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Predicting Acute Kidney Injury following Transcatheter Aortic Valve Replacement

Abstract

Purpose: Acute kidney injury occurs in up to a quarter of patients following transcatheter aortic valve replacement (TAVR) and has been associated with increased short and long-term mortality rates. A variety of patient characteristics predictive of post-TAVR acute kidney injury (AKI) have been identified, however discrepancies among studies exist almost uniformly. We investigated the hypothesis that the change in glomerular filtration rate (Δ GFR) in response to contrast administered during pre-TAVR coronary angiography is predictive of Δ GFR post-TAVR.

Methods: The study comprised 195 patients who underwent TAVR at a single center between August 2008 and June 2015 and were prospectively included in the CAPITAL TAVR registry. Multiple linear regression analysis was conducted to estimate the effect of independent variables on the change in renal function post-TAVR.

Results: There was no relationship identified between the Δ GFR post-angiogram and the Δ GFR post-TAVR ($r=0.043$, $P=0.582$). Multiple linear regression analysis revealed that a significant amount of the change in renal function post-TAVR can be explained by the patient's baseline creatinine (beta coefficient, -0.310 , $P<0.001$) and the volume of contrast administered during TAVR (beta coefficient, -0.225 , $P=0.002$). The presence of an AKI following diagnostic coronary angiogram was not predictive of the change in renal function post-TAVR using the Valve Academic Research Consortium (VARC) definitions: VARC1 (beta coefficient, 0.102 , $P=0.170$) or VARC2 (beta coefficient, 0.124 , $P=0.099$).

Conclusions: A patient's previous renal response to contrast administered during coronary angiogram is not predictive of their response post-TAVR; instead, as demonstrated previously, baseline renal function and contrast volume administered are two of the most important predictors of post-TAVR AKI.

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Transcatheter aortic valve replacement (TAVR) is the standard of care for managing patients with symptomatic, severe aortic valve stenosis of prohibitive and high operative risk [1,2]. Recent improvements in transcatheter valves, increased clinician experience and studies demonstrating the safety and efficacy in various patient populations and levels of risk continue to expand the role of TAVR [3,4]. Consequently, TAVR is now widely used for the treatment of native aortic valve disease.

Although physician experience and technology are improving, procedural complications remain a common issue affecting patient outcomes. Specifically, acute kidney injury (AKI) occurs in as many as 8.3–58% of patients, and has been shown to be associated with increased 30-day and three-year post-TAVR mortality rates [5,6]. A recent systematic review confirmed this relationship by demonstrating that developing an AKI, results in a 2–6 fold increase in post-TAVR mortality [5]. Consequently, identifying patients at increased risk for developing AKI following TAVR is critical, as it has substantial implications for the management of these patients in the perioperative period. Once identified, the risk of post-TAVR AKI can potentially be mitigated through medical optimization in the preoperative, intraoperative and postoperative periods.

Existing literature has documented a variety of patient characteristics that place individuals at increased risk for post-TAVR AKI. Diabetes mellitus, hypertension, chronic kidney disease and both pre- and post-procedural anemia have all been identified as independent predictors for the development of post-procedure AKI [6–9]. Beyond individual patient factors, several intraoperative (general anesthesia, trans-apical approach), and postoperative (transfused >3 units of packed red blood cells, hemoglobin drop, contrast media volume, major bleeding) factors have been shown to increase the risk of AKI post-TAVR [7,10–12]; however, these results have not been consistently replicated. In fact, among studies that have evaluated the predictors of post-TAVR AKI discrepancies exist almost uniformly. Chatani *et al.* reported that baseline creatinine is the only independent predictor of AKI, while Elhmidi *et al.* stated that, in addition to renal insufficiency, peripheral vascular disease and blood transfusion are independent predictors [5,13].

As a result of these discrepancies it remains unknown if, beyond baseline renal insufficiency, an accurate predictor of post-TAVR AKI exists. Since patients undergo diagnostic angiography prior to TAVR for the assessment of procedural candidacy, we investigated the hypothesis that the occurrence of AKI in response to contrast during angiography would be

predictive of the change in a patient's glomerular filtration rate (Δ GFR) post-TAVR. In other words, the diagnostic angiogram would be akin to a contrast stress test. If true, this marker could predict risk and potentially alter pre-procedural monitoring to include surveillance of AKI post-angiogram, with the goal of more accurate risk stratification and prognostication of renal outcomes in patients undergoing TAVR.

