Journal of the Society for Cardiovascular Angiography & Interventions 1 (2022) 100038

Contents lists available at ScienceDirect

Journal of the Society for Cardiovascular Angiography & Interventions

journal homepage: www.jscai.org

Original Research

Renal Function–Based Contrast Threshold Predicts Kidney Injury in Transcatheter Aortic Valve Replacement

Sarah K. Gualano, MD, MBA [a,*], Milan Seth, MS [a], Hitinder S. Gurm, MBBS [a], Devraj Sukul, MD, MSc [a], Stanley J. Chetcuti, MD [a], Himanshu J. Patel, MD [b], William Merhi, DO [c], Charles Schwartz, MD [d], William W. O'Neill, MD [e], Francis Shannon, MD [f], P. Michael Grossman, MD [a]

[a] Division of Cardiovascular Medicine, Department of Internal Medicine, University of Michigan, Ann Arbor, Michigan
[b] Department of Cardiac Surgery, University of Michigan, Ann Arbor, Michigan
[c] Spectrum Health, Grand Rapids, Michigan
[d] Elliott Estes Heart Institute, St. Joseph Mercy, Oakland, Michigan
[e] Division of Cardiology, Department of Medicine, Henry Ford Hospital, Detroit, Michigan
[f] Beaumont Health, Royal Oak, Michigan

A B S T R A C T

Background: Acute kidney injury (AKI) after contrast-guided interventions is associated with adverse outcomes, but the role of contrast in the context of renal function is less well described for patients undergoing transcatheter aortic valve replacement (TAVR).

Methods: Patients from the Michigan TAVR registry between January 2016 and December 2019 were included. AKI was defined using Valve Academic Research Consortium 2 definitions. An integer cut point for the ratio of contrast volume (CV) to renal function (estimated glomerular filtration rate [eGFR]) as a predictor of AKI was calculated.

Results: Of 7112 cases, AKI occurred in 629 (8.8%) patients. Unadjusted mortality was higher among patients with AKI (32.5% vs 9.0%, $P < .0001$). AKI remained significantly associated with the risk of mortality after multivariable adjustment (hazard ratio = 4.50, $P < .001$). Procedural characteristics associated with AKI included CV/eGFR >2 (adjusted odds ratio [aOR] = 1.36, $P = .003$, 95% CI = 1.10-1.67), CV/eGFR >3 (aOR = 1.38, $P = .009$, 95% CI = 1.09-1.77), and use of general anesthesia (aOR = 1.67, $P < .0001$, 95% CI = 1.38-2.03).

Conclusions: CV in the context of renal function administrated during TAVR is a robust tool to predict AKI. AKI after TAVR is associated with an increased risk of mortality. Incorporation of thresholds of >2× and > 3× eGFR into procedural planning should be considered as a quality initiative.

Introduction

Acute kidney injury (AKI) after contrast-guided procedures such as percutaneous coronary intervention (PCI) and peripheral vascular intervention (PVI) has been associated with adverse outcomes including mortality.[1,2] AKI after cardiac surgery is also independently associated with mortality. Reports of AKI in patients undergoing transcatheter aortic valve replacement (TAVR) have demonstrated increased risk of mortality; however, the role of contrast volume (CV) in the development of AKI after TAVR remains unclear.[3–8]

A more refined risk predictor of AKI may not simply be absolute CV used, but rather CV administered in the context of baseline renal function. Though prior studies have focused on CV as a continuous variable, grouping patients into ranges of contrast administered based on renal function with subsequent analysis of CV as a categorical variable may be more informative. CV greater than 3 times creatinine clearance was shown to be associated with increased risk of contrast nephropathy in a large registry of patients undergoing percutaneous coronary and peripheral arterial interventions.[1,9] It remains unknown if a CV ratio threshold may predict AKI after TAVR. The purpose of this study was to

Abbreviations: AKI, acute kidney injury; CV, contrast volume; GFR, glomerular filtration rate; MITAVR, Michigan Transcatheter Aortic Valve Replacement Registry; OR, odds ratio; PCI, percutaneous coronary intervention; PVI, percutaneous vascular intervention; STS, Society of Thoracic Surgeons; TAVR, transcatheter aortic valve replacement; TVT, transcatheter valve therapy.

Keywords: Transcatheter aortic valve replacement; valvular heart disease; mortality; acute kidney injury; contrast nephropathy.

* Corresponding author.
E-mail address: sgualano@med.umich.edu (S.K. Gualano).

https://doi.org/10.1016/j.jscai.2022.100038
Received 19 November 2021; Received in revised form 24 February 2022; Accepted 28 February 2022

investigate the incidence, risk factors, survival implications, and role of contrast in the development of AKI after TAVR in contemporary practice using a statewide registry.

