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Changes in kidney function among patients undergoing transcatheter aortic valve replacement

Charat Thongprayoon[1], Wisit Cheungpasitporn[1], Wonngarm Kittanamongkolchai[1], Narat Srivali[2], Kevin L Greason[3], Kianoush Kashani[1,2*]

[1]Division of Nephrology and Hypertension, Mayo Clinic, Rochester, MN, USA
[2]Division of Pulmonary and Critical Care Medicine, Mayo Clinic, Rochester, MN, USA
[3]Division of Cardiovascular Surgery, Department of Medicine, Mayo Clinic, Rochester, MN, USA

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ABSTRACT

Introduction: The patients selected for transcatheter aortic valve replacement (TAVR) usually have a high prevalence of chronic kidney disease (CKD). Little is known regarding the impact of TAVR on changes in renal function.

Objectives: This study aimed to assess the change in estimated glomerular filtration rate (eGFR) after TAVR.

Patients and Methods: Adult patients with aortic stenosis (AS) who underwent TAVR between January 2008 and June 2014, at Mayo Clinic, Rochester, MN. Changes in renal function during six months follow-up were evaluated.

Results: Of 386 patients undergoing TAVR, 106 (28%) developed acute kidney injury (AKI). There was significant reduction in eGFR at the hospital discharge and at 6 months post-TAVR in AKI patients in comparison with non-AKI individuals, (mean differences -7.1; 95% CI -9.8, -4.3 mL/min/1.73 m^2, $P<0.001$ and -4.2; 95% CI -7.1, -1.3 mL/min/1.73 m^2, $P=0.005$, respectively). In non-AKI patients with baseline eGFR ≥60 mL/min/1.73 m^2, there was a modest decrease in eGFR at 6 month (mean difference -4.0; 95% CI -6.4, -1.6 ml/min/1.73 m^2, $P=0.001$). Conversely, in non-AKI patients with eGFR 30-59 and <30 mL/min/1.73 m^2, there was an increase in eGFR at 6 months (mean difference 2.4; 95% CI 0.8, 2.4 mL/min/1.73 m^2; $P=0.004$ and 5.3; 95% CI 2.8, 7.8 mL/min/1.73 m^2; $P=0.001$, respectively).

Conclusion: In patients undergoing TAVR, change in renal function is significantly related to pre-procedural kidney function. AKI significantly impacts renal function at six months post TAVR. CKD patients who do not develop AKI, may benefit from TAVR by an increase in eGFR at six months.

Implication for health policy/practice/research/medical education:
Impact of transcatheter aortic valve replacement (TAVR) on renal function especially in patients with chronic kidney disease (CKD) is unclear. In this current study, we investigated the change in eGFR following TAVR in non-advanced CKD patients with severe aortic stenosis (AS) stratified by levels of eGFR (≥ 60, 30-59, and <30 mL/min/1.73m^2). Our findings were as follows; 1) acute kidney injury (AKI) significantly decreased eGFR at 6 months after TAVR and 2) In patients with no AKI after TAVR, individuals with CKD (particularly eGFR <30 mL/min/1.73m^2) had a significant increase in eGFR (decrease in SCr) at 6 months.

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Introduction

Transcatheter aortic valve replacement (TAVR) has globally expanded in the past decade and now is acknowledged as a standard approach for patients who have severe aortic stenosis (AS) deemed inoperable with high surgical risk for open-heart aortic valve replacement surgery (1-6). In addition, the recently published studies also suggest that patient selection for TAVR is evolving toward treating lower surgical risk patients (7-9). Thus, to date, more than 200 000 TAVR procedures have been

***Corresponding author**: Kianoush B Kashani, Email: kashani.kianoush@mayo.edu

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performed worldwide (3,10).

Despite increasing evidence of treating intermediate surgical-risk patients (7-9), many patients elected for TAVR commonly have renal insufficiency (11,12), one of important predictors for acute kidney injury (AKI) development (13). In addition, AKI following TAVR is very prevalent, varying from 15% up to 57% (10,11,14-16). Although patients with chronic kidney disease (CKD) carry a higher risk of developing AKI, improvement in estimated glomerular filtration rate (eGFR) following surgical aortic valve replacements (SAVRs) has been demonstrated in patients with CKD after relief of severe aortic valve diseases (17,18).

