Original Article on Novel Concepts in Cardiopulmonary and Structural Heart Disease

Tricuspid regurgitation and in-hospital outcomes after transcatheter aortic valve replacement in high-risk patients

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Background: The prognostic impact of tricuspid regurgitation (TR) following transcatheter aortic valve replacement (TAVR) is uncertain, and the management of patients with severe aortic stenosis and significant TR undergoing TAVR is unclear.

Methods: Retrospective study investigating the role of TR severity on hospital outcomes in high risk patients with severe aortic stenosis undergoing TAVR.

Results: A total of 174 participants were included in the present study. The median age was 84 years and 48% were women. The median (IR) STS score was 7.3 (4.7–13.6). The pre-procedural mean (SD) aortic valve area (AVA) was 0.69 (0.2) cm^2 and the average (SD) peak and mean gradients were 71 [23]/42 [15] mmHg. Pre TAVR, 28.7% of patients had significant (moderate or severe) TR. Significant TR pre-TAVR increased the risk of in-hospital cardiovascular (CV) and all-cause and mortality [adjusted relative risk (RR) (95% CI): 14.67 (1.35–159.51) and 5.09 (1.14–22.72), respectively], and those with severe TR post-TAVR had longer hospital stay [median (IR): 9.9 (2.9–17.0) days]. No improvement or worsened TR (greater than mild) post-TAVR was associated with higher CV and all-cause mortality [adjusted RR (95% CI): 21.5 (1.81–255.96) and 8.19 (1.67–40.29), respectively]. Right ventricular systolic pressure (RVSP) was independently associated with TR severity pre and post TAVR.

Conclusions: Significant TR was common among patients undergoing high risk TAVR, and is associated with increased in hospital mortality and longer hospital stay. Patients with elevated RVSP and persistent moderate or severe TR after TAVR are at higher risk of in hospital death.

Keywords: Tricuspid regurgitation (TR); aortic stenosis; transcatheter aortic valve replacement (TAVR); hospital outcomes

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Introduction

Tricuspid regurgitation (TR) is present in 15% to 20% of patients referred for echocardiography and increases mortality commensurate to the degree of valve insufficiency (1). Moreover, non-treated significant TR at the time of left-sided valve surgery is associated with post-operative morbidity and current guidelines recommend concomitant tricuspid valve intervention for patients with severe TR

and/or annular dilatation (2-6).

Transcatheter aortic valve replacement (TAVR) is approved for treatment of symptomatic severe aortic stenosis (AS) in patients at low to high surgical risk. In this population the reported prevalence of moderate to severe TR ranges between 10% and 31% (7-14) and may lessen the clinical benefits of TAVR. In the PARTNER II trial (inoperable cohort), moderate or severe TR were associated with a 1.6- and 3.2-fold increased risk of post-TAVR mortality, as was right atrial or ventricular enlargement (8). Yet, the prognostic impact of TR following TAVR is uncertain and there are no clear guideline recommendations for the management of patients with severe AS and significant TR in the TAVR era.

In this study, we aimed to investigate the impact of TR on short-term TAVR outcomes and ascertain the role of clinical factors associated with TR severity after TAVR.

Methods

Data collection

This study was approved by the Institutional Review Board of Mount Sinai Medical Center. From August 2014 to January 2017, data was collected on all patients with severe AS that underwent TAVR at Mount Sinai Medical Center, Miami Beach, Florida. All patients underwent evaluation by a multidisciplinary Structural Heart Team including interventional and noninvasive cardiologists and cardiothoracic surgeons. TAVR was performed using the self-expandable Medtronic CoreValve System (Medtronic Inc., Minneapolis, MN, USA) or the balloon expandable Edwards-SAPIEN System (Edwards Lifesciences Inc., Irvine, CA, USA) at the discretion of the implanting team. Valve sizing was determined using preoperative three-dimensional transesophageal echocardiography or computed tomography. All procedures were performed in a hybrid cardiac catheterization suite under general anesthesia, under fluoroscopy and transesophageal or transthoracic echocardiographic guidance.