Methods

Registry design and population

The Michigan Transcatheter Aortic Valve Replacement (MITAVR) registry is a regional, multihospital, physician-led collaboration between the Michigan Society of Thoracic and Cardiovascular Surgeons and Blue Cross and Blue Shield of Michigan Cardiovascular Consortium (BMC2). MITAVR is designed to improve the quality of care and outcomes of patients undergoing TAVR within Michigan at the nonfederal participant hospitals. Data on consecutive patients undergoing TAVR at participating programs are collected through the Society of Thoracic Surgeons/American College of Cardiology (STS/ACC) Transcatheter Valve Therapy (TVT) registry using standardized data forms and endpoints as defined in the TVT database. Data are then downloaded and analyzed by MITAVR. Data quality and completeness are confirmed by TVT and enhanced accuracy of data through annual on-site audits at all participating MITAVR hospitals. Participation in the MITAVR registry for each hospital has either been approved by or been waived at local institutional review boards as part of continuous clinical care quality improvement initiatives.

Since the goal of this study was focused on AKI in a contemporary patient population with current generation valves, cases prior to January 2016 were excluded to avoid confounding the analysis.

Study endpoints

Preprocedural creatinine (the creatinine value closest to the time of the procedure), peak creatinine, and discharge creatinine were recorded in accordance with TVT definitions.[10] Renal function beyond discharge was not measured, as the purpose of this study was to explore the relationship between in-hospital AKI and mortality events. The estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration formula.[11] The ratio of CV (mL) to eGFR (mL/min) was calculated for each patient.

AKI was defined as a creatinine increase to at least stage 1 of the Acute Kidney Injury Network classification system in accordance with Valve Academic Research Consortium 2 definitions.[12] At a minimum, patients must have an increase in serum creatinine compared with baseline greater than 150% or an increase of $\geq$0.3 mg/dL. Urine output was not available and thus not used to categorize patients. Postdischarge mortality at 90 days and 1 year was included as a secondary endpoint. Patients on dialysis or with missing creatinine values were excluded from the analysis.

Statistical analysis

Baseline clinical characteristics, procedural characteristics, and mortality up to 1 year were compared between those who did and did not develop AKI. *t* tests were used to compare continuous variables, and χ^2 tests were used for categorical variables. Cox regression was used for adjusted mortality comparisons.

Analysis of the CV/eGFR ratio as a categorical variable rather than a continuous variable was performed with the goal of determining a procedural threshold for prediction of AKI as in prior PCI and PVI studies.[1,9] The ROC01 and Youden methods were used for determining an integer cut point for the CV/eGFR metric. This cut point and the PCI and PVI threshold of 3 were then used to predict the odds of developing AKI. Logistic regression models were used to assess the association of CV on the risk of AKI, adjusting for baseline patient clinical and demographic characteristics (Supplemental Table 1).

Baseline patient and procedural characteristics were chosen from many candidate predictors for inclusion in regression models using bidirectional stepwise selection using the Akaike information criteria. CV and use of general anesthesia were included in the analysis as these variables were considered modifiable. Use of nonfemoral access was not included as no reasonable operator would choose to forego transfemoral access if that was an option. Cardiopulmonary bypass was also not used as a modifiable variable as the need for mechanical support may arise from a variety of intraoperative complications and thus may not be a modifiable procedural variable. Statistical analysis was performed with R 4.0.5 software.

Results

Study cohort and procedural outcomes

From January 2016 to December 2019, 7432 consecutive TAVR cases performed in Michigan were included in the registry. Patients on dialysis prior to the procedure (n = 268; 3.6%) or with missing creatinine values (n = 72; 1.0%) were excluded. The remaining 7112 patients were included in the analysis. The mean patient age was 79.6 ($\pm$8.8) years, and 3783 (53.2%) patients were male. The mean STS-predicted risk of mortality score was 5.8% (SD = 4.2%). The median follow-up was 345 (interquartile range: 37-379) days. There were 572 deaths (8.0%) within 1 year, with 82 (1.2%) in-hospital deaths.

Acute kidney injury

AKI occurred in 629 (8.8%) patients. Demographic, clinical, and procedural characteristics of patients with and without AKI included in the analysis are represented in Table 1. Patients with AKI had higher unadjusted rates of acute heart failure (52.5% vs 46.8%, P = .007), peripheral arterial disease (42.6% vs 33.7%, P < .001), atrial fibrillation/flutter (44.1% vs 38.8%, P = .011), diabetes (55.2% vs 37.9%, P < .001), and severe lung disease (17.4% vs 9.7%, P < .001), lower baseline eGFR (mL/min per 1.73 m^2) (21.6% vs 19.1%, P < .001), lower ejection fraction (54.6% vs 55.8%, P = .027), lower mean aortic valve gradient (40.8 mm Hg vs 42.4 mm Hg, P .008), nontransfemoral approach (88.6% vs 95.3%, P < .001), general anesthesia (52.1% vs 35.5%, P < .001), CV (63.4 mL vs 54.3 mL, P = .005), and cardiopulmonary bypass used (2.1% vs 0.1%, P < .001).

