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Predictors and Prognostic Implications of Myocardial Injury After Transcatheter Aortic Valve Replacement

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Myocardial injury (MI) is not unusual after transcatheter aortic valve replacement (TAVR). To determine precipitating factors and prognostic outcomes of MI after TAVR, we retrospectively investigated relationships between MI after TAVR and aortic root dimensions, baseline patient characteristics, echocardiographic findings, and procedural features.

Of 474 patients who underwent transfemoral TAVR for severe aortic stenosis in our tertiary center from June 2011 through June 2018, 188 (mean age, 77.7 ± 7.7 yr; 96 women [51%]) met the study inclusion criteria. Patients were divided into postprocedural MI (PMI) (n=74) and no-PMI (n=114) groups, in accordance with high-sensitivity troponin T levels.

We found that MI risk was associated with older age (odds ratio [OR]=1.054; 95% CI, 1.013–1.098; P=0.01), transcatheter heart valve type (OR=10.207; 95% CI, 2.861–36.463; P=0.001), distances from the aortic annulus to the right coronary artery ostium (OR=0.853; 95% CI, 0.731–0.995; P=0.04) and the left main coronary artery ostium (OR=0.747; 95% CI, 0.616–0.906; P=0.003), and baseline glomerular filtration rate (OR=0.985; 95% CI, 0.970–1.000; P=0.04). Moreover, the PMI group had a longer time to hospital discharge (P=0.001) and a higher permanent pacemaker implantation rate (P=0.04) than did the no-PMI group.

Our findings may enable better estimation of which patients are at higher risk of MI after TAVR and thus improve the planning and course of clinical care. ***(Tex Heart Inst J 2022;49(4):e207380)***

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Aortic stenosis progresses slowly and has a long latency period. Usually, there is an extended period between disease onset and the emergence of symptoms. However, once symptoms develop, the disease progresses rapidly.[1,2] Surgical aortic valve replacement (AVR) improves symptoms and reduces mortality rates in comparison with conservative medical therapy.[3,4] Approximately 30% of patients with severe aortic stenosis are not candidates for surgical AVR, because they either are at high surgical risk or are otherwise inoperable.[5] The need for less-invasive treatment options for these patients prompted the development of transcatheter AVR (TAVR), which soon became the preferred technique in patients with severe aortic stenosis in whom surgery would pose an intermediate or high procedural risk.[5,6]

Myocardial injury (MI) and myocardial infarction are frequent complications of TAVR that can have severe consequences for patients. The Valve Academic Research Consortium (VARC)-2 defines MI as an increase in cardiac troponin values more than 15 times the upper reference limit or a 5-fold increase in the creatine kinase-MB isoform (CK-MB) within 72 hours after TAVR.[7] Embolization to one or more coronary arteries is also crucial in the pathogenesis of MI after TAVR.[8] For these reasons, aortic root dimensions are predictive factors for the development of MI. However, to our knowledge, data regarding the anatomic properties of the aortic root and its measurements are lacking.

We investigated the relationship between the post-TAVR development of MI and aortic root dimensions, baseline patient characteristics, echocardiographic findings, and procedural features, and we report our findings.

Patients and Methods

In this retrospective study, patients with severe aortic stenosis who underwent TAVR at our center from June 2011 through June 2018 were identified from electronic medical records, during clinic visits, and by telephone. The diagnosis of aortic stenosis had been made by a multidisciplinary heart team on the basis of clinical and comorbid conditions. Patients were excluded from the study if they had acute decompensated heart failure when they were referred for TAVR, myocardial infarction within 4 weeks after referral, or a major complication during or after the procedure; or if they died before any postoperative blood samples were taken or if blood sampling for measuring cardiac troponin T (TnT) levels was not done at prespecified times (baseline and 4, 12, 24, and 48 hr after the procedure).

The European System for Cardiac Operative Risk Evaluation (EuroSCORE)[9] and Society of Thoracic Surgeons (STS) scores[10] were calculated by using online calculators. Coronary angiograms were used to determine the presence of coronary artery disease (CAD). In-hospital death and death at 30 days and 1 year were used to determine the clinical outcomes of MI.

This retrospective study was approved by our hospital ethics committee. All patients had given written informed consent before undergoing TAVR.