Objectives

Little is known regarding the impact of TAVR on renal function especially in patients with CKD. Thus, we conducted this retrospective study to evaluate the change in eGFR after TAVR.

Patients and Methods

We conducted a retrospective observational study at Mayo Clinic Hospital, a quaternary referral hospital in Rochester, Minnesota. Adult patients (age ≥18 years) with AS, who underwent TAVR between January 1st, 2008 and June 30th, 2014 were enrolled. Exclusion criteria were: (*a*) patients who had advanced CKD stage 5 (eGFR <15 mL/min/1.73 m^2), (*b*) patients who received dialysis (≤14 days prior to TAVR), and (*c*) patients without research authorization.

Clinical, laboratory, pre- and post-procedural data were obtained from our institutional electronic medical record system. The Society of Thoracic Surgeons' (STS) adult cardiac surgery risk score was calculated for each patient as a surrogate for operative mortality risk (19-21). The eGFR was calculated using the CKD epidemiology collaboration equation (22). Primary outcomes were the changes in eGFR after TAVR; at the day of hospital discharge and at 6 months. We stratified patients based on AKI and CKD stages. AKI after TAVR was defined by an increase in serum creatinine (SCr) of ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours after TAVR or a relative increase of ≥50%) of the KDIGO definition (23).

Ethical issues

1) The research followed the tenets of the Declaration of Helsinki; 2) The Institutional Review Board affirmed our study and informed consent for patients with research authorization was waived; and 3) This study was approved by the Ethics Committee of Mayo Clinic.

Statistical analysis

To identify the differences in clinical characteristics between patients with and without AKI after TAVR, student's *t* test was used for continuous variables and the chi-squared test, or Fisher's exact test was used for categorical variables, as appropriate. The changes in eGFR and SCr before and after TAVR were tested using paired *t* test. A two-sided *P* value of <0.05 was considered statistically significant. All analyses were performed using JMP statistical software version 10 (SAS, Cary, NC).

Results

A total of 390 TAVR procedures for AS were conducted during the study time period. Three were excluded due to advance CKD stage 5 or receiving dialysis (≤14 days before TAVR). One patient had no research authorization and therefore was excluded. A total of 386 patients were included in the analysis.

Patient characteristics

In our cohort, the mean age was 81 ± 8 years, and 56% were male. Ninty-seven percent of patients were Caucasian. Mean baseline eGFR of patients undergoing TAVR was 55 ± 21 mLmin/1.73 m^2. The mean STS adult cardiac surgery risk score was 8.6 ± 6.3. Most of the TAVR procedures were performed via transfemoral (51%), followed by transapical (44%), and transaortic (5%) approaches. Table 1 demonstrated baseline characteristics of the enrolled cohort.

Change in kidney function at in patients with or without AKI after TAVR

Of 386 patients undergoing TAVR, 106 (28%) developed AKI. Overall baseline eGFR was 48 ± 22 mL/min/1.73 m^2 in patients who developed AKI after TAVR and 58 ± 19 mL/min/1.73 m^2 in those who did not have AKI. In patients with AKI, there was significant reduction in eGFR at the hospital discharge, which remained significantly reduced at 6 months post TAVR (mean differences -7.1; 95% CI -9.8, -4.3 mL/min/1.73 m^2, $P < 0.001$ and -4.2; 95% CI -7.1, -1.3 mL/min/1.73 m^2, $P = 0.005$, respectively), shown in Table 2.

In non-AKI patients with baseline eGFR ≥60 mL/min/1.73 m^2, there was a modest decrease in eGFR at 6 month after TAVR (mean difference -4.0; 95% CI -6.4, -1.6 mL/min/1.73 m^2, $P = 0.001$). Conversely, in non-AKI patients with eGFR 30-59 and <30 mL/min/1.73 m^2, there was an increase in eGFR at 6 months (mean difference 2.4; 95% CI 0.8, 4.0 ml/min/1.73 m^2; $P = 0.004$ and 5.3; 95% CI 2.8, 7.8 mL/min/1.73 m^2; $P = 0.001$, respectively), shown in Table 2.