After multivariable analysis, patient characteristics that remained statistically associated with the odds of developing AKI included preprocedural creatinine (adjusted odds ratio [aOR] = 1.64 per increase of 1 mg/dL, P < .001), albumin (aOR = 0.62, P < .001), the Society of Thoracic Surgeons Predictive Risk of Mortality score (aOR = 1.04, P < .001), 1), prior coronary artery bypass graft (aOR = 0.69, P = .017), prior aortic valve procedure (aOR = 0.57, P < .001), peripheral arterial disease (aOR = 1.30, P = .01), tobacco use (aOR = 0.65, P = .049), diabetes (aOR = 1.49, P < .001), home oxygen use (aOR = 1.41, P = .021), preprocedural hemoglobin (aOR = 0.88, P < .001), severe tricuspid valve regurgitation (compared to no regurgitation) (aOR = 2.32, P < .001), and number of diseased coronary arteries (aOR for 1 vs none: 1.40, P = .020) (Table 2).

Contrast volume

CV was recorded for 6578 patients (92.5%) in the cohort, with 558 (8.48%) experiencing AKI. Among those with recorded CV values, logistic regression models were utilized to assess the impact of CV on incidence of AKI, adjusting for the GFR and other baseline patient clinical and demographic characteristics. A predictive model was used to determine the effects of the ratio of CV/eGFR on the likelihood of developing

Table 1. Univariate patient and procedural factors associated with acute kidney injury

	All patients, N = 7112	No AKI, n = 6483	AKI, n = 629	*P*-value
Patient characteristics				
Age, y	79.6 ± 8.8	79.6 ± 8.7	79.1 ± 9.2	.141
Male	3783 (53.2)	3457 (53.3)	326 (51.8)	.499
Prior coronary intervention	2598 (36.5)	2349 (36.2)	249 (39.6)	.099
Prior coronary bypass surgery	1484 (20.9)	1352 (20.9)	132 (21.0)	.984
Acute heart failure	3364 (47.4)	3034 (46.8)	330 (52.5)	.007
Prior stroke	859 (12.1)	772 (11.9)	87 (13.8)	.179
Prior vascular disease	2448 (34.5)	2181 (33.7)	267 (42.6)	<.001
Atrial fibrillation/flutter	2791 (39.3)	2515 (38.8)	276 (44.1)	.011
Diabetes mellitus	2804 (39.4)	2457 (37.9)	347 (55.2)	<.001
Severe lung disease	737 (10.4)	628 (9.7)	109 (17.4)	<.001
Estimated GFR, mL/min/1.73 m^2	59.6 ± 19.5	60.5 ± 19.1	51.1 ± 21.6	<.001
Renal insufficiency (Cr > 2 mg/dL)	271 (3.8)	195 (3.0)	76 (12.1)	<.001
Ejection fraction, %	55.7 ± 13.0	55.8 ± 13.0	54.6 ± 13.8	.027
Body mass index, kg/m^2	30.1 ± 12.3	30.0 ± 12.7	30.9 ± 7.9	.089
Mean aortic valve gradient, mm Hg	42.2 ± 13.9	42.4 ± 13.9	40.8 ± 13.8	.008
Aortic valve area, cm^2	0.70 ± 0.26	0.70 ± 0.27	0.70 ± 0.23	.956
STS mortality risk score, %	5.82 ± 4.20	5.63 ± 4.04	7.79 ± 5.15	<.001
Procedural characteristics				
Transfemoral approach	6738 (94.7)	6181 (95.3)	557 (88.6)	<.001
General anesthesia	2631 (37.0)	2303 (35.5)	328 (52.1)	<.001
Contrast volume, mL	106.4 ± 55.2	105.8 ± 54.3	112.7 ± 63.4	.005
Cardiopulmonary bypass used	21 (0.3)	8 (0.1)	13 (2.1)	<.001
In-hospital outcomes				
Cardiac arrest	177 (2.5)	110 (1.7)	67 (10.7)	<.001
New requirement for dialysis	46 (0.6)	4 (0.1)	42 (6.7)	<.001
Ischemic stroke	138 (1.9)	101 (1.6)	37 (5.9)	<.001
Bleeding at the access site	113 (1.6)	88 (1.4)	25 (4.0)	<.001
Major vascular complications	66 (0.9)	38 (0.6)	28 (4.5)	<.001
Myocardial infarction	14 (0.2)	9 (0.1)	5 (0.8)	.002
New pacemaker	679 (9.5)	566 (8.7)	113 (18.0)	<.001
New pathological Q wave or LBBB	1642 (23.1)	1472 (22.7)	170 (27.1)	.015
RBC/whole blood transfusion	587 (8.3)	393 (6.1)	194 (30.9)	<.001
Atrial fibrillation	170 (2.4)	127 (2.0)	43 (6.8)	<.001

