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Incidence, risk factors and prognostic impact of acute kidney injury on 30-day mortality after transcatheter aortic valve implantation

Transkateter aort kapak implantasyonu sonrası akut böbrek hasarının sıklığı, risk faktörleri ve 30-günlük mortalite üzerine prognostik etkisi

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ABSTRACT

Background: This study aims to investigate the incidence, risk factors, and prognostic impact of acute kidney injury on 30-day mortality after transcatheter aortic valve implantation.

Methods: Between October 2010 and July 2012, 42 consecutive patients (15 males, 27 females; mean age 77.7±6.3 years; range 58 to 91 years) who underwent transcatheter aortic valve implantation in a single center were retrospectively analyzed. Acute kidney injury was defined according to the second consensus report of the Valve Academic Research Consortium definitions. Baseline, peri- and post-procedural characteristics were compared between the patients with and without acute kidney injury.

Results: Fourteen patients (33.3%) patients developed acute kidney injury, and two of them (14.3%) required temporary hemodialysis. Twenty patients (47.6%) had chronic kidney disease before the procedure. The incidence of chronic kidney injury was 71.4% and 35.7% in the patients with and without acute kidney injury, respectively ($p<0.05$). There was no statistically significant difference in the baseline creatinine levels of the two groups. In multivariable analyses, independent predictors of post-procedural acute kidney injury were post-procedural abnormal leukocyte count (OR: 2.52; 95% CI: 1.17-5.42) and low platelet count (OR: 1.05; 95% CI: 1.01-1.10). The 30-day mortality was 11.9% in five patients. The mortality rate was higher in the patients with acute kidney injury than those without (28.6% vs 3.6%, $p<0.05$).

Conclusion: Acute kidney injury following transcatheter aortic valve implantation occurred in 33.3% of our patient population and was found to be associated with an increased 30-day mortality rate. We also found that post-procedural abnormal leukocyte count and low platelet count were the independent predictors of acute kidney injury.

Keywords: Acute kidney injury; aortic stenosis; prognosis; transcatheter aortic valve implantation.

ÖZ

Amaç: Bu çalışmada transkateter aort kapak implantasyonu sonrası akut böbrek hasarının sıklığı, risk faktörleri ve 30 günlük mortalite üzerine prognostik etkisi araştırıldı.

Çalışma planı: Ekim 2010 - Temmuz 2012 tarihleri arasında tek merkezde transkateter aort kapak implantasyonu uygulanan 42 ardışık hasta (15 erkek, 27 kadın; ort. yaş 77.7±6.3 yıl; dağılım 58-91 yıl) retrospektif olarak incelendi. Akut böbrek hasarı, ikinci Kapak Akademik Araştırma Konsorsiyumu konsensüs bildirisi tanımlarına göre yapıldı. Akut böbrek hasarı gelişen ve gelişmeyen hastalar arasında başlangıç, işlem sırası ve işlem sonrası özellikler karşılaştırıldı.

Bulgular: On dört hastada (%33.3) akut böbrek hasarı gelişti ve bunların ikisinde (%14.3) geçici hemodiyaliz gerekli oldu. Yirmi hastada (%47.6) işlem öncesinde kronik böbrek hastalığı vardı. Kronik böbrek hasarının görülme sıklığı, akut böbrek hasarı olan ve olmayan hastalarda sırasıyla %71.4 ve %35.7 idi ($p<0.05$). İki grubun başlangıç kreatinin düzeyleri arasında istatistiksel olarak anlamlı bir fark saptanmadı. Çok değişkenli analizlerde, anormal lökosit sayısı (OR: 2.52; 95% CI: 1.17-5.42) ve düşük trombosit sayısı (OR: 1.05; 95% CI: 1.01-1.10) işlem sonrası akut böbrek hasarının bağımsız öngördürücüleri idi. Otuz günlük mortalite oranı beş hastada %11.9 idi. Akut böbrek hasarı olan hastalarda, olmayanlara kıyasla, mortalite oranı daha yüksekti (%28.6'ya kıyasla %3.6, $p<0.05$).

Sonuç: Hasta popülasyonumuzun %33.3'ünde transkateter aort kapak implantasyonu sonrası akut böbrek hasarı gelişti ve bu da, artmış 30 günlük mortalite oranı ile ilişkili bulundu. Bununla birlikte, işlem sonrası anormal lökosit sayısı ve düşük trombosit sayısı akut böbrek hasarının bağımsız öngördürücüleri idi.