Prior (within 45 days) to the TAVR procedure patients had a complete transthoracic echocardiogram which was again repeated after the procedure. Measurements of left ventricular (LV) chamber dimensions, ejection fraction, LV mass, and left atrial volume were made as recommended by the American Society of Echocardiography (ASE) (15). TR severity was categorized as trace/mild (0–1+), moderate (2+), or severe (3–4+) according to ASE guidelines (16,17). Change in the severity of TR was also analyzed in three groups as follows: (I) patients who *remained mild* (≤1+) had pre and post TAVR TR assessed as mild; (II) patients who *improved* had moderate or severe TR that improved by 1 or more grades; and, (III) patients who had *no change/worsened* had baseline and post-TAVR persistent moderate or severe TR. TR was divided into functional (annular dilation, leaflet tethering), pacemaker mediated, or primary TR. Continuous Doppler was used to calculate the peak and mean aortic valve gradients using the modified Bernoulli equation. The aortic valve area (AVA) was calculated by the continuity equation. Stroke volume was calculated using the left ventricular outflow tract velocity integral and the left ventricular outflow tract diameter. Right ventricular systolic pressure (RVSP) was evaluated using the TR jet peak velocity and the inferior vena cava collapsibility index.

Outcomes

The main outcomes were all-cause and cardiovascular mortality as well as hospital length of stay (LOS). All-cause mortality was defined as death from any cause during hospitalization. Cardiovascular (CV) mortality was defined as mortality as a result of cardiac related causes. Hospital LOS was calculated as the difference (in days) between the date of procedure to the date of discharge or death.

Statistical methods

All continuous variables were graphed and visually assessed for normality. Normally distributed variables were expressed as mean and standard deviations of the mean (SD) while the non-normally distributed variables were expressed as median and interquartile range (IQR). Categorical variables were expressed in percentages. Median values across ordinal categories were compared with trend analysis (unless otherwise stated) while the chi-square was used to compare proportions or percentages. The student t-test was used to assess for differences in mean of normally distributed continuous variables between two categories or before and after TAVR. To compare all-cause and cardiovascular disease related mortality among TR groups, Poisson regression coefficients were computed and were transformed into risk ratios and 95% confidence interval (CI). For LOS analysis, median regression coefficients were computed. Multivariate models were adjusted for age, sex, cigarette smoking, left ventricular ejection fraction, NYHA class in the preceding two weeks, mitral regurgitation

Table 1 Baseline demographics and characteristics

Variable	n=174
Median age (IQR)	83.5 (78.4–88.0)
Female	84 (48.3)
Diabetes	59 (33.9)
Hypertension	159 (91.4)
Active smoking	8 (4.6)
Median left ventricular ejection fraction (IQR)	57.5 [43–65]
Median hemoglobin (IQR)	11.1 (9.7–12.6)
Median serum creatinine (IQR)	1.0 (0.8–1.3)
Cerebrovascular accident	18 (10.4)
Atrial fibrillation	75 (43.1)
New York Heart Association Function Class III or IV Heart Failure (within preceding two weeks)	158 (90.8)
Myocardial infarction	80 (45.9)
Percutaneous coronary intervention	71 (40.8)
Coronary artery bypass graft surgery	34 (19.5)
Peripheral arterial disease	40 (23.0)
Median Society of Thoracic Surgeons Mortality Risk Score (IQR)	7.3 (4.7–13.6)
Mean (SD) aortic valve area (cm^2)	0.69 (0.2)
Average (SD) peak/mean aortic valve gradients (mmHg)	71 [23]/42 [15]
Femoral approach	166 (95.4)
Type of valve	
COREVALVE	98 (56.3)
Edwards sapiens	76 (43.7)

Variables presented as number (percentage), unless otherwise noted. IQR, interquartile range; SD, standard deviation.

severity, a history of prior myocardial infarction and prior coronary artery bypass grafting (CABG), and The Society of Thoracic Surgeons (STS) score (Data Version 2.9; http://riskcalc.sts.org/stswebriskcalc/calculate). All analyses were conducted in Stata version 16 (StataCorp. 2019. Stata Statistical Software: Release 16. College Station, TX: StataCorp LLC).