Laboratory Data

All laboratory findings obtained before and after TAVR were extracted from medical records. Blood samples were taken before the procedure and 4, 12, 24, and 48 hours after. High-sensitivity TnT (hs-TnT) levels were measured with use of Elecsys Troponin T high-sensitivity assays (Roche Diagnostics) in Cobas E 601 analyzers (Roche), and CK-MB levels with use of a Roche Hitachi 911 Chemistry Analyzer. A diagnosis of MI was made if hs-TnT levels were more than 15 times the upper reference limit within 72 hr of the procedure, or if cardiac biomarker levels were already elevated at baseline (>99th percentile) or increased more than 50% after TAVR versus before TAVR.[7]

Echocardiography

All patients undergoing TAVR in our clinic underwent echocardiography with use of the Philips iE33 device and PHRULTIE33 system (Philips Medical Systems). Patients underwent evaluation at baseline and then postoperatively at 24 hours, 30 days, and 1 year, unless an urgent medical condition necessitated earlier examination. Left ventricular ejection fraction, AV maximal and mean pressure gradients, aortic annulus diameters, AV area, ascending aortic diameters, postprocedural pressure gradients of the bioprosthetic valve, and aortic insufficiencies were recorded and analyzed.

Multislice Computed Tomography

We examined computed tomographic (CT) images obtained preprocedurally with use of a Somatom Definition Flash 256-slice CT scanner (Siemens Medical Solutions). Diameters, ellipticity indices, areas, and perimeters of the aortic annulus, sinus of Valsalva, and sinotubular junction were recorded, and distances between the aortic annulus and left main coronary artery (LMCA) and the aortic annulus and right coronary artery (RCA) were measured.

Procedural Preparation

Doppler and 2-dimensional transthoracic echocardiograms obtained in parasternal long-axis, parasternal short-axis, apical 4-chamber, and apical 3-chamber views were used for initial evaluation of the AV. Transesophageal echocardiography was performed when evaluable transthoracic images could not be obtained. Multislice CT enabled preprocedural evaluation of valve morphology, the aortic annulus, aortic annulus–LMCA and aortic annulus–RCA distances, and the availability of peripheral arteries for TAVR. Coronal, sagittal, and axial CT images of the annulus were reconstructed with use of the OsiriX MD v.9.5 program (DICOM viewer and image-analysis program; Pixmeo SARL). An experienced operator performed annular measurements.

A transfemoral route was used for all TAVR procedures. Coronary angiography was performed in any patient who had not undergone it in the past 6 months. We defined CAD either as coronary plaque exceeding 50% in diameter on coronary angiograms or as previous myocardial infarction, coronary artery bypass grafting, or percutaneous coronary intervention.

Statistical Analysis

We used SPSS version 22.0 for Windows (SPSS, an IBM company) for all statistical analysis. $P < 0.05$ was considered statistically significant. Descriptive statistics were presented as number and percentage for categorical variables and as mean ± SD for continuous variables. Visual (histograms and probability graphics) and analytic (Kolmogorov-Smirnov or Shapiro-Wilk tests) criteria were used to evaluate normal distribution of continuous variables. The independent-sample *t* test was used for comparative analysis between 2 groups for normally distributed data. The Pearson-Fisher exact test and χ^2 test were used for comparative analysis between independent groups for categorical variables.

Factors affecting MI development were analyzed by using univariate logistic regression. Independent factors for MI development risk that were statistically relevant

(P <0.05) in univariate logistic regression analysis were used in multivariate logistic regression analysis.

Results

During the study period, 474 patients with severe aortic stenosis underwent TAVR. After the exclusion criteria were applied, the cases of 188 patients were retrospectively analyzed (Fig. 1). The mean age was 77.7 ± 7.7 years, and 96 (51%) were women. The mean STS score was 6.98 ± 3.91, and the mean EuroSCORE was 22.07 ± 14.4 (Table I).

Patients were divided into 2 groups: those with postprocedural MI (PMI) and those without PMI. We found that 74 patients (39.4%) had PMI, and 114 (60.6%) did not. The PMI group was older (79.5 ± 8.0 vs 76.5 ± 7.3 yr; P=0.009) and had a lower baseline glomerular filtration rate (GFR) (60.39 ± 20.13 vs 66.38 ± 19.17 mL/min/1.73 m^2; P=0.04) when compared with the no-PMI group.