Methods

Study cohort and data source

The study population was comprised of all patients ($n=195$) who underwent TAVR at the University of Ottawa Heart Institute between August 18, 2008 and June 15, 2015 (Figure 1). Patients are prospectively included in the CAPITAL TAVR registry. Clinical data obtained from the registry included demographic characteristics (e.g., age, gender) and comorbidities (Table 1). We retrospectively collected contrast volume administered during angiogram and during TAVR, as well as baseline creatinine levels and daily creatinine levels post-procedure until discharge. The estimated glomerular filtration rate (eGFR) was determined by the Modification of Diet in Renal Disease (MDRD) Study equation. The Δ GFR was defined as the difference between the patient's baseline GFR and their minimum GFR post-procedure.

Study outcomes

The primary outcomes of interest were the relationship between the Δ GFR following coronary angiography, and the Δ GFR following TAVR, and if the occurrence of an AKI following coronary angiography was predictive of a Δ GFR and AKI following TAVR. The secondary outcomes measured were the frequency of AKI following angiography and TAVR, and the stages of AKI observed.

AKI definitions

AKI was defined for all patients according to the Acute Kidney Injury Network (AKIN), the Valve Academic Research Consortium (VARC) 1 / RIFLE, and the VARC2 / KDIGO classifications for diagnostic angiogram and TAVR. (Table 2)

Statistical analysis

All analyses were completed using SPSS software (V 24.0). Tests to see if the data met the assumption of collinearity indicated that multicollinearity was not a concern (TAVR baseline creatinine, tolerance = .918, VIF = 1.090; TAVR

TABLE 1. Baseline characteristics of total cohort and according to occurrence of VARC2 acute kidney injury post-angiogram and post-TAVR

		Total Study Cohort (N = 195)	VARC 2 AKI Post-Angiogram (N=31)	VARC 2 AKI Post-TAVR (N=60)
Age (years)	Mean	84.47	83.66	84.00
	<75	18 (9.2%)	3 (9.7%)	9 (15%)
	75 - 84	68 (34.9%)	16 (51.6%)	20 (33.3%)
	85 - 94	102 (52.3%)	12 (38.7%)	30 (50.0%)
	>95	7 (3.6%)	0 (0%)	1 (1.7%)
Women		101 (51.8%)	18 (58.1%)	33 (55.0%)
BMI	<18.5	9 (4.6%)	1 (3.2%)	3 (5.0%)
	18.5 - 25	84 (43.1%)	13 (41.9%)	26 (43.3%)
	25.1 - 30	75 (38.5%)	11 (35.5%)	23 (38.3%)
	>30.1	27 (13.8%)	6 (19.4%)	8 (13.3%)
Smoking history	Never	140 (71.8%)	21 (67.7%)	46 (76.7%)
	Current	5 (2.6%)	0 (0%)	1 (1.7%)
	Former	50 (25.6%)	10 (32.3%)	12 (20.0%)
Dyslipidemia		122 (62.6%)	19 (61.3%)	41 (68.3%)
Diabetes		78 (40%)	18 (58.1%)	29 (48.3%)
Hypertension		138 (70.8%)	20 (64.5%)	41 (68.3%)
CKD		191 (97.8%)	31 (100%)	60 (100%)
CAD		109 (55.9%)	16 (51.6%)	34 (56.7%)
NYHA III/IV		127 (65.1%)	19 (61.3%)	39 (65.0%)
Prior CABG		39 (20%)	4 (12.9%)	11 (18.3%)
PAD		27 (13.8%)	3 (9.7%)	12 (20.0%)
Prior TIA or stroke		32 (16.4%)	9 (29.0%)	10 (16.7%)
Atrial fibrillation/flutter		85 (43.6%)	14 (45.2%)	28 (46.7%)
Ejection fraction <40%		79 (40.5%)	12 (38.7%)	23 (38.3%)

contrast volume, tolerance = .970, VIF = 1.031). The data met the assumption of independent errors (Durbin-Watson value = 1.47). A multiple linear regression analysis was conducted to estimate the effect of several independent variables on the change in renal function post-TAVR. The regression model used contained the following five independent variables: age; diabetes mellitus; AKI following diagnostic angiogram; TAVR baseline creatinine; and the contrast volume administered during the TAVR. The model was evaluated using all three

AKI definitions. A $P < 0.05$ was considered to be statistically significant. Additionally, the frequency and stages of AKI were calculated following both the coronary angiogram, and TAVR according to the AKIN, VARC1 and VARC2 classifications.