Results

Population characteristics

A total of 174 participants with complete pre and post TAVR data on TR were included in the study. The median age was 84 years with 48% being female. Most participants (91%) were NYHA class III or IV in the preceding 2 weeks. The median (IR) STS score was 7.3% (4.7–13.6%). Median (IR) LVEF was 57% (43–65%). The pre-procedural mean (SD) AVA was 0.69 (0.2) cm^2 and the peak/mean gradients were 71 [23]/42 [15] mmHg. 98 patients (56%) received a CoreValve and the remaining of patients (44%) the Edwards-SAPIEN valve. Trans-apical approach was used in 5 patients, trans-iliac access in 2 and trans-aortic approach in one patient. For the rest of cases (95%), percutaneous-femoral access was used. Post implantation AVA and mean (SD) gradients were 1.64 (0.9) cm^2 and 10 [12] mmHg, respectively. The rest of the baseline (pre-TAVR) characteristics are presented in *Table 1*.

TR severity before and after TAVR

Pre TAVR, 28.7% of patients had moderate or severe TR. The majority of cases had functional TR and only three patients had mixed functional/pacemaker related etiology. After TAVR (*Table 2*) there was an increase in the prevalence of mild TR from 71% to 80% while moderate and severe TR decreased with TAVR from 20% to 13% and 9% to 7%, respectively (P<0.001).

TR severity and cardiovascular disease and all-cause mortalities

Table 3 shows the frequency of both CV and all-cause in-hospital mortalities among participants based on their pre and post-TAVR TR severity. Patients with *no change/worsened* (moderate or severe TR both pre and post-TAVR) had a higher frequency of all-cause and cardiovascular related-mortality when compared with those whose TR *improved* or *remained mild* (*Table 4*).

In both univariate and multivariate analysis, TR pre-TAVR was significantly associated with increased risk of CV and all-cause mortality [relative risk (RR): 14.67 (1.35–159.51) and 5.09 (1.14–22.72), respectively]. Similarly, those who experienced no change/worsened TR

Table 2 Tricuspid regurgitation severity pre versus post-transcatheter aortic valve replacement

Tricuspid regurgitation grade	Pre TAVR (N=174)	Post TAVR (N=174)	P value
Trace/mild (0–1+)	71.3	79.9	<0.001
Moderate (2+)	19.5	12.6	
Severe (3–4+)	9.2	7.5	

Variables presented as percentage. TAVR, transcatheter aortic valve replacement.

Table 3 In-hospital mortality and hospital length of stay analyzed by pre-transcatheter aortic valve replacement tricuspid regurgitation severity

Variable	Timing	Tricuspid regurgitation severity			P value
		Trace/mild (0–1+)	Moderate (2+)	Severe (3–4+)	
All-cause mortality	Pre-TAVR	4/124 (3.2)	7/34 (20.6)	2/16 (12.5)	0.007
	Post-TAVR	7/139 (5.0)	3/22 (13.6)	3/13 (23.1)	0.008
Cardiovascular mortality	Pre-TAVR	2/124 (1.6)	5/34 (14.7)	1/16 (6.3)	0.027
	Post-TAVR	4/140 (2.9)	1/22 (4.5)	3/13 (23.1)	0.003
Median length of hospital stay (days, IQR)	Pre-TAVR	5 [3–10]	4 [6–20]	4 [3–9]	0.756
	Post-TAVR	5 [3–9]	6 [4–11]	15 [4–20]	0.156

Variables presented as number (percentage), unless otherwise noted. TAVR, transcatheter aortic valve replacement.