Values are mean ± standard deviation or n (%).
AKI, acute kidney injury; GFR, glomerular filtration rate; LBBB, left bundle branch block; RBC, red blood cell; STS, Society of Thoracic Surgeons.

AKI. Analysis of the optimal cut point using ROC01 (cut point = 2.02, sensitivity = 0.543, specificity = 0.645) and the Youden index method (2.25, sensitivity = 0.473, specificity = 0.718) suggested an integer cut point of 2 (Supplemental Figure 1). As the ratio of 3 has been used as a cut point in prior PCI and PVI studies, both cut points of 2 and 3 were evaluated in this analysis.

Use of the CV/eGFR threshold of 3 as in prior PCI and PVI studies identified a smaller population of increased risk (<15%) compared to a ratio of 2, which included an additional 23% of patients. The risk of AKI was significantly increased in patients who had a CV/eGFR >3 (16.1% vs 7.2%, $P < .0001$) (Figure 1) as well as in patients who had a CV/eGFR >2 (12.2% vs 6.2%, $P < .0001$). After adjusting for patient factors, procedural characteristics that were associated with postprocedural AKI included CV/eGFR >3 (aOR = 1.38, $P = .009$, 95% CI = 1.09-1.77) as well as CV/eGFR >2 (aOR = 1.36, $P = .003$, 95% CI = 1.10-1.67), with the 2 cut points evaluated in separate models, and the use of general anesthesia (aOR = 1.67, $P < .0001$, 95% CI = 1.38-2.03) (Table 2).

Observed mortality

The Kaplan-Meier–estimated 1-year mortality was 11.1% (95% CI = 10.2%-12.0%) in the overall cohort. Unadjusted mortality was higher among patients who developed AKI (32.5% [28.2%-36.6%] vs 9.0% [8.1%-9.8%], $P < .0001$), and AKI remained significantly associated with the risk of mortality after multivariable adjustment (hazard ratio [HR] = 4.50, $P < .001$). There was a significant association between AKI and in-hospital mortality (11.6% vs 0.64%, $P < .0001$). Among patients who survived to discharge, the association between AKI and mortality was highest in the first 90 days after discharge (HR = 5.118, $P < .0001$) and remained significant in a landmark analysis of 90-day survivors to 1 year (HR = 1.59, $P = .007$) (Figure 2).

Table 2. Adjusted patient and procedural factors significantly associated with acute kidney injury

	Odds ratio	95% Confidence interval	*P*-value
Contrast/GFR >2	1.361	1.110-1.669	.0031
Contrast/GFR >3	1.384	1.086-1.765	.0087
General anesthesia	1.670	1.376-2.028	< .0001
Creatinine (mg/dL)	1.640	1.359-1.980	< .0001
Total albumin	0.623	0.505-0.768	< .0001
STS-PROM risk (%)	1.043	1.020-1.066	.0002
Coronary artery bypass graft	0.694	0.514-0.937	.0170
Aortic valve procedure	0.565	0.410-0.780	.0005
Peripheral arterial disease	1.297	1.065-1.581	.0099
Tobacco use	0.647	0.419-0.998	.0492
Diabetes	1.485	1.208-1.825	.0002
Home O_2	1.405	1.052-1.875	.0212
Hemoglobin	0.882	0.834-0.933	< .0001
Number of diseased major native coronary vessel systems (vs none)			
1	1.396	1.053-1.851	.0203
2	1.421	1.037-1.945	.0287
3 or more	1.498	1.067-2.104	.0196
Tricuspid valve regurgitation severity (vs none)			
Trace/trivial	1.293	0.896-1.866	.1695
Mild	1.168	0.824-1.654	.3833
Moderate	1.450	0.979-2.148	.0638
Severe	2.315	1.383-3.875	.0014

GFR, glomerular filtration rate; STS-PROM, Society of Thoracic Surgeons Predicted Risk of Mortality.