Anahtar sözcükler: Akut böbrek hasarı; aort darlığı; prognoz; transkateter aort kapak implantsayonu.

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Symptomatic severe aortic stenosis is an increasingly prevalent disease in elderly and has a poor prognosis, when treated medically.[1] Surgical aortic valve replacement (SAVR) is currently the standard of care and is generally accepted to alleviate symptoms and prolong survival. However, about 30 to 50% of the patients are unable to undergo SAVR due to non-referral, deemed to be inoperable or have a high risk for surgery by the cardiothoracic team or the patient refusal of SAVR.[2] Transcatheter aortic valve implantation (TAVI) is a newer and less invasive alternative approach which yields favorable results and low mortality in these high-surgical-risk patients.[3] However, due to the comorbidities in these patients, even TAVI is associated with a number of complications which may lead to impaired outcomes. These complications include impaired renal functions which increase the risk for acute kidney injury (AKI) following TAVI due to the hemodynamic changes during the procedure and the use of contrast agents. Several studies have investigated the incidence and predictors of AKI after TAVI.[4-14] Previous studies have shown that AKI occurs in about 11.7 to 41.7% of patients undergoing TAVI and has been associated with 2- to 5.47-fold increased risk of mortality.[5-10]

In the present study, we aimed to determine the incidence, predictors, and prognostic impact of AKI on 30-day mortality following TAVI.

PATIENTS AND METHODS

A total of 49 consecutive patients with severe aortic stenosis who underwent TAVI between October 2010 and July 2012 at Mehmet Akif Ersoy Thoracic and Cardivascular Surgery Training and Research Hospital either using the Medtronic CoreValve (Medtronic CoreValve, Irvine, CA, USA) or the Edwards SAPIEN (Edwards Lifesciences, Inc., Irvine, CA, USA) bioprosthetic valve were screened and 42 eligible patients (15 males, 27 females; mean age 77.7±6.3 years; range 58 to 91 years) were retrospectively analyzed. The study protocol was approved by the Istanbul Mehmet Akif Ersoy Thoracic and Cardivascular Surgery Training and Research Hospital Ethics Committee. A written informed consent was obtained from each patient. The study was conducted in accordance with the principles of the Declaration of Helsinki.

All patients had severe aortic stenosis and the New York Heart Association (NYHA) Class III-IV symptoms and were also at a high risk for surgery due to existing comorbidities. The decision for TAVI was rendered by a consensus at a meeting of the heart team, and preoperative risk was assessed on the basis of the European System for Cardiac Operative Risk Evaluation (EuroSCORE) or the Society of Thoracic Surgeons (STS) risk calculator systems.[15,16] In the absence of other contraindications to surgical valve replacement, high-risk status was defined as a logistic EuroSCORE >20% or an STS score >10%.

Exclusion criteria were as follows: severe left ventricular systolic dysfunction (<20%), acute myocardial infarction, severe coronary artery disease requiring revascularization, the presence of an active infection, and a life expectancy of less than 12 months due to non-cardiac causes.

Procedural details

The severity of aortic stenosis, aortic valve structure, and aortic root were evaluated by transthoracic echocardiography and transesophageal echocardiography. Multi-slice computed tomography and angiography were performed for the assessment of aortic root-arch calcification, diameters of the femoral and iliac arteries, and calcifications and tortuosities. Coronary arteries were evaluated before the procedure through standard coronary angiography. The patients were assigned to either the transfemoral (TF) or transapical (TA) approach depending on the condition and the size of the iliofemoral arteries and the degree of calcification. The patients were considered to be eligible for the TF approach, if their iliac and femoral arteries were at least 6 mm in diameter.

The TAVI procedure was performed in a sterile environment (catheterization laboratory) under general anesthesia. The femoral artery with its greater diameter and less tortuosity was selected. Two sheaths were placed in the contralateral femoral artery and femoral vein for placement of a pigtail catheter in the aorta and a pacemaker lead in the right ventricle, respectively. For the proper procedure, pre-implantation balloon dilatation was carried out after passing the native valve with a straight tip guide wire and an Amplatz left guide catheter. During balloon dilatation, ventricular tachycardia was induced by rapid ventricular pacing, providing an optimal reduction in cardiac output by creating transient cardiac standstill. This was usually achieved at a heart rate of 200 bpm. The aortic root and peripheral arteries were evaluated after the deployment of the valve by the aortography and peripheral angiography.

