 0.35	0.82
Peak gradient (mm Hg)	18.9 ± 7.8	19.2 ± 8.1	18.8 ± 8.2	0.48
Mean gradient (mm Hg)	10.0 ± 4.4	10.2 ± 4.8	9.9 ± 4.5	0.57
Left ventricular ejection fraction, %	54.2 ± 10.6	55.1 ± 10.9	53.6 ± 10.1	0.68
Paravalvalvular aortic regurgitation ≥ 2+	27 (14.7%)	6 (14.6%)	21 (14.7%)	0.99

Predictive factors of acute kidney injury

Baseline creatinine, GFR, NLR, MPV, and the presence of CKD and post-procedural WBC count, thrombocytopenia, MPV, NLR ≥ 5.7, and MPV ≥ 9.15 fL had a p-value <0.10 on univariable analysis. These variables were included in the model in the multivariable logistic regression analysis. Baseline GFR (OR: 0.98; 0.94-0.99, p = 0.001), post-procedural WBC count (OR: 1.17; 1.03-1.33, p= 0.02), post-procedural NLR≥ 5.7 (OR: 5.16; 1.84-14.48, p= 0.002), and post-procedural MPV ≥ 9.15 fL (OR: 3.70; 1.54–8.91, p= 0.004) were found to be the independent predictors of AKI after TAVI in multivariable analysis (Table 3).

Table 3. Independent predictors of AKI after TAVI in univariable and multivariable logistic regression analyses

	Univariable analysis			Multivariable analysis		
Variables	**OR**	**95% CI**	**p**	**OR**	**95% CI**	**p**
Chronic kidney disease	2.66	1.29 – 5.49	**<0.001**			
Baseline creatinine	1.76	0.97 – 3.19	0.06			
Baseline glomerular filtration rate	0.97	0.95 – 0.99	0.003	0.98	0.94 – 0.99	**0.01**
Baseline NLR	1.19	1.02 – 1.40	0.02			
Baseline MPV	1.25	1.02 – 1.53	0.03			
Post-procedural Thrombocytopenia	2,40	1.03 – 5.61	**0.04**			
Post-procedural MPV	1.48	1.18 – 1.85	**0.001**			
Post-procedural MPV ≥ 9.15 fL	4.23	1.99 – 8.99	**<0.001**	3.70	1.54 – 8.91	**0.004**
Post-procedural white blood cell count	1.30	1.16 – 1.45	**<0.001**	1.17	1.03 – 1.33	**0.02**
Post-procedural NLR ≥ 5.7	7.28	3.12 – 17.00	**<0.001**	5.16	1.84 – 14.48	**0.002**

CI: confidence interval, **OR**: Odds ratio

DISCUSSION

AKI occurred in 41 patients (22.3%) after TAVI, and six patients (3.3%) required temporary hemodialysis in this study. The independent predictors of AKI after TAVI were found to be baseline GFR, post-procedural WBC count, post-procedural NLR≥ 5.7, and post-procedural MPV≥ 9.15 fL.

The incidence of AKI after TAVI varies in the literature due to the use of different definition criteria (RIFLE, AKIN, VARC 1 criteria). AKI is a common complication after TAVI and ranging between 6% and 41%.[3-5] The incidence of AKI in this study is consistent with the observations in the literature.

TAVI-related AKI has been linked to a variety of baseline characteristics, co-morbidities, and peri-procedural factors. Several studies investigated the association between baseline kidney function and the development of AKI after TAVI. For this purpose, baseline creatinine, GFR, and the presence of CKD were used. Elhmidi et al.[9] found that the baseline creatinine concentration was the only independent risk factor for AKI. Also, baseline creatinine concentration of ≥1.58 mg/dL, and >104 mol/L, was revealed to be an independent risk factor for AKI by Sinning et al.[29]and Alassar et al.[30], respectively. Nominally we found higher risk for AKI with the baseline creatinine concentration (OR: 1.76; p=0.06; per 1-unit increase) in our study. However, baseline creatinine was not an independent risk factor for AKI.

Sudarsky et al.[31] discovered that baseline GFR of ≤45 mL/min/1.73 m2 was an independent risk factor for AKI. The baseline GFR was also reported by Bassat et al.[32] to be an independent risk factor for AKI. In our study, baseline GFR was an independent risk factor for AKI in multivariable analysis.

Wang et al.[33] reported in a meta-analysis that CKD was related with an increased risk of AKI after TAVI (OR; 2.81; 1.96–4.03). CKD was related with an increased risk of AKI in univariable analysis in our study. However, it was not an independent risk factor in the multivariable analysis.

Increased thromboinflammatory response after TAVI is associated with poor outcomes.[13-14] This inflammatory process might be effective in the pathogenesis of AKI. Leukocyte count, C-reactive protein, and procalcitonin levels all increased after SIRS in patients as a result of the production of IL-6 and IL-8 being greatly enhanced.[29] Nuis et al.[10] and Turen et al.[12] found that abnormal leukocyte count was an independent risk factor for AKI. Consistent with these findings, post-procedural leukocyte (WBC) count was an independent risk factor for AKI in our study (OR: 1.30; p= 0.02).

The NLR has been identified as a predictive marker of systemic inflammation in various diseases.[15-17] NLR reflects the dynamic interaction between innate (neutrophils) and adaptive cellular immunological responses to disease and diverse pathological circumstances, The NLR has been associated with AKI. Yelgeç et al.[18] found that post-procedural NLR was an independent risk factor for AKI (OR: 1.79; p=0.01). Olasinska-Wisniewska et al.[19] also discovered that NLR increased significantly after TAVI in both the lowest and highest quartiles, with the increase being significantly greater in patients who developed AKI (median NLR of 8.3 vs. 4.9, p= 0.005). Another study by Olasinska-Wisniewska et al.[20] revealed that NLR was decreased significantly after TAVI. We also found

that the post-procedural NLR≥ 5.7 was an independent risk factor for AKI.

MPV has been shown to be increased in AS and decreased after TAVI.[23-24] We found that the post-procedural MPV≥ 9.15 was an independent risk factor for AKI after TAVI (OR: 3.70; p= 0.004) in our study.

CONCLUSION

AKI following TAVI occurred in 22.3% of the patients. Baseline GFR, post-procedural WBC count, post-procedural NLR≥ 5.7, and post-procedural MPV≥ 9.15 fL were found to be independent predictors of AKI. NLR and MPV are objective and easily obtainable hematological markers of thromboinflammatory activation. They can be used as a tool for the prediction of AKI after TAVI.

Study limitations

This was a single-center, retrospective, observational study. NLR and MPV were evaluated at a specific point in time and their changes over time were not assessed.

Yazar Katkıları
Çalışma fikri/tasarımı: ST, EY
Veri toplama: ST, EY
Veri analizi ve yorumlama: ST, EY
Literatür tarama: ST, EY
Makalenin yazımı: ST, EY
Eleştirel inceleme: ST
Son onay ve sorumluluk: ST, EY

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