Table 4 In-hospital mortality and hospital length of stay analyzed by change in tricuspid regurgitation severity after transcatheter aortic valve replacement

Variable	Change in tricuspid regurgitation severity			P value
	Remained mild[a] (N=115)	Improved[b] (N=30)	No change/worsened[c] (N=29)	
All-cause mortality	4/115 (3.5)	3/30 (10.0)	6/28 (21.4)	0.002
Cardiovascular mortality	2/115(1.7)	2/30 (6.7)	4/29 (13.8)	0.005
Median length of hospital stay (days, IQR)	5 [3–10]	4.5 [4–8]	7 [4–20]	0.241

Variables presented as number (percentage), unless otherwise noted. [a], patients with trace/mild tricuspid regurgitation before TAVR and after TAVR; [b], patients with moderate or severe tricuspid regurgitation that improved by 1 or more grades after TAVR; [c], patients with baseline and post-TAVR persistent moderate or severe tricuspid regurgitation. IQR, interquartile range; TAVR, transcatheter aortic valve replacement.

had a significantly increased risk of both CV and all-cause mortality when compared with *improved* or *remained mild*, even after adjusting for possible confounders. Finally, post-TAVR TR was only associated with all-cause and cardiac mortality in univariate analysis (*Table 5*).

TR severity and hospital LOS

As shown in *Tables 3* and *4*, severe post-TAVR TR showed higher hospital LOS relative to those with mild or moderate TR, however, this did not achieve statistical significance on trend analysis. In median regression analysis, there was no association between pre-TAVR TR, or change in TR after TAVR, with hospital LOS. However, those who remained with severe TR post-TAVR had a statistically significantly higher median LOS after controlling for confounders [9.9 (2.9–17.0) days]. Details can be found in *Table 6*.

TR severity and RVSP

Ninety-four of the 174 participants had complete data on RVSP before and after TAVR. The mean (SD) RVSP

Table 5 Risk ratios for in-hospital all-cause and cardiovascular mortality

Variable	All-cause mortality		Cardiac mortality	
	Univariate	Multivariate	Univariate	Multivariate
Pre-TAVR TR (moderate or severe *vs*. mild)	*5.58* (1.72–18.12)	*5.09* (1.14–22.72)	*7.44* (1.50–36.86)	*14.67* (1.35–159.51)
Post TAVR TR (moderate or severe *vs*. mild)	*3.14* (1.14–10.13)	3.21 (0.87–1.79)	3.97 (0.99–15.88)	5.47 (0.93–32.24)
TR change (no change/worsened *vs*. the rest)	*5.94* (1.68–21.08)	*8.19* (1.67–40.29)	*7.93* (1.45–43.3)	*21.5* (1.81–255.96)

Variables presented as risk ratio and 95% CI. Multivariate models were adjusted for age, sex, smoking, left ventricular ejection fraction, mitral regurgitation severity, STS risk score, history of CABG and history of myocardial infarction. Italic indicates statistically significant values. TAVR, transcatheter aortic valve replacement; TR, tricuspid regurgitation.

Table 6 Median regression coefficients for the association between pre and post transcatheter aortic valve replacement tricuspid regurgitation severity and hospital length of stay

Variable	TR severity	Regression coefficients	
		Univariate	Multivariate
Pre-TAVR tricuspid regurgitation severity	Moderate *vs.* mild	1.0 (–1.5 to 3.5)	0.83 (–3.7 to 5.4)
	Severe *vs*. mild	–1.0 (–3.4 to 1.4)	1.61 (–5.6 to 8.9)
Post-TAVR tricuspid regurgitation severity	Moderate *vs*. mild	1.0 (–1.5 to 3.5)	3.5 (–1.5 to 8.5)
	Severe *vs*. mild	*10.0 (6.8 to 13.2)*	*9.9 (2.9 to 17.0)*
TR no change/worsened *vs*. remained mild		2.0 (–0.45 to 4.5)	3.6 (–1.6 to 8.8)