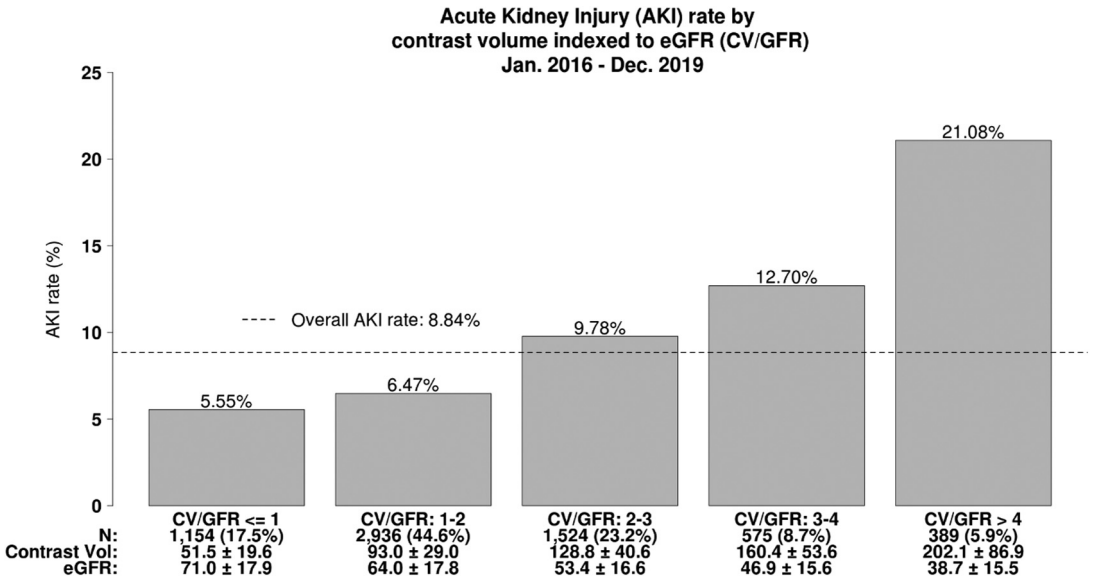


Figure 1. Acute kidney injury by contrast volume to estimated glomerular filtration rate ratio.

Discussion

CV administrated during a TAVR in the context of eGFR is a robust tool to predict AKI. Though prior studies have demonstrated the dose-dependent risk of AKI based on contrast, this analysis is the first to provide a practical threshold which may be used during TAVR procedures to avoid AKI (Central Illustration).

AKI remains common after TAVR in this large, real-world registry, and it is independently associated with a higher risk of 1-year mortality, particularly within the first 90 days after the procedure. The predictors of AKI included preprocedural creatinine, albumin, Society of Thoracic Surgeons Predictive Risk of Mortality, prior coronary artery bypass graft, prior aortic valve procedure, peripheral arterial disease, tobacco use, diabetes, home oxygen use, preprocedural hemoglobin, severe tricuspid valve regurgitation, and number of diseased coronary arteries.

This study complements and extends prior clinical trial and TVT registry data and represents a robust analysis of clinical risk in a contemporary, real-world population.[4,5,13–19] Though distinguishing between these causes of AKI in an individual case may be difficult, we attempted to estimate the risk of AKI attributable to procedural variables. Indeed, procedural factors such as contrast media volume or anesthetic choice were associated with increased AKI risk and can be targeted as a potential action item for quality improvement initiatives at TAVR sites.

The present study is the first to have demonstrated the clear risk of AKI based on procedural CV in the context of baseline renal function for patients undergoing TAVR. After adjusting for patient characteristics, procedural factors such as CV/eGFR >2 and >3 and use of general anesthesia were significantly associated with the development of AKI, with CV/eGFR >3 correlated with almost double the risk of AKI. TAVR operators must be aware of these patient-specific thresholds to minimize the risk of AKI and associated mortality.

Incorporation of both the 2× and 3× eGFR CV thresholds in the preprocedural timeout is a simple method of reminding TAVR operators to remain mindful of AKI risk. Members of the procedural team such as