After the procedure, all patients were transferred to the intensive care unit and typically extubated within two to four hours. The post-procedural anti-platelet regimen consisted of clopidogrel 75 mg daily for six

months and aspirin 100 mg daily indefinitely. The patients with atrial fibrillation or other indications for anticoagulation received warfarin and aspirin without clopidogrel.

Definition of AKI

Acute kidney injury was defined according to the Valve Academic Research Consortium (VARC)-2 definitions as an absolute (<48 h) reduction in kidney function and as follows: *(i)* an absolute increase in the highest value of serum creatinine (SCr) ≥0.3 mg/dL (≥26.4 μmol/L) or *(ii)* a percentage increase in the highest value of SCr ≥50% (1.5-fold from baseline). The patients who developed AKI were classified according to the degree of the severity of AKI in Stage I (an increase in the SCr of 150-200% or increase of ≥0.3 mg/dL (≥26.4 μmol/L), Stage II (an increase in the SCr of 200-300%), or Stage III (an increase in the SCr of >300% or patients with a baseline SCr of ≥4.0 mg/dL (≥354 μmol/L) with an acute increase of ≥0.5 mg/dL (44 μmol/L) or requirement of renal replacement therapy [RRT] regardless of SCr level).[17] However, due to the incomplete data of urinary output and the heterogeneity of volume status and diuretic use in our study population, we did not include this variable in the classification.

Data collection and study endpoints

Medical records and our dedicated TAVI database were reviewed and the following information was collected: baseline and peri-procedural characteristics, comorbidities, laboratory (complete blood count and renal function) and echocardiographic parameters before and after TAVI. The primary endpoint was renal outcomes which included the incidence of AKI after TAVI procedure. Secondary endpoints were in-hospital and 30-day all-cause mortality and the independent risk factors for AKI.

The procedural success was defined as the expansion of the bioprosthetic valve in the proper position and its functioning with a tolerable degree of aortic insufficiency. All-cause mortality and cardiovascular mortality were recorded. Blood samples for complete blood count and chemistry (SCr and blood urea nitrogen [mg/dL]) were taken 24 hours before the procedure, immediately after the procedure, and once daily thereafter during hospitalization or up to seven days. The estimated glomerular filtration rate (eGFR) was calculated by the Modification of Diet in Renal Disease (MDRD) equation: eGFR= 186 × (plasma creatinine level [in mg/dL]$^{-}$1.154 × (age [in years])$^{-}$0.203, and for women, the product of this equation was multiplied by a correction factor of 0.742.[18] Chronic kidney disease (CKD) was defined as AN eGFR of <60 mL/min/1.73m^2 calculated by the MDRD formula.

In addition, anemia was defined according to the American College of Physicians and World Health Organization criteria as a hemoglobin level of <13 g/dL in men and of <12 g/dL in women.[19] Thrombocytopenia was defined as a platelet count of <140×109/L. Leukocyte counts <4.0 and >12 were considered abnormal. Throughout the study, the terms peri-procedural and post-procedural correspond to a time-frame of 24 and 72 h after the start of the procedure, respectively.

Statistical analysis

Statistical analysis was performed using the PASW for Windows version 17.0 software (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed in mean ± standard deviation (SD), while categorical variables were expressed in percentages (%). Initially, univariate analyses using the chi-square statistic for categorical variables and the t-test or Mann-Whitney U test for continuous variables were performed to identify pre-, peri- and post-procedural variables associated with AKI. Uni- and multivariate predictors of AKI were assessed by logistic regression analysis and odds ratios (ORs) were reported. A backward stepwise multivariate logistic regression model was carried out to assess the independent relationship between significant factors and AKI. Variables exhibiting a *p* value of <0.1 in the univariate analysis were included in the multivariate logistic regression model. For all tests, two-sided *p* values of <0.05 were considered statistically significant.

RESULTS

Of a total of 49 consecutive patients who underwent TAVI, seven patients were excluded due to death within the first 24 hours (n=4), requirement of immediate conversion to SAVR (n=2), and preoperative CKD requiring chronic dialysis (n=1). The procedure was performed mainly via TF approach in this study. A total of 41 patients (97.6%) received valve replacement via TF and only one patient (2.4%) via TA approach. Baseline, peri- and post-procedural clinical and echocardiographic characteristics of the study population are shown in Table 1. Acute kidney injury occurred in 14 patients (33.3%) and two of them (4.7%) required dialysis during index hospitalization.