Among the preprocedural CT findings, the aortic annulus–LMCA ostium distance was shorter in the PMI group than in the no-PMI group (13.03 ± 1.47 vs 13.79 ± 1.74 mm; P=0.002), as was the aortic annulus–RCA ostium distance (13.61 ± 1.98 vs 14.26 ± 2.19 mm; P=0.04) (Table II).

Edwards SAPIEN XT valves (Edwards Lifesciences Corporation) were implanted in 129 patients (68.6%), Edwards SAPIEN 3 valves (Edwards Lifesciences) in 40 (21.3%), and LOTUS valves (Boston Scientific Corporation) in 19 (10.1%). The 4 patients (2.1%) in whom surgical AVR had been performed underwent valve-in-valve TAVR. We detected MI in 16 (84.2%) of the 19 patients who underwent LOTUS valve implantation, and MI was significantly more prevalent in patients who received a LOTUS valve versus either Edwards valve (P=0.001) (Table III).

Permanent pacemaker (PPM) implantation was performed in 27 patients (14.4%) after TAVR (Table IV). The mean time between implantation and discharge from the hospital was 5.3 ± 3.4 days. The PPM implantation rate was higher in the PMI group than in the no-PMI group (21.6% vs 9.6%; P=0.04), and time to hospital discharge was also longer (6.3 ± 4.1 vs 4.7 ± 2.7 d; P=0.001). The in-hospital mortality rate was 1.1% (2 patients in the PMI group), and the 30-day mortality rate was 3.8% (7 patients). The cumulative 1-year mortality rate after the procedure was 15.4% (29 patients). The 2 groups had similar in-hospital and follow-up mortality rates.

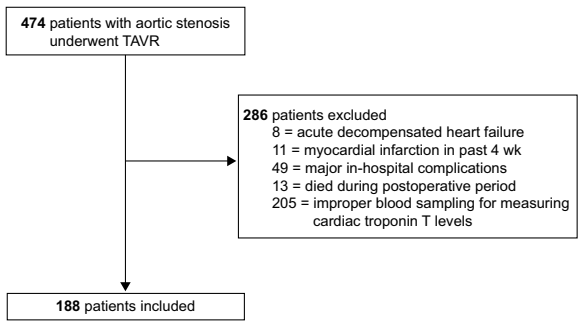

Fig. 1 *Diagram shows the study inclusion process.*

TAVR = transcatheter aortic valve replacement

We used multivariate logistic regression to investigate the prognostic value of variables found to be significantly related to MI development after TAVR as indicated by the increase in hs-TnT values. Independent predictors were older age (odds ratio [OR]=1.054; 95% CI, 1.013–1.098; P=0.01), LOTUS valve implantation (OR=10.207; 95% CI, 2.861–36.463; P=0.001), aortic annulus–LMCA ostium distance (OR=0.747; 95% CI, 0.616–0.906; P=0.003), aortic annulus–RCA ostium distance (OR=0.853; 95% CI, 0.731–0.995; P=0.04), and basal GFR value (OR=0.985; 95% CI, 0.970–1.000; P=0.04) (Table V).

Discussion

Our study produced several major findings. First, procedure-related MI developed in 39.4% of our patients. Second, although few patients were given a LOTUS valve, that valve type was associated with MI after TAVR. Third, older age, the distance of the aortic annulus to each of the coronary ostia, and baseline GFR value were independent predictors of MI development after TAVR. Fourth, hospital stays were longer and PPM implantation rates were higher in the PMI group. Fifth, development of MI caused no difference in mortality rates between groups.

For more than 50 years, surgical AVR was the only means of controlling symptoms and increasing survival rates in patients with severe aortic stenosis. After its first use in human beings in 2002, however, TAVR became an alternative for patients who are not surgical candidates, and it is now extensively used worldwide.[6] Given advances in catheter-based techniques and the development of new-generation AVs, TAVR has become the preferred approach for patients who are at intermediate or high surgical risk.[11]

However, the widespread use of TAVR and other percutaneous cardiac procedures has made MI a serious complication associated with adverse events and poor prognosis.[12] Elevation in cardiac biomarkers (Tn and CK-MB) is the reference standard for diagnosing MI.[13] As highly sensitive, new-generation Tn assays become increasingly available, elevated biomarker levels can be more readily detected after almost all cardiac interventions.