TABLE 2. Acute kidney injury according to the RIFLE, VARC1 and VARC2 Classifications

	AKIN		VARC1/RIFLE		VARC2/KDIGO	
Stage	Serum creatinine	Urine output	Serum creatinine	Urine output	Serum creatinine	Urine output
1	1.5-2.0 fold increase OR ≥26.5 µmol/L (≥0.3 mg/dL) within 48 hours	<0.5 mL/kg/hr for >6 hours	1.5 fold increase OR GFR decrease >25%	<0.5 mL/kg/hr for 6 hours	1.5-1.9 fold increase within the prior 7 days OR Absolute creatinine increase of ≥26.5 µmol/L (≥0.3 mg/dL) within 48 hours	<0.5 mL/kg/hr for 6-12 hours
2	2.0-3.0 fold increase	<0.5 mL/kg/hr for >12 hours	2 fold increase OR GFR decrease >50%	<0.5 mL/kg/hr for >12 hrs	2.0-2.9 fold increase	<0.5 mL/kg/hr for >12 hours
3	>3 fold increase to ≥4.0 mg/dL OR Increase in creatinine to ≥353.6 µmol/L (≥4 mg/dL) with acute increase of at least 44 µmol/L (0.5 mg/dL)	<0.3 mL/kg/hr for ≥24 hrs OR Anuria for 12 hours	≥3 fold increase OR GFR decrease 75% OR Increase in creatinine to ≥353.6 µmol/L (≥4 mg/dL)	<0.3 mL/kg/hr for ≥24 hrs OR Anuria for ≥12 hours	≥3 fold increase OR Increase in creatinine to ≥353.6 µmol/L (≥4 mg/dL) OR Initiation of renal replacement therapy	<0.3 mL/kg/hr for ≥24 hrs OR Anuria for ≥12 hours

TABLE 3. TAVR procedural characteristics of total cohort and according to occurrence of VARC2 acute kidney injury

	Total Study Cohort (n = 195)	VARC2 AKI – (n=135)	VARC2 AKI + (n=60)	P-value
Contrast volume (mL) (mean)	250.35 (82.2)	249.05	261.66	0.21
Conscious sedation	10 (5.1%)	7 (5.2%)	3 (5.0%)	0.798
Approach (femoral)	109 (55.9%)	82 (60.7%)	27 (45.0%)	0.022
Self-expanding TAVR valve	116 (59.5%)	80 (59.3%)	36 (60.0%)	0.306
Duration (mean)	120	122	121	0.533
Mortality				
30-Day	10 (5.1%)	2 (1.5%)	8 (13.3%)	0.002
6-Month	32 (16.4%)	11 (8.1%)	21 (35.0%)	<0.001
1-Year	42 (21.5%)	20 (14.8%)	22 (36.7%)	0.002

Abbreviation: TAVR, transcatheter aortic valve replacement

TABLE 4. Predictors of change in GFR post-TAVR on multiple linear regression analysis

	Variables	Standardized coefficients beta	t-test statistic	P-value
AKIN AKI*	(Constant)		1.339	0.183
	Age	0.029	0.392	0.696
	Diabetes	0.013	0.165	0.869
	Angiogram AKI	0.169	2.311	0.022
	TAVR baseline creatinine	-0.307	-4.184	<0.001
	TAVR contrast volume	-0.216	-2.985	0.003
VARC1/RIFLE AKI†	(Constant)		1.750	0.082
	Age	5.73x10 ⁻⁵	0.055	0.956
	Diabetes	0.014	0.177	0.860
	Angiogram AKI	0.102	1.379	0.170
	TAVR baseline creatinine	-0.314	-4.274	<0.001
	TAVR contrast volume	-0.227	-3.13	0.002
VARC2/KDIGO AKI‡	(Constant)		1.45	0.149
	Age	0.023	0.304	0.761
	Diabetes	0.011	0.141	0.888
	Angiogram AKI	0.124	1.658	0.099
	TAVR baseline creatinine	-0.310	-4.165	<0.001
	TAVR contrast volume	-0.225	-3.081	0.002