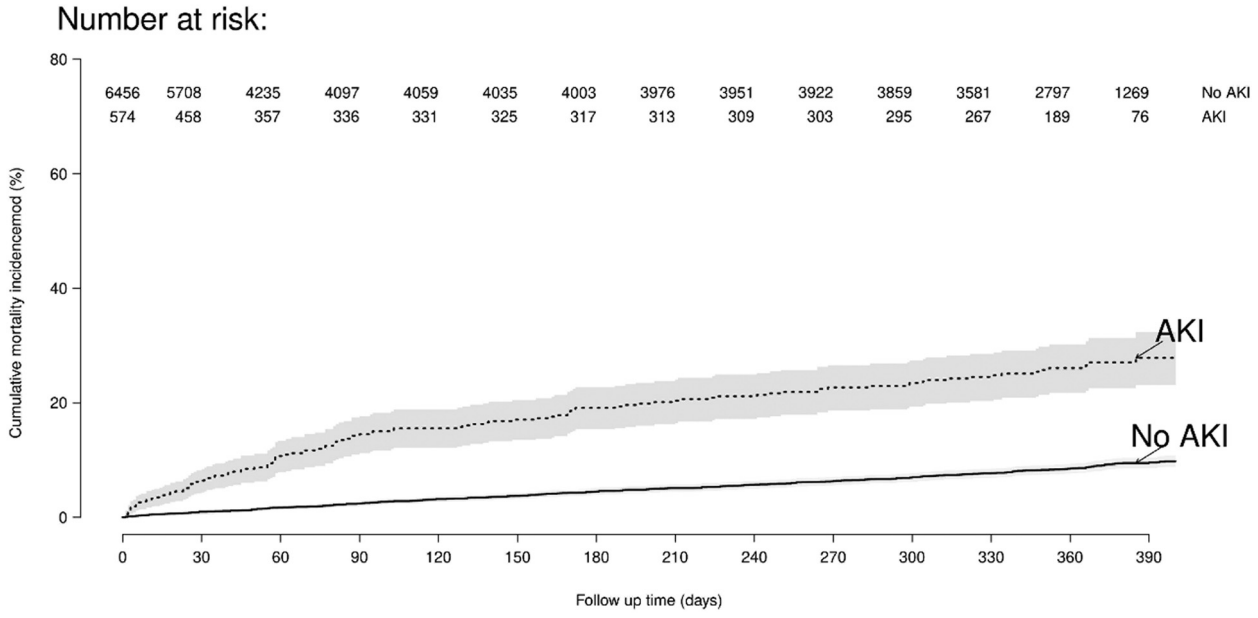
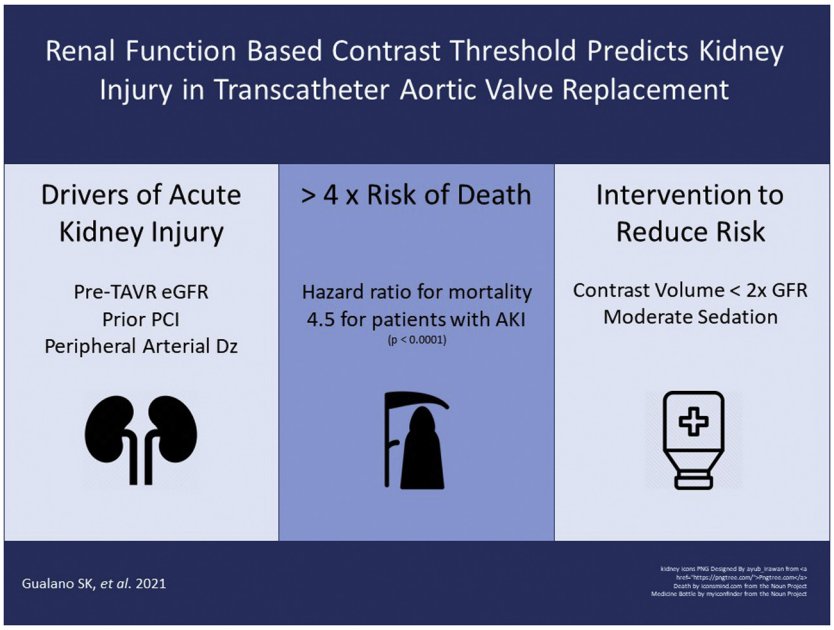


Figure 2. Kaplan-Meier survival curve for patients alive at discharge with and without postprocedural acute kidney injury (AKI).



Central Illustration. Renal function-based contrast thresholds predict acute kindey injury and provide a practical guide to reduce the likelihood of acute kidney injury.

nurses and technicians should be empowered to remind operators when contrast use is nearing these thresholds during a TAVR. Operators should consider a CV/eGFR ratio of <2 ideal but should make every effort possible to remain under the threshold of 3. Reduction of contrast dosing could be achieved through a variety of means, for example, elimination of a pelvic angiogram using 30-40 cc of contrast and instead using selective iliac angiography with 5 cc of contrast for assessment of the access sites after implantation. Alternatively, Doppler assessments of access sites may be performed. The optimal implant view may be determined using only 10 cc of contrast or less. Implantation views can also be obtained from pre-TAVR computed tomography angiography and during the procedure with the use of 2 pigtail catheters in different sinuses.