In this study, MI was detected in 74 patients (39.4%) during the first 48 hours after TAVR, according to

TABLE I. Baseline Characteristics of the Patients

Variable	All Patients (N=188)	No PMI (n=114)	With PMI (n=74)	*P* Value
Age (yr)	77.7 ± 7.7	76.5 ± 7.3	79.5 ± 8.0	0.009
Female	96 (51.0)	50 (43.9)	46 (62.2)	0.13
Body mass index	27.98 ± 7.09	28.49 ± 7.77	27.19 ± 5.88	0.22
STS score	6.98 ± 3.91	6.91 ± 4.07	7.10 ± 3.67	0.74
EuroSCORE (%)	22.07 ± 14.40	22.18 ± 14.79	21.92 ± 13.87	0.91
Coronary artery disease	—	—	—	0.47
None	16 (8.5)	8 (7.0)	8 (10.8)	—
Nonobstructive	116 (61.7)	69 (60.5)	47 (63.5)	—
Obstructive	56 (29.8)	37 (32.5)	19 (25.7)	—
NYHA functional class	—	—	—	0.42
II	12 (6.4)	6 (5.3)	6 (8.1)	—
III	132 (70.2)	84 (73.7)	48 (64.9)	—
IV	44 (23.4)	24 (21.1)	20 (27.0)	—
Medical history				
CABG	51 (27.1)	33 (28.9)	18 (24.3)	0.49
PCI	31 (16.5)	16 (14.0)	15 (20.3)	0.26
Valve surgery	10 (5.3)	9 (7.9)	1 (1.4)	0.46
Myocardial infarction	58 (30.9)	33 (28.9)	25 (33.8)	0.48
Stroke	13 (6.9)	8 (7.0)	5 (6.8)	0.95
Peripheral artery disease	67 (35.6)	39 (34.2)	28 (37.8)	0.61
COPD	54 (27.7)	30 (26.3)	24 (32.4)	0.56
Diabetes	64 (34.0)	38 (33.3)	26 (35.1)	0.80
Hypertension	173 (92.0)	105 (92.1)	68 (91.9)	0.96
Hyperlipidemia	68 (36.2)	44 (38.6)	24 (32.4)	0.39
Atrial fibrillation	54 (28.7)	33 (28.9)	21 (28.4)	0.93
Chronic kidney disease	41 (21.8)	23 (20.2)	18 (24.3)	0.50
Baseline GFR (mL/min/1.73 m^2)	64.02 ± 19.72	66.38 ± 19.17	60.39 ± 20.13	0.04
Echocardiographic findings				
LV ejection fraction (%)	52.3 ± 13.5	50.6 ± 13.4	52.1 ± 14.6	0.46
LV mass (g)	149.1 ± 37.9	152.9 ± 39.2	143.7 ± 40.9	0.12
Aortic valve area (cm^2)	0.69 ± 0.17	0.69 ± 0.17	0.68 ± 0.18	0.89
Aortic gradient (mmHg)	50.8 ± 15.8	50.5 ± 15.8	51.3 ± 15.8	0.73
sPAP (mmHg)	46.0 ± 15.5	44.6 ± 14.7	48.2 ± 16.4	0.15

CABG = coronary artery bypass grafting; COPD = chronic obstructive pulmonary disease; EuroSCORE = European System for Cardiac Operative Risk Evaluation; GFR = glomerular filtration rate; LV = left ventricular; NYHA = New York Heart Association; PCI = percutaneous coronary intervention; PMI = postprocedural myocardial injury; sPAP = systolic pulmonary artery pressure; STS = Society of Thoracic Surgeons

Data are presented as mean ± SD or as number and percentage. P <0.05 was considered statistically significant.

VARC-2 criteria (an elevation of hs-TnT >15 times the upper reference limit within 72 hr of the procedure).[7] In the medical literature, MI rates after TAVR have varied between 16% and 99%.[14] Differences between studies in how MI was identified, the use of a transfemoral or transapical route, the analyses used to determine Tn values, and the heterogeneity of patient groups may explain this wide range of rates. In a study in which VARC-2 criteria were used to detect MI after solely transfemoral TAVR,[15] high-sensitivity TnI was elevated in 51.6% of patients and CK-MB in 7.4%. In a different study with similar criteria, the MI rate was 77%.[16] The relatively low MI rates in our study may be due to use of the transfemoral route in all patients and exclusion of patients with major postprocedural complications that could increase cardiac biomarker levels. Of note, MI is