Change in SCr after TAVR was also shown in Table 3. Consistent with change in eGFR after TAVR, in patients with AKI, there was significant increase in SCr at the hospital discharge, which remained significantly elevated at 6 months after TAVR (mean differences 0.27; 95% CI 0.18, 0.36 mg/dL, $P < 0.001$ and 0.17; 95% CI 0.07, 0.26 mg/dL, $P = 0.001$, respectively). In non-AKI patients with baseline eGFR ≥60 mL/min/1.73 m^2, there was a modest increase in SCr at 6 months after TAVR (mean differences 0.06; 95% CI 0.02, 0.09 mg/dL, $P = 0.001$). Conversely, in non-AKI patients with eGFR <30 mL/min/1.73 m^2, there was a decrease in SCr at 6 months (mean differences -0.25;

Table 1. Clinical characteristics and outcomes of TAVR

Characteristics	All (n = 386)	AKI (n = 106)	No AKI (n = 280)	*P* value
STS risk score[a]	8.6 ± 6.3	9.7 ± 6.0	8.1 ± 6.4	0.02
Age (year)[a]	81 ± 8	82 ± 7	81 ± 8	0.22
Male sex[b]	217 (56)	62 (58)	155 (55)	0.58
White[b]	374 (97)	101 (95)	273 (98)	0.26
Body Mass Index (kg/m^2)[a]	30.4 ± 7.5	30.3 ± 7.0	30.4 ± 7.6	0.96
Baseline eGFR (mL/min/1.73 m^2)[a]	55 ± 21	48 ± 22	58 ± 19	<0.001
NYHA class III-IV[b]	335 (87)	97 (92)	238 (85)	0.09
Comorbidity				
Diabetes mellitus[b]	157 (41)	51 (48)	106 (38)	0.07
Hypertension[b]	349 (90)	100 (94)	249 (89)	0.11
Dyslipidemia[b]	346 (90)	98 (92)	248 (89)	0.26
Myocardial infarction[b]	139 (36)	41 (39)	98 (35)	0.50
Congestive heart failure[b]	223 (58)	68 (64)	55 (155)	0.12
Stroke[b]	110 (28)	30 (28)	80 (29)	0.96
Peripheral vascular disease[b]	226 (59)	68 (64)	158 (56)	0.17
Anemia[b]	10 (3)	4 (4)	6 (2)	0.37
Chronic lung disease[b]	240 (62)	64 (60)	176 (63)	0.65
Smoking within 1 year[b]	11 (3)	3 (3)	8 (3)	0.99
Prior cardiac intervention				
PCI[b]	199 (52)	61 (58)	138 (49)	0.15
Cardiac surgery[b]	181 (47)	44 (42)	137 (49)	0.19
CABG[b]	167 (43)	43 (41)	124 (44)	0.51
Valve surgery[b]	84 (22)	28 (26)	56 (20)	0.17
Aortic valve surgery[b]	10 (3)	1 (1)	9 (3)	0.21
Echocardiographic finding				
Ejection fraction[a]	56 ± 13	54 ± 13	57 ± 13	0.08
Aortic valve gradient[a]	48 ± 14	46 ± 14	49 ± 14	0.07
Aortic valve insufficiency[b]	209 (54)	58 (55)	151 (54)	0.89
Mitral valve dysfunction[b]	300 (78)	81 (76)	219 (78)	0.70
Preoperative medication				
ACEI/ARB[b]	157 (41)	42 (40)	116 (41)	0.75
Beta-blocker[b]	266 (69)	72 (68)	194 (69)	0.80
Statin[b]	281 (73)	74 (70)	207 (74)	0.42
Aspirin[b]	284 (74)	71 (67)	213 (76)	0.07
Normal sinus rhythm[b]	280 (73)	71 (67)	209 (75)	0.13
Elective surgery[b]	368 (96)	98 (92)	270 (96)	0.10
Arterial approach				<.001
Transfemoral[b]	195 (51)	36 (34)	159 (57)	
Transapical[b]	171 (44)	65 (61)	106 (38)	
Transaortic[b]	20 (5)	5 (5)	15 (5)	
Surgery duration (min)[a]	128 ± 52	132 ± 63	126 ± 47	0.39
RBC transfusion needed[b]	129 (33)	48 (45)	81 (29)	0.002
Contrast amount (mL)[a]	94 ± 56	96 ± 57	93 ± 55	0.32
Intra-aortic balloon pump[b]	7 (2)	5 (5)	2 (1)	0.02