In the univariate analysis, the patients with AKI had a higher rate of baseline CKD (71.4% vs 35.7%, p=0.029), post-procedural thrombocytopenia (71.4%

Table 1. Baseline, peri-procedural and post-procedural characteristics

	Study population (n=42)			AKI + (n=14, 33.3%)			AKI - (n= 28, 66.7%)			
Clinical characteristics	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			77.7±6.3			78.1±7.5			77.6±5.8	0.80
Gender										
Male	15	35.7		5	35.7		10	35.7		1.00
Body Mass Index (kg/m^2)			27.3±5.4			25.0±4.4			28.5±5.7	0.35
Logistic EuroSCORE (%)			24.2±7.7			24.9±8.2			23.9±7.7	0.96
Society of Thoracic Surgeons score (%)			14.7±6.7			14.1±7.0			15.0±6.6	0.68
New York Heart Association										0.19
Class III	21	50		5	23.8		16	76.2		
Class IV	21	50		9	42.8		12	57.2		
Coronary artery disease	30	71.4		8	57.1		22	78.6		0.16
Chronic obstructive pulmonary disease	29	69.0		10	71.4		19	67.9		0.81
Diabetes mellitus	12	28.6		1	7.1		11	39.3		0.036
Hypertension	32	76.2		11	78.6		21	75.0		0.79
Coronary artery bypass graft surgery	12	28.6		2	14.3		10	35.7		0.27
Pulmonary hypertension	22	52.4		7	50.0		15	53.6		0.82
Peripheral vascular disease	22	52.4		7	50.0		15	53.6		0.82
Cerebrovascular disease	3	7.1		2	14.3		1	3.6		0.25
Atrial fibrillation	10	23.8		5	35.7		5	17.9		0.25
Chronic Kidney disease	20	47.6		10	71.4		10	35.7		0.029
Glomerular filtration rate (mL/min/1.73 m^2)			65.3±24.9			51.4±17.2			72.2±25.4	0.096
Hemoglobin (g/dL)			11.5±2.1			11.4±2.3			11.5±2.0	0.38
Serum creatinine (mg/dL)			1.0±0.2			1.1±0.3			0.9±0.2	0.14
Echocardiographic data										
Baseline aortic valve area (cm^2)			0.6±0.11			0.6±0.1			0.6±0.1	0.68
Baseline peak gradient (mmHg)			87.9±18.2			90.2±12.5			86.8±20.6	0.45
Baseline mean gradient (mmHg)			53.9±10.5			53.3±6.9			54.2±11.9	0.63
Moderate or severe mitral regurgitation	8	19.0		3	21.4		5	17.9		0.78
Left ventricle ejection fraction			52.8±10.5			54.3±10.9			52.1±10.4	0.84
≤%40	6	14.3		1	7.1		5	17.9		0.64
Peri-procedural data										
Contrast amount (mL)			218.2±6.3			239.6±44.0			207.5±42.0	1.00
Implanted valve size (mm)			24.2±1.8			24.9±2.2			24.9±1.7	0.27
Valve type										0.33
Edwards Sapien	35	83.3		11	31.4		24	68.6		
Medtronic CoreValve	7	16.7		3	42.9		4	57.1		
Blood transfusion (%)	28	66.7		9	64.3		19	67.8		0.81
Red blood cell (U)			1.5±1.4			2.4±2.3			1.3±1.2	0.084
Post-procedural (third day)										
Platelet count (×10^9/L)			150.7±59.1			111.4±32.6			166.1±60.2	0.007
Thrombocytopenia (≤140×10^9/L)	23	54.8		10	71.4		13	46.4		0.002
Leukocyte count (×10^9/L)			9.4±2.6			10.8±2.7			8.8±2.3	0.025
Abnormal leucocyte count (≤4 or 12×10^9/L)	6	14.3		4	28.6		2	7.1		0.004
Paravalvalvular aortic regurgitation ≥2+	6	14.3		2	14.3		4	14.3		1.00
Postoperative aortic valve area (cm^2)			1.9±0.2			1.9±0.2			1.9±0.2	0.86
Peripheral vascular complication	6	14.3		4	28.6		2	7.1		0.15

SD: Standard deviation; EuroSCORE: European System for Cardiac Operative Risk Evaluation.

Table 2. Independent predictors of akut kidney injury after transcatheter aortic valve implantation in multivariable regression analysis

Variable	Odds ratio (95% CI)	*p*
Post-procedural thrombocytopenia	1.05 (1.01-1.10)	0.021
Post-procedural abnormal leukocyte count	2.52 (1.17-5.42)	0.022

CI: Confidence interval.

vs 46.4%, p=0.002) and post-procedural abnormal leukocyte count (28.6% vs 7.1%, p=0.004). They also received more peri-procedural red blood cell (RBC) transfusions (2.35 vs 1.32 Unit, p=0.084). Baseline rate of diabetes mellitus (DM) was lower in the patients with AKI (7.1% vs 39.3%, p=0.036).