The coefficient regression coefficients represent the difference in hospital stay (days; median, 95% CI). Multivariate models were adjusted for age, sex, smoking, left ventricular ejection fraction, mitral regurgitation severity, STS risk score, history of CABG and history of myocardial infarction. Italic indicates statistically significant values. TAVR, transcatheter aortic valve replacement; TR, tricuspid regurgitation.

Table 7 Pre and post transcatheter aortic valve implantation right ventricular systolic pressure analyzed by baseline tricuspid regurgitation severity

Variable	Tricuspid regurgitation severity		
	None/mild (0–1+) (N=59)	Moderate (2+) (N=26)	Severe (3–4+) (N=9)
Pre-TAVR RVSP	37.5 (10.8)	55.7 (14.5)	61.6 (16.1)
Post-TAVR RVSP	39.2 (11.3)	44.6 (13.2)	47.2 (17.2)
P value	0.326	<0.001	0.072

Ninety-four patients had available pre and post TAVR RVSP. Variables presented as mean (standard deviation). RVSP, right ventricular systolic pressure; TAVR, transcatheter aortic valve implantation.

declined from 46.0 (15.3) to 40.4 (12.6) mmHg with TAVR (P<0.001) (*Table 7*). Among those with mild TR at baseline (n=59), the mean (SD) RVSP did not statistically change from pre to post TAVR (P=0.326). However, among those with moderate TR there was a statistically significant decline in RVSP from a mean (SD) of 55.7 (14.5) to 44.6 (13.2) mmHg (P<0.001), and from 61.6 (16.1) to 47.2 (17.2) mmHg in patients with severe TR (n=9) (P=0.072). Details can be found in *Table 7*. As shown in *Table 8*, among the *remained mild* and no *change/worsened* TR groups, there was no difference in the mean RVSP with TAVR. However, in patients with *improved* TR after TAVR, there was a significant reduction in the mean RVSP from 58.5 (15.6) to 40.4 (14.5) mmHg (P<0.001).

Discussion

In this cohort of high surgical risk individuals with severe AS undergoing TAVR, the following important observations were noted post-intervention: (I) approximately one-third of patients had moderate or greater TR (≥2+), of

Table 8 Right ventricular systolic pressure analyzed by change in tricuspid regurgitation severity after transcatheter aortic valve replacement

Variable	Change in tricuspid regurgitation severity		
	Remained mild[a] (N=54)	Improved[b] (N=20)	No change/worsened[c] (N=20)
Pre-TAVR right ventricular systolic pressure	39.3 (11.8)	58.5 (15.6)	51.6 (14.2)
Post-TAVR right ventricular systolic pressure	36.5 (10.5)	40.4 (14.5)	51.0 (9.9)
P value	0.146	<0.001	0.855

[a], patients with trace/mild tricuspid regurgitation before TAVR and after TAVR; [b], patients with moderate or severe tricuspid regurgitation that improved by 1 or more grades after TAVR; [c], patients with baseline and post-TAVR persistent moderate or severe tricuspid regurgitation. 94 patients have available pre and post TAVR RVSP. Variables presented as mean (standard deviation). TAVR, transcatheter aortic valve replacement.

which less than half had improvement in TR with TAVR; (II) pre and post-TAVR TR severity were associated with increased in-hospital CV and all-cause mortality, and those with moderate or greater TR who *remained same/worsened* had the least favorable outcomes; (III) patients with severe TR post-TAVR experienced the longest hospital LOS; (IV) RVSP was independently associated with TR severity, and a reduction of RVSP after TAVR was associated with improvement in TR severity; and, (V) RVSP remained unchanged post-TAVR in those in whom TR *remained same/worsened*.