[a]Continuous variables are reported as mean±standard deviation; [b]Categorical variables are reported as count (percentage).
Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AKI, acute kidney injury; ARB, angiotensin II receptor blocker; CABG, coronary artery bypass graft surgery; eGFR, estimated glomerular filtration rate; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; RBC, red blood cell; STS, Society of Thoracic Surgeons; TAVR, transcatheter aortic valve replacement.

95% CI -0.36, -0.14 mg/dL, $P<0.001$).

Discussion

Previous studies have demonstrated an association between CKD and poor outcomes in patients undergoing TAVR (24,25). However, data on the change of renal function after relief of severe AS by TAVR are limited. In the current study, we examined the change in eGFR following TAVR in non-advanced CKD patients with severe AS stratified by levels of eGFR (≥ 60, 30-59, and <30 mL/min/1.73 m^2). Our findings were as follows; 1) AKI significantly decreased eGFR at six months after TAVR and 2) In patients with no AKI after TAVR, individuals with CKD (particularly eGFR <30 mL/min/1.73 m^2) had a significant increase in eGFR (decrease in SCr) at 6 months. Although the precise underlying mechanisms of recovery of kidney function among CKD patients after TAVR is still unclear, we believe that the increase in eGFR is likely

Table 2. Change in eGFR at hospital discharge and 6 months after TAVR

Group	Baseline	Hospital discharge	Mean difference (95% CI)	*P* value	6 months after TAVR	Mean difference (95% CI)	*P* value
All	55 ± 21	55 ± 22	0.2 (-1.0, 1.4)	0.78	54 ± 21	-1.4 (-2.7, -0.1)	0.03
GFR group							
≥ 60	77 ± 11	75 ± 14	-1.9 (-4.0, 0.1)	0.07	72 ± 15	-5.0 (-7.4, -2.7)	<0.001
30-59	44 ± 8	46 ± 14	2.0 (0.3, 3.6)	0.02	45 ± 14	0.8 (-0.9, 2.4)	0.36
<30	24 ± 4	24 ± 8	-0.4 (-2.6, 1.8)	0.73	27 ± 9	2.3 (-0.2, 4.7)	0.06
AKI status							
Non-AKI	58 ± 19	61 ± 20	2.9 (1.8, 4.1)	<0.001	58 ± 19	-0.4 (-1.8, 1.0)	0.62
≥ 60	76 ± 11	78 ± 13	1.3 (-0.5, 3.1)	0.16	72 ± 14	-4.0 (-6.4, -1.6)	0.001
30-59	45 ± 8	49 ± 12	4.3 (2.8, 5.9)	<0.001	47 ± 13	2.4 (0.8, 4.0)	0.004
<30	26 ± 3	31 ± 4	4.1 (1.7, 6.5)	0.003	32 ± 4	5.3 (2.8, 7.8)	0.001
AKI	48 ± 22	41 ± 20	-7.1 (-9.8, -4.3)	<0.001	43 ± 22	-4.2 (-7.1, -1.3)	0.005

Abbreviations: AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; TAVR, transcatheter aortic valve replacement.