* AKI post-diagnostic angiogram using the AKIN definition

† AKI post-diagnostic angiogram using the VARC1/RIFLE definition

‡ AKI post-diagnostic angiogram using the VARC2/KDIGO definition

Abbreviation: AKI, acute kidney injury; TAVR, transcatheter aortic valve replacement

Results

A cohort of 195 patients underwent TAVR during the study period and was included in the final analysis. The patient population was comprised of 101 women (51.8%), and the mean age and BMI were 84.47 years and 25.34, respectively. The patients were predominantly non-smokers (71.8%) and the majority of them had a history of dyslipidemia (62.6%), hypertension (70.8%), coronary artery disease (55.9%) and New York Heart Association (NYHA) class III or IV symptoms (65.1%). Chronic kidney disease was quite common with almost 90% of patients having baseline CKD Stage 2 or higher (Table 1).

Overall the TAVR procedure details were similar between patients who had AKI post-TAVR, and those who did not. VARC2 AKI was observed less frequently among patients who had a femoral approach (60.7% vs. 45.0%, $P=0.022$). There was also a significant increase in 30-day, 6-month and 1-year mortality among patients who had VARC2 AKI (Table 3).

The primary outcome failed to identify a relationship between each patient's Δ GFR post-angiogram and their Δ GFR post-TAVR ($r=0.043$, $P=0.582$) (Figure 2A). There was also no difference in the mean Δ GFR when adjusted for the volume of contrast administered (Figure 2B). Multiple linear regression analysis revealed that a significant amount of the change in renal function post-TAVR can be explained by the patient's baseline creatinine (beta coefficient, -0.310, $P<0.001$)

FIGURE 1. Patient flow.

FIGURE 2. A) Scatterplot demonstrating each patient's Δ GFR post-diagnostic angiogram versus their Δ GFR post-TAVR. B) Mean Δ GFR per milliliter contrast post-diagnostic angiogram and post-TAVR

and the volume of contrast administered during TAVR (beta coefficient, -0.225 , $P=0.002$) (Table 4). Both of these variables remained predictive across all three AKI definitions. Notably, the presence of an AKI following diagnostic coronary angiogram was not predictive of the change in renal function post-TAVR using either the VARC1 (beta coefficient, 0.102 , $P=0.170$) or VARC2 (beta coefficient, 0.124 , $P=0.099$) definitions. The AKIN definition did demonstrate a weak

correlation between angiogram AKI and the Δ GFR TAVR (beta coefficient, 0.169 , $P=0.022$).

Incidence of AKI post-angiogram and post-TAVR was calculated based on the AKIN, VARC1 and VARC2 definitions. AKIN, VARC1, and VARC2 AKI occurred in 24%, 38% and 31% of patients post-TAVR, respectively. These values are comparable to 14%, 17% and 16% of patients post-angiogram (Figure 3). Fewer than 10% of patients

FIGURE 3. Rate of Acute Kidney Injury (AKI) according to the Acute Kidney Injury Network (AKIN), Valve Academic Research Consortium (VARC) VARC1/RIFLE and VARC2/Kidney Disease Improving Global Outcomes (KDIGO) definitions. Patients were identified as having no acute kidney injury (AKI), AKI following angiogram, AKI following transcatheter aorta valve replacement (TAVR) or AKI following both angiogram and TAVR.

FIGURE 4. Frequency of acute kidney injury (AKI) according to stage. A) AKIN definition B) VARC1/RIFLE definition C) VARC2/KDIGO definition.

experienced AKI both post-angiogram and post-TAVR regardless of definition.