The prevalence of significant TR in this cohort was similar to that reported in prior studies of TAVR patients (8-14). Moreover, our findings agree with results from other observational studies reporting increased in-hospital, 30-day and one-year mortality for those patients with significant TR undergoing TAVR (8,13). In agreement with the literature (18,19), the etiology of TR in the great majority of these patients with severe AS was functional. Hence, TR was related to elevated ventricular pressures and pulmonary hypertension (PHT). TR severity can be a marker of high risk individuals. In fact, the severity of TR increases along with the risk estimated by the STS score (13). However, the presence of moderate or severe TR increased the risk of in-hospital mortality independently of the STS scores, MR severity and LV ejection fraction. Thus, moderate or severe TR is an independent risk factor for post TAVR in-hospital complications in high surgical risk individuals.

In our study, TAVR improved TR severity; however, the early salutary effect of TAVR on TR was only seen in a third of patients, and mostly in those with moderate TR (35% improvement), with a very modest effect (18% improvement) in patients with severe TR. Hence, the great majority of individuals with severe TR had persistent moderate to severe TR post-TAVR. The reported improvement of TR grade in prior studies ranges from 15% to 50% following TAVR (8-14) and surgical AVR (6). Thus, in a sizable percentage of individuals with moderate or greater TR at the time of TAVR, TR severity remains stable or worsens after the procedure (*remained same/worsened*). Importantly, mortality was the highest in this subgroup of patients. Interestingly, Worku *et al*. showed that in those with moderate or severe TR post TAVR, TR severity is unchanged or worsen after one year, and is associated with worse long-term survival (12). Hence, these observations suggest that this subgroup of individuals may benefit from additional tricuspid valve intervention.

In the present study, RVSP was higher in patients with significant TR. RVSP improved after TAVR but remained high in those in whom *remained same/worsened*, indicating that persistent PHT may play an important etiological role in the pathophysiology of persistent TR post-TAVR. Likewise, Worku *et al*. (12) and McCarthy *et al*. (13) found that in TAVR patients, RVSP increased along with TR severity and was associated with increased mortality (12,13). Alushi *et al*. investigated the role of PHT in patients undergoing TAVR (20). The authors found that in most patients post TAVR there is an early and late reduction of pulmonary pressures. Moreover, those patients with reversible PHT were at lower risk of all-cause mortality, however, in patients with residual PHT there was a higher risk of all-cause mortality at 30 days, 1 year and long-term. Importantly, absence of significant TR was an independent predictor of pulmonary pressure reduction post TAVR (20). These observations suggest that preoperative (or at the time of TAVR) right heart catheterization in a selected group of patients (e.g., those with RV dysfunction) with significant TR/elevated RVSP may help to further risk stratify and tailor medical treatment pre or immediately post-TAVR.

Limitations

This was a retrospective study and it is subject to information bias. Moreover, the analysis was conducted in a cohort of high risk individuals and it is unclear if these findings would apply to moderate or low risk TAVR patients. We relied upon incomplete hemodynamic data obtained by echocardiography to estimate RVSP, which results in a form of attrition bias. Moreover, RVSP may be underestimated in patients with severe TR. However, the association of RVSP and TR severity after TAVR remained when individuals with severe TR were excluded from the analysis. TR and RVSP were evaluated only immediately post TAVR and their severity may vary in the short-term post TAVR. However, this would not change the hospital outcomes measured in the present analysis.

Conclusions

In this cohort of high surgical risk individuals with severe AS undergoing TAVR, moderate or greater TR was associated with increased in-hospital CV and all-cause mortality. Furthermore, patients who remained with significant TR after TAVR had the least favorable outcomes. RVSP correlated with TR severity and improved after TAVR, however, RVSP remained elevated in those patients in whom TR remained significant post TAVR, suggesting that residual significant PHT contributes to significant TR and poor outcomes post TAVR.

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