Study limitations

Our study has several limitations. Although we adjusted for patient risk by incorporating multiple variables associated with patient risk, as an observational study, there may be residual confounding. Analysis is also limited to variables as part of the registry without ability to modify. Variability in practice patterns between TAVR sites was not characterized. Specific type of contrast used at each site was unknown. Preprocedural intravenous volume expansion and renal function–based contrast mitigation strategies which have been shown to be effective in reducing AKI during PCI procedures are unknown in this population.[1,20] The rationale for selection of general anesthesia was also not known. Use of general anesthesia because of operator comfort vs requirement for alternative access or procedure complexity was not assessed. Further details about clinical decision-making regarding initiation of dialysis or predialysis supportive care were unavailable.

Conclusions

CV administrated during a TAVR in the context of eGFR is a practical metric which predicts AKI, and awareness of this threshold should be incorporated into modern practice. AKI remains common after TAVR and is associated with an increased risk of 90-day and 1-year mortality and increased hospital length of stay. Modifiable risk factors associated with AKI after TAVR included CV used in the context of renal function and the use of general anesthesia. A CV greater than 2 times eGFR was strongly associated with AKI. Procedure techniques including contrast-sparing strategies and the use of moderate sedation could reduce the incidence of AKI and should be included in quality improvement initiatives.

Declaration of competing interest

S. Gualano, W. Merhi, C. Schwartz, and F. Shannon have no disclosures. S. Chetcuti has been a consultant and proctor for Medtronic, with research support from Medtronic, Gore, Boston Scientific, Edwards Lifesciences, and JenaValve. D. Sukul receives research funding from Blue Cross Blue Shield of Michigan. H. Patel is a consultant for Medtronic and WL Gore. H. Gurm receives research funding from Blue Cross Blue Shield of Michigan and the National Institutes of Health Center for Accelerated Innovation and is a consultant for Osprey Medical. W. O'Neill is a consultant for Abbott, Edwards Lifesciences, Boston Scientific, and Abiomed, with research grant support from Abiomed, Abbott, Edwards Lifesciences, and Medtronic. P. Grossman is a consultant for Medtronic Cardiovascular, has research support from Medtronic Cardiovascular, Edwards Life Sciences, and Cardiovascular Systems Incorporated, has registry support from Blue Cross Blue Shield of Michigan, and has research support from National Institutes of Health. None of the authors have any conflicts directly relevant to this study.

Acknowledgments

The authors are indebted to all the study coordinators, investigators, and patients who participated in the BMC2 registry.

Funding sources

Support for the BMC2 is provided by Blue Cross Blue Shield of Michigan as part of the Blue Cross Blue Shield of Michigan Value Partnerships program; the opinions, beliefs, and viewpoints expressed by the authors do not necessarily reflect those of Blue Cross Blue Shield of Michigan. Dr Patel is supported in part by the Joe D. Morris Collegiate Professorship, Phil Jenkins Breakthrough Fund, and David Hamilton Fund.

Supplementary material

To access the supplementary material accompanying this article, visit the online version of the *Journal of the Society for Cardiovascular Angiography & Interventions* at https://doi.org/10.1016/j.jscai.2022.100038.