TABLE II. Comparison of Multislice Computed Tomographic Values

Variable	All Patients (N=188)	No PMI (n=114)	With PMI (n=74)	*P* Value
Aortic annulus size (mm)				
Large diameter	25.70 ± 3.77	24.44 ± 3.22	24.67 ± 3.42	0.20
Small diameter	21.80 ± 2.27	21.92 ± 2.20	21.61 ± 2.37	0.36
Perimeter	77.13 ± 6.41	77.66 ± 6.11	76.31 ± 6.81	0.16
Sinus of Valsalva size				
Large diameter (mm)	30.54 ± 3.57	30.58 ± 3.93	30.49 ± 2.96	0.86
Small diameter (mm)	28.76 ± 3.83	28.94 ± 4.17	28.49 ± 3.23	0.43
Area (mm^2)	783.40 ± 190.12	790.40 ± 205.74	772.6 ± 1 64.0	0.53
Perimeter (mm)	98.60 ± 11.54	98.96 ± 12.36	98.04 ± 10.21	0.59
Sinotubular junction				
Area (mm^2)	683.86 ± 189.44	694.04 ± 211.85	668.17 ± 148.48	0.36
Perimeter (mm)	91.56 ± 11.83	92.51 ± 1 2.92	91.10 ± 9.96	0.43
AA–LMCA ostium distance (mm)	13.48 ± 1.68	13.79 ± 1.74	13.03 ± 1.47	0.002
AA–RCA ostium distance (mm)	14.01 ± 2.13	14.26 ± 2.19	13.61 ± 1.98	0.04

LMCA = left main coronary artery; MI = myocardial injury; RCA = right coronary artery

Data are presented as mean ± SD. *P* <0.05 was considered statistically significant.

TABLE III. Procedural Characteristics

Variable	All Patients (N=188)	No PMI (n=114)	With PMI (n=74)	*P* Value
Predilation	124 (66.0)	70 (61.4)	54 (73.0)	0.10
Postdilation	1 (0.5)	1 (0.9)	0	0.61
Valve type	—	—	—	0.001
SAPIEN XT	129 (68.6)	83 (72.8)	46 (62.2)	—
SAPIEN 3	40 (21.3)	28 (24.6)	12 (16.2)	—
LOTUS	19 (10.1)	3 (2.6)	16 (21.6)	—
Valve size (mm)				
SAPIEN XT or 3 (n=169)	—	—	—	0.94
23	55 (32.5)	37 (33.3)	18 (31)	—
26	81 (47.9)	53 (47.7)	28 (48.3)	—
29	33 (19.5)	21 (18.9)	12 (20.7)	—
LOTUS (n=19)	—	—	—	0.57
23	8 (42.1)	2 (66.7)	6 (37.5)	—
25	8 (42.1)	1 (33.3)	7 (43.8)	—
27	3 (15.8)	0	3 (18.8)	—
Valve-in-valve implantation	4 (2.1)	4 (3.5)	0	0.13

PMI = postprocedural myocardial injury

Data are presented as number and percentage. *P* <0.05 was considered statistically significant.

expected to develop less frequently after TAVR than after surgical AVR because TAVR involves no cardioplegia, aortic clamping, or other procedural steps.

Hypotheses have been proposed to explain MI during TAVR. Cardiac biomarkers are elevated in most patients with severe aortic stenosis even in the absence of intervention, suggesting that a stenotic AV increases intraventricular pressure. The transapical route is a major cause of injury because a catheter is inserted directly through the myocardium.[17] Hypotension during valve implantation and rapid ventricular pacing may cause MI by altering the oxygen supply to the myocardium.[18] Predilation

TABLE IV. Clinical Outcomes

Variable	All Patients (N=188)	No PMI (n=114)	With PMI (n=74)	*P* Value
Permanent pacemaker implantation	27 (14.4)	11 (9.6)	16 (21.6)	0.04
Death				
In-hospital	2 (1.1)	0	2 (2.7)	0.15
30-day	7 (3.8)	4 (3.5)	3 (4.2)	0.55
6-month	7 (3.8)	4 (3.5)	3 (4.2)	0.55
1-year	13 (7.0)	11 (9.6)	2 (2.8)	0.07
Cumulative 1-year	29 (15.4)	19 (16.7)	10 (13.5)	0.42
Discharge from hospital (d)	5.3 ± 3.4	4.7 ± 2.7	6.3 ± 4.1	0.001

PMI = postprocedural myocardial injury

Data are presented as number and percentage or as mean ± SD. *P* <0.05 was considered statistically significant.