The majority of AKIs observed post-angiogram and post-TAVR were classified as Stage 1, with only a very small number classified as Stage 3. Furthermore, the frequency of AKI post-TAVR exceeded post-angiogram across all stages regardless of AKI definition (Figure 4). The VARC1 classification was the most sensitive for identifying AKI across all stages following angiogram and TAVR, identifying 73 AKIs post-TAVR and 33 post-angiogram, as compared with 46 and

27 with the AKIN definition, and 60 and 31 with the VARC2 definition ($P < 0.001$).

Discussion

Previous studies have documented the incidence of AKI post-TAVR, as well as its associated short- and long-term mortality [14,15]. Our study sought to answer the question of whether a patient's previous response to a "renal stress test" in the form of a diagnostic angiogram would enable us to predict

their future response following TAVR. Based on our analysis of renal injury, it is clear that a patient's risk of having AKI post-TAVR is independent of previous responses to intravenous contrast.

These results are unlikely to be missing an important relationship between diagnostic angiogram and TAVR AKI for several reasons. First, our sample size of 195 patients is similar to prior studies that have identified important predictors of post-TAVR AKI [11,16,17]. Second, the incidence of AKI post-TAVR (24-38%) in our study is in line with previously reported frequencies [5,6]. Third, although our results did not demonstrate a correlation between angiogram AKI and TAVR AKI, they did identify baseline renal function and contrast volume as independent predictors of post-TAVR AKI. Again, these results are consistent with previously identified predictors of post-TAVR AKI [5,14]. A weak yet statistically significant relationship was identified using the AKIN definition, although the magnitude of the relationship was not clinically meaningful.

As previously discussed, our study confirms the results of prior studies demonstrating that the volume of contrast administered during TAVR is predictive post-TAVR AKI. The inability to demonstrate a similar correlation following contrast administration during coronary angiogram is likely multifaceted. TAVR requires large artery access (femoral, aortic, subclavian) and utilizes larger sheaths and catheters than those used during an angiogram. With the larger equipment required for TAVR, there is an increased risk of distal embolization and increased frequency of hemodynamic instability during valve deployment; thus, contrast administration is not the only renal insult during TAVR and likely partly explains why the renal response to contrast administration during angiogram is not directly correlated to the response observed post-TAVR.

Strengths of our study design include the relatively large cohort size and the use of numerous assessments of renal function. First, we utilized Δ GFR as a continuous variable to enable us the greatest sensitivity to detect a relationship between changes in renal function. Second, we utilized all three accepted clinical definitions for the identification of AKI. Koifman et al. demonstrated that the VARC1 and VARC2 classification systems were associated with distinct predictor variables, and that reclassification of AKI grade occurred in approximately 10% of patients when different definitions were applied [18]. Through classification of each patient by the AKIN, VARC1 and VARC2 definitions, we have increased our sensitivity for identifying AKI and ensured that all patients are included in our analysis. Additionally, we

completed multiple linear regression modelling separately for each definition of AKI to determine if AKI post-angiogram was predictive in any of the definitions.

There were several limitations to our study that must be noted. It was a single center study with a long enrolment period that was unable to account for the potential changes in procedural practice during the study period. Secondly, there was no standard pre-hydration regimen that all patients received prior to their angiogram, or their TAVR. Instead the pre- and post-procedure fluid orders were at the discretion of the individual practitioner, which has the potential to contribute to the frequency of observed AKI. Moreover, there is the potential for practitioners to alter application of renal protective strategies in patients undergoing TAVR who experienced AKI during coronary angiography—which could confound our findings. Finally, through our data collection, we were unable to identify accurately episodes of hemodynamic instability and, therefore, episodes of hypotension, which may have contributed to AKI, were not included in our analysis.

In conclusion, our study is the first to address the question of whether a patient's previous renal response to contrast is predictive of a future response during TAVR. Our results failed to identify this as a predictive relationship; however, they support the results of previous studies that have shown baseline renal function and contrast volume as two of the most important predictors of post-TAVR AKI. AKI continues to occur in a significant number of patients post-TAVR and is associated with substantially increased mortality; therefore, it is imperative that we continue to improve our ability to identify patients at increased risk for post-TAVR AKI, and perhaps more importantly, develop perioperative treatment strategies to prevent it.

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