References

1. Gurm HS, Dixon SR, Smith DE, et al. Renal function-based contrast dosing to define safe limits of radiographic contrast media in patients undergoing percutaneous coronary interventions. *J Am Coll Cardiol*. 2011;58(9):907–914. https://doi.org/10.1016/j.jacc.2011.05.023.
2. Kooiman J, Seth M, Nallamothu BK, Heung M, Humes D, Gurm HS. Association between acute kidney injury and in-hospital mortality in patients undergoing percutaneous coronary interventions. *Circ Cardiovasc Interv*. 2015;8(6):e002212. https://doi.org/10.1161/CIRCINTERVENTIONS.114.002212.
3. Arnold SV, Zhang Y, Baron SJ, et al. Impact of short-term complications on mortality and quality of life after transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2019;12(4):362–369. https://doi.org/10.1016/j.jcin.2018.11.008.
4. Bagur R, Webb JG, Nietlispach F, et al. Acute kidney injury following transcatheter aortic valve implantation: predictive factors, prognostic value, and comparison with surgical aortic valve replacement. *Eur Heart J*. 2010;31(7):865–874. https://doi.org/10.1093/eurheartj/ehp552.
5. Codner P, Levi A, Gargiulo G, et al. Impact of renal dysfunction on results of transcatheter aortic valve replacement outcomes in a large multicenter cohort. *Am J Cardiol*. 2016;118(12):1888–1896. https://doi.org/10.1016/j.amjcard.2016.08.082.
6. Aregger F, Wenaweser P, Hellige GJ, et al. Risk of acute kidney injury in patients with severe aortic valve stenosis undergoing transcatheter valve replacement. *Nephrol Dial Transplant*. 2009;24(7):2175–2179. https://doi.org/10.1093/ndt/gfp036.
7. Cubeddu RJ, Asher CR, Lowry AM, et al. Impact of transcatheter aortic valve replacement on severity of chronic kidney disease. *J Am Coll Cardiol*. 2020;76(12):1410–1421. https://doi.org/10.1016/j.jacc.2020.07.048.
8. Julien HM, Stebbins A, Vemulapalli S, et al. Incidence, predictors, and outcomes of acute kidney injury in patients undergoing transcatheter aortic valve replacement: insights from the Society of Thoracic Surgeons/American College of Cardiology National Cardiovascular Data Registry-Transcatheter Valve Therapy Registry. *Circ Cardiovasc Interv*. 2021;14(4):e010032. https://doi.org/10.1161/CIRCINTERVENTIONS.120.010032.
9. Grossman PM, Ali SS, Aronow HD, et al. Contrast-induced nephropathy in patients undergoing endovascular peripheral vascular intervention: incidence, risk factors, and outcomes as observed in the Blue Cross Blue Shield of Michigan Cardiovascular Consortium. *J Interv Cardiol*. 2017;30(3):274–280. https://doi.org/10.1111/joic.12379.
10. Registry SAT. TAVR DCF for TVT Registry. Accessed October 21, 2021, 2021. https://www.ncdr.com/WebNCDR/docs/default-source/tvt-public-page-documents/tvt-registry-2_0_coderdatadictionary.pdf?sfvrsn=2
11. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med*. 2009;150(9):604–612. https://doi.org/10.7326/0003-4819-150-9-200905050-00006.
12. Kappetein AP, Head SJ, Genereux P, et al. Updated standardized endpoint definitions for transcatheter aortic valve implantation: the Valve Academic Research Consortium-2 consensus document. *Eur Heart J*. 2012;33(19):2403–2418. https://doi.org/10.1093/eurheartj/ehs255.
13. Sinning JM, Ghanem A, Steinhauser H, et al. Renal function as predictor of mortality in patients after percutaneous transcatheter aortic valve implantation. *JACC Cardiovasc Interv*. 2010;3(11):1141–1149. https://doi.org/10.1016/j.jcin.2010.09.009.
14. Oguri A, Yamamoto M, Mouillet G, et al. Impact of chronic kidney disease on the outcomes of transcatheter aortic valve implantation: results from the FRANCE 2 Registry. *EuroIntervention*. 2015;10(9):e1–e9. https://doi.org/10.4244/EIJV10I9A183.
15. Dumonteil N, van der Boon RM, Tchetche D, et al. Impact of preoperative chronic kidney disease on short- and long-term outcomes after transcatheter aortic valve implantation: a Pooled-RotterdAm-Milano-Toulouse in Collaboration Plus (PRAGMATIC-Plus) initiative substudy. *Am Heart J*. 2013;165(5):752–760. https://doi.org/10.1016/j.ahj.2012.12.013.
16. Munoz-Garcia AJ, Munoz-Garcia E, Jimenez-Navarro MF, et al. Clinical impact of acute kidney injury on short- and long-term outcomes after transcatheter aortic valve implantation with the CoreValve prosthesis. *J Cardiol*. 2015;66(1):46–49. https://doi.org/10.1016/j.jjcc.2014.09.009.
17. Thourani VH, Forcillo J, Beohar N, et al. Impact of preoperative chronic kidney disease in 2,531 high-risk and inoperable patients undergoing transcatheter aortic valve replacement in the PARTNER trial. *Ann Thorac Surg*. 2016;102(4):1172–1180. https://doi.org/10.1016/j.athoracsur.2016.07.001.
18. Ifedili IA, Bolorunduro O, Bob-Manuel T, et al. Impact of pre-existing kidney dysfunction on outcomes following transcatheter aortic valve replacement. *Curr Cardiol Rev*. 2017;13(4):283–292. https://doi.org/10.2174/1573403X13666170804151608.
19. Beohar N, Doshi D, Thourani V, et al. Association of transcatheter aortic valve replacement with 30-day renal function and 1-year outcomes among patients presenting with compromised baseline renal function: experience from the PARTNER 1 trial and registry. *JAMA Cardiol*. 2017;2(7):742–749. https://doi.org/10.1001/jamacardio.2017.1220.
20. Ad-hoc Working Group of ERBP, Fliser D, Laville M, et al. A European Renal Best Practice (ERBP) position statement on the Kidney Disease Improving Global Outcomes (KDIGO) clinical practice guidelines on acute kidney injury: part 1: definitions, conservative management and contrast-induced nephropathy. *Nephrol Dial Transplant*. 2012;27(12):4263–4272. https://doi.org/10.1093/ndt/gfs375.