TABLE V. Independent Predictors of MI After Transcatheter Aortic Valve Implantation

Variable	Odds Ratio (95% CI)	*P* Value
Age	1.054 (1.013–1.098)	0.01
LOTUS valve	10.207 (2.861–36.463)	0.001
Aortic annulus–LMCA distance	0.747 (0.616–0.906)	0.003
Aortic annulus–RCA distance	0.853 (0.731–0.995)	0.04
Baseline GFR	0.985 (0.970–1.000)	0.04

GFR = glomerular filtration rate; LMCA = left main coronary artery; MI = myocardial injury; RCA = right coronary artery

P <0.05 was considered statistically significant.

of the native AV, postdilation of the bioprosthetic valve, and MI during manipulation of catheters and guidewires may also contribute to PMI. One of the most important causes of injury is the embolization of calcific material from the native AV to the coronary arteries.[19] Intraoperative or postoperative complications also may contribute to MI development.[20] Moreover, occlusion of the coronary artery ostium by the bioprosthetic valve or native AV cusps may cause substantial MI and a severe clinical course.[21]

The type of bioprosthetic AV is another important factor affecting the development of MI. Among the 3 valve types used in our study (SAPIEN XT, SAPIEN 3, and LOTUS), the LOTUS valve increased our patients' risk for MI. Stundl and colleagues[15] similarly showed that MI developed more often in patients who had undergone LOTUS valve implantation. Myocardial tissue compression and mechanical trauma caused by the additional adaptive seal around the outer aspect of the lower valve frame (designed to minimize paravalvular leaks) may be the mechanism. Moreover, a self-expanding aortic prosthesis like the LOTUS valve requires extended and continuous pressure application over the aortic annulus. This contrasts with balloon-expanding valves like the SAPIEN types, in which high pressures are applied briefly. Longer procedural times and potential resheathing processes may also contribute to MI development during LOTUS implantation. Considering that the SAPIEN 3 valve has a seal around its outer rim but is associated with lower MI rates, the last 3 mechanisms may most readily explain the high rates of MI during LOTUS valve implantation.[15]

Aortic root and AV anatomy also contribute to MI development. In one investigation of MI,[19] post-TAVR cardiac magnetic resonance detected small, subendocardial, or mural localized, multifocal embolic lesions, and the investigators concluded that the migration of microembolic material from the AV to the coronary arteries during TAVR contributed substantially to MI. In the same study, an inverse relationship was found between the aortic annulus–coronary ostium distance and MI ratios, similar to our results; however, unlike our results, the correlation was not statistically significant.[19] In an experimental study in porcine hearts,[22] multiple embolic particles were detected in all coronary arteries after in vitro balloon valvuloplasty of calcified AVs.

The distance between the aortic annulus and the coronary artery ostium is inversely related to the risk for total or subtotal occlusion of the coronary arteries during TAVR.[21] Our study contributes to the literature by reporting a statistically significant inverse relationship between the risk for MI and the distance between the aortic annulus and each of the coronary ostia, even though there was no total or subtotal obstruction of the coronary arteries. The proximity of the coronary ostium to the aortic annulus possibly affects the density of the

calcified material that embolizes from the AV to the coronary arteries.

Our highly selective study was conducted in a homogeneous cohort; all procedures were transfemoral, and patients with major complications were excluded, thereby eliminating the effects of multiple confounding factors that contribute to MI. This refinement in patient selection ensures that our data on the inverse relation between MI risk and aortic annulus–coronary ostium distance are even more meaningful than are other data in the literature. Our data also strengthen the hypothesis that embolic material emitted from the calcified AV during TAVR distinctly affects MI development. This embolic material may play a role in other clinical conditions, as well; asymptomatic cerebral emboli have been reported in 84% of patients after TAVR.[23] Our data may influence clinicians to avoid unnecessary AV manipulation and to select prostheses with less MI risk when performing TAVR in patients who have shorter aortic annulus–coronary ostium distances.