Independent predictors of AKI in the multivariate logistic regression analysis were the post-procedural leukocyte count (OR: 2.52; 95% CI: 1.17-5.42, p=0.022) and post-procedural thrombocytopenia (OR: 1.05; 95% CI: 1.01-1.10, p=0.021). Although it was not statistically significant, there was a trend towards an increased risk of AKI with an increased amount of peri-procedural RBC transfusion (OR: 2.13; 95% CI: 0.83-5.48, p=0.11) (Table 2).

Overall 30-day mortality rate was 11.9% (n=5). It was significantly higher in the patients with AKI, compared to those without AKI (28.6% vs 3.6%, respectively, p=0.035) (Figure 1). The causes of death were sepsis (n=2), heart failure (n=1), and asystole (n=2). One of two patients with AKI requiring dialysis died within 30 days after TAVI. The 30-day mortality rate was higher in the patients who required dialysis, compared to those who did not, although it was not statistically significant (20.0% vs 2.7%, respectively, p=0.22).

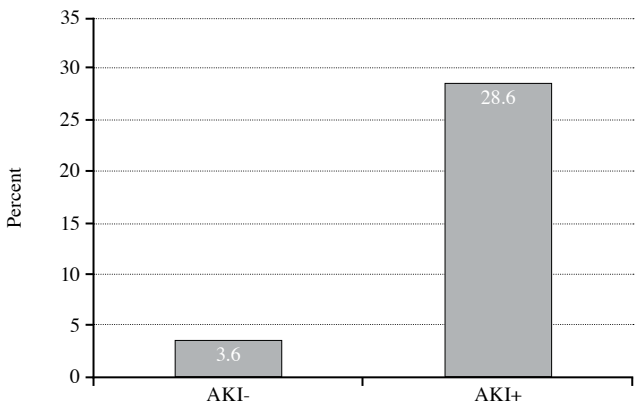

Figure 1. 30-day mortality rates according to development of akut kidney injury.

DISCUSSION

In the present study, AKI occurred in 14 patients (33.3%) following TAVI and two of them (4.7%) required hemodialysis. Independent predictors of AKI were the post-procedural abnormal leukocyte count and post-procedural thrombocytopenia. The patients with AKI had an increased risk of 30-day mortality by 28.6%, which was almost eight-fold higher than the patients without AKI (3.6%).

The occurrence of AKI following TAVI in this study is consistent with the findings of Aregger et al.,[4] Bagur et al.,[5] and Nuis et al.[8] who reported a frequency of 28%, 12%, and 19% in a series of 58, 213, and 118 patients, respectively. Of note, the definitions of AKI within the first two studies (RIFLE criteria) were almost similar to the VARC criteria herein reported, which are also in the study by Nuis et al.,[8] except for the time at which AKI was defined: 72 h in this study compared to 48 h.

Although renal histology was not available in these patients to establish the underlying pathophysiology of the AKI, the occurrence of AKI was most likely to be the cumulative effect of multiple factors: contrast induced nephropathy, hemodynamic instability due to intra- or post-procedural bleeding or during rapid ventricular pacing, calcified embolism after device manipulation over the stenotic aortic valve or cholesterol embolism as a result of catheter manipulation through the atherosclerotic vascular bed and RBC transfusion. In addition, high grade aortic stenosis with impaired cardiac output and the use of diuretics and other vasoactive agents might also reduce renal perfusion.

In our study, the univariate analysis showed that baseline SCr levels were not associated with AKI. However, Elhmidi et al.,[6] Alassar et al.,[14] and Strauch et al.[20] found that baseline SCr levels were independent predictors of AKI following TAVI.

Acute kidney injury is a well-known complication of angiography with the use of iodinated contrast media and is associated with significantly worse prognosis.[21] Similar to the previous findings,[4-9] baseline eGFR and the amount of contrast media used were not associated

with AKI in the univariate analysis in our study. The finding that AKI was not related to the amount of the contrast agent used suggests that other factors may be more important for the development of renal impairment in the population undergoing TAVI.