Table 3. Change in SCr at hospital discharge and 6 months after TAVR

Group	Baseline	Hospital discharge	Mean difference (95% CI)	*P* value	6 months after TAVR	Mean difference (95% CI)	*P* value
All	1.2 ± 0.5	1.3 ± 0.6	0.03 (0.001, 0.07)	0.04	1.3 ± 0.5	0.05 (0.02, 0.08)	0.004
GFR group							
≥ 60	0.9 ± 0.2	0.9 ± 0.2	0.03 (-0.001, 0.05)	0.06	0.9 ± 0.2	0.07 (0.04, 0.11)	<0.001
30-59	1.4 ± 0.3	1.4 ± 0.4	0.01 (-0.04, 0.06)	0.69	1.4 ± 0.4	0.04 (-0.01, 0.08)	0.11
<30	2.1 ± 0.4	2.3 ± 0.8	0.19 (-0.006, 0.39)	0.06	2.1 ± 0.8	-0.003 (-0.21, 0.20)	0.98
AKI status							
Non-AKI	1.2 ± 0.4	1.1 ± 0.4	-0.06 (-0.08, -0.04)	<0.001	1.2 ± 0.4	0.003 (-0.02, 0.03)	0.85
≥ 60	0.9 ± 0.2	0.8 ± 0.2	-0.02 (-0.04, 0.004)	0.10	0.9 ± 0.2	0.06 (0.02, 0.09)	0.001
30-59	1.3 ± 0.3	1.3 ± 0.3	-0.08 (-0.11, 0.04)	<0.001	1.3 ± 0.3	-0.02 (-0.06, 0.02)	0.23
<30	2.0 ± 0.4	1.7 ± 0.4	-0.19 (-0.29, -0.09)	0.001	1.6 ± 0.4	-0.25 (-0.36, -0.14)	<0.001
AKI	1.4 ± 0.6	1.7 ± 0.8	0.27 (0.18, 0.36)	<0.001	1.6 ± 0.7	0.17 (0.07, 0.26)	0.001

Abbreviations: AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; TAVR, transcatheter aortic valve replacement, SCr, serum creatinine.

attributed to an improvement in the cardiac forward flow after relief of severe AS resulting in a higher organ perfusion including kidneys (17,26). Also, improvement in a right heart function can lead to a decrease in renal venous congestion (17,27). As our finding of an increase in eGFR in CKD patients after TAVR, improvement in eGFR following SAVR has been demonstrated (17,18). Thus, improvement in cardiac functions after TAVR likely plays a significant role in an improvement in kidney function. In patients with advanced CKD or dialysis dependence, studies have shown a higher rate of early and late mortality following TAVR (28,29). Despite having poorer outcomes after TAVR compared with non-dialysis patients, TAVR is comparable with SAVR in ESRD patients on dialysis based on a propensity-matched comparison of all Medicare fee-for-service patients undergoing TAVR or SAVR (29). Data on renal function change after TAVR in advanced CKD (nondialysis stage 5 CKD) are limited. Unfortunately, the proportion of nondialysis stage 5 CKD undergoing TAVR at our institution during the study period was very small and thus we did not enroll in our study. Interestingly, a case of reversal of end-stage renal disease in a patient after TAVR was reported (30). Future studies are required to assess renal function change in this high-risk patient population.

Conclusion

In conclusion, our study demonstrates that change in eGFR is significantly related to baseline kidney function of patients undergoing TAVR. AKI significantly reduces eGFR at 6 months post TAVR. Without AKI after TAVR, patients with CKD, particularly baseline eGFR between 15 and 30 mL/min/1.73 m^2 may benefit from TAVR by an improvement in eGFR at 6 months.

Limitations of the study

There are several limitations to our study. First, our study has a retrospective observational design and patients in our center are predominantly Caucasian populations, conceivably causing selection bias and restricting the generalizability of our findings. Second, the cause of a slight reduction in eGFR (approximately -4.0 mL/min/1.73 m^2) at 6 months in non-AKI patients with baseline eGFR ≥ 60 mL/min/1.73 m^2 is unclear. Calculating eGFR based on SCr has a few limitations (31,32). It is possible that patients after TAVR had improved function status and muscle mass, resulting in higher SCr levels (33). Unfortunately, our data regarding body mass index (BMI) at 6 months follow-up are limited. Future studies with a more accurate assessment of GFR are needed.

Authors' contribution

CT, WC, WK, and NS performed data acquisition for the cohort, statistical analysis, and participated in initial manuscript creation. KLG and KK conceived of the study, participated in design and coordination, performed data analysis, and drafted and edited the manuscript. KK supervised the project. All authors read and approved the manuscript.

Conflicts of interest

The authors declare that they have no conflicting interest.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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