Whether CAD increases the risk for MI after TAVR is not settled. Although Koskinas and colleagues[24] found that MI was more prevalent among patients with complex CAD, other investigators found no such relationship. The 188 patients in our study had normal coronary arteries (n=16; 8.5%), nonobstructive CAD (n=116; 61.7%), or obstructive CAD (n=56; 29.8%). Because PCI within 4 weeks before TAVR was an exclusion criterion in our study, the obstructive-CAD group comprised patients who were managed medically or were scheduled for revascularization after TAVR.

We found no significant relationship between the presence of CAD and the development of MI. Four patients had previously undergone surgical AVR and then underwent valve-in-valve TAVR, and none sustained MI. Despite the lack of data to prove it, a previously implanted bioprosthetic AV may prevent the new AV from compressing the myocardium, and less embolic material may be dislodged from these patients' AVs.[24] Larger studies are needed to characterize this pathophysiologic process.

Patients in whom VARC-2 class 2–3 acute kidney injury developed after TAVR were considered to have a major complication and were excluded from our study. We found a reverse relationship between patients' baseline GFR values and MI risk. Kidney failure itself is an independent factor for chronic cardiac biomarker elevation and MI, even without cardiac intervention. Direct toxic effects of elevated urea levels on the myocardium and increased myocardial stretch due to amplified intravascular volume are possible contributing factors.[25] Others' findings regarding the effect of baseline renal function on MI development are similar to our study's outcomes.[26,27]

Although one study[26] showed a significant relationship between rapid ventricular pacing time and MI, Koifman and associates[16] found none between rapid pacing times and myocardial damage. Activated clotting time (ACT) was strictly monitored, with additional heparin doses administered as needed to maintain times longer than 250 sec. In our study, however, we did not measure ACT.

We found relationships between MI and both the duration of hospital stay and the need for PPM implantation. Another study with findings similar to ours also noted a relationship between MI and the need for PPMs.[16] Conduction defects after TAVR are associated with mechanical compression of the aortic bioprosthesis onto myocardial tissue, involving conduction fibers. Given that MI results from a similar pathophysiologic process, the relationship between MI and the need for PPMs is not surprising.

We found no relationships between MI development and mortality rates in our study. Evidence in the literature regarding these relationships is contradictory. Stundl and colleagues[15] found no correlation between MI defined as an elevation of both CK-MB and Tn values and mortality rates at 30 days and 1 year.[13] Chorianopoulos and associates[26] also found no correlation between MI defined by TnT elevation and mortality rates at 30 days and 1 year.[24] In contrast, Koskinas and coworkers[24] found that MI defined by elevated TnT values was related to increased 30-day and 1-year mortality rates.[14] In a study by Koifmann and colleagues,[16] CK-MB elevation, but not TnT elevation, was associated with 1-year mortality rates. Similarly, Yong[28] and Ribeiro[29] and their colleagues found a relationship between CK-MB elevation and 30-day mortality rates. A subgroup analysis of the PARTNER study by Paradis and associates[8] revealed a relationship between MI and 30-day mortality rates, whereas 1-year mortality rates were related only to elevated CK-MB levels. Last, a meta-analysis of 9 studies by Michail and colleagues[30] revealed an association between MI and a significantly increased risk for 30-day and 1-year all-cause death.

Elevated TnT is more sensitive and specific than is CK-MB elevation in detecting MI; however, with the widespread application of hs-TnT assays, elevated TnT levels can be detected after even minor cardiovascular procedures.[31] For this reason, larger studies are needed to define new Tn cutoff values for predicting mortality rates after TAVR.

Study Limitations

The main limitation of this study is its retrospective, single-center design. Other limitations include the absence of data on total procedure recording, rapid ventricular pacing, and ACT. In addition, having fewer patients in the LOTUS group than in the other valve groups might have introduced bias into certain comparisons, and the few patients in the LOTUS group might have affected the positive correlation between

LOTUS valve implantation and MI incidence. Further research in wider patient populations is needed to confirm the relationship between prosthesis type and MI risk.

Conclusion

The clinical presentation of patients with MI can vary widely, from asymptomatic to exhibiting severe complications. We found that older age, AV type, baseline GFR value, and the distances from the aortic annulus to the LMCA ostium and to the RCA ostium were significantly related to MI risk. In addition, we found that MI was associated with longer hospital stays and a high risk for PPM implantation after TAVR. Use of the LOTUS valve may be associated with greater MI risk. Our findings may influence clinicians to select bioprostheses with less MI risk when performing TAVR in patients who have shorter annulus-ostium distances.

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