In addition, univariate analysis showed that DM and CKD (eGFR of <60 mL/min/1.73 m^2) were associated with AKI (p=0.036 and p=0.029, respectively); however, multivariate analyses did not confirm these findings. To the best of our knowledge, only in a study by Alassar et al.,[14] DM was found to be an independent predictor of AKI following TAVI. Wessely at al.[11] also evaluated the evolution of renal function, the incidence of AKI, and the need for RRT after TAVI in the patients with CKD. The authors found no significant differences between the patients with or without CKD in terms of the incidence of AKI and RRT. However, they observed an increase in the eGFR in 36.3% of the patients with CKD following the intervention. In another study, Kong et al.[12] reported a higher incidence of AKI in the patients with CKD, although it was not statistically significant (p=0.07). In the aforementioned study, renal functions of the patients who did not experience AKI were significantly improved immediately after TAVI and this improvement sustained up to 12 months after the procedure. This was attributed to the possible reversion of pathophysiological processes in type 2 cardiorenal syndrome (CRS) which describes the complex interplay between the heart and kidneys where acute or chronic dysfunction in one organ initiates and perpetuates the combined disorder of the two organs through a complex combination of hemodynamic and neurohormonal feedback mechanisms.[12] Type 2 CRS is characterized by chronic cardiac dysfunction, causing progressive CKD.[22]

Furthermore, RBC transfusion has been associated with an increased risk of AKI after TAVI in several reports.[4,5,8,11,12] In this study, the amount of peri-procedural RBC transfusion was found to be higher in the patients with AKI, although it was not statistically significant (OR: 2.13; 95% CI: 0.83-.48, p=0.11). In several studies, it has shown that the number of blood transfusions is an independent predictor of AKI after TAVI.[4,5,8,11,12] Complications causing bleeding after the procedure occur very frequently, which results in a higher incidence of blood transfusions as well as a higher incidence of renal hypoperfusion. Both these factors have been shown to be associated with AKI.[5,12] However, only the transfusion has been found to be an independent predictor of AKI. This detrimental effect of RBC transfusion may be due the red-cell storage: stored RBCs undergo progressive functional and structural changes, leading to a reduction in the RBC function and viability, and accumulate proinflammatory molecules and all these changes may favor renal dysfunction.[23]

Recent data have suggested a correlation between the systemic inflammatory response syndrome (SIRS) characterized by abnormal post-procedural leukocyte count and AKI.[4,7] Ischemia of the kidneys results in a release of inflammatory cytokines and subsequent inflammatory reaction characterized by abnormal leukocyte count. This inflammatory reaction may play an essential role in the development of AKI.[24] Transient hypoperfusion of the kidney occurs during the several steps of the TAVI procedure (during rapid pacing and BAV or as a consequence of vascular complications, and bleeding events), and this may lead to ischemia of the kidneys. In a recent study, occurrence of SIRS in TAVI patients was characterized by a significantly elevated release of interleukins 6 and 8 with subsequent increased the leukocyte counts, C-reactive protein, and procalcitonin.[25] It was also demonstrated that higher levels of interleukin 8 was associated with an increased rate of AKI following open heart surgery.[26] Abnormal leukocyte count as seen in the SIRS was an independent risk factor for AKI in the multivariate analysis in a study by Nuis et al.[8] Consistent with this finding, we also found that post-procedural abnormal leukocyte count was an independent risk factor for AKI in the multivariate analysis (OR: 2.52; 95% CI: 1.17-5.42, p=0.022).

In addition, thrombocytopenia developed in 54.8% of the patients and the incidence was significantly higher in the patients with AKI than those without AKI (71.4% vs 46.4%, p=0.002). Also, post-procedural thrombocytopenia was an independent risk factor for AKI in the multivariate analysis in our study (OR: 1.05; 95% CI: 1.01-1.10, p=0.021). Aregger et al.[4] also showed that post-procedural thrombocytopenia was an independent predictor of AKI after TAVI. Although the mechanism of thrombocytopenia following TAVI is unknown, our study and previous studies suggest that it is a relatively common finding and is associated with adverse outcomes including AKI.

On the other hand, small sample size and retrospective design and monocentric nature of the study are the main limitations.

In conclusion, based on our study results, acute kidney injury following transcatheter aortic valve implantation is associated with increased mortality. In addition, abnormal post-procedural leukocyte count and post-procedural thrombocytopenia are independent

predictors of acute kidney injury. However, further studies are needed to elucidate the mechanisms of post-procedural acute kidney injury and whether improved peri-procedural management reduces the incidence of acute kidney injury.

Declaration of conflicting interests

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