# Mitral Valve Infective Endocarditis after Trans-Catheter Aortic Valve Implantation

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**Scarce data exist on mitral valve (MV) infective endocarditis (IE) after transcatheter aortic valve implantation (TAVI). This multicenter study included a total of 579 patients with a diagnosis of definite IE after TAVI from the IE after TAVI International Registry and aimed to evaluate the incidence, characteristics, management, and outcomes of MV-IE after TAVI. A total of 86 patients (14.9%) had MV-IE. These patients were compared with 284 patients (49.1%) with involvement of the transcatheter heart valve (THV) only. Two factors were found to be associated with MV-IE: the use of self-expanding valves (adjusted odds ratio 2.49, 95% confidence interval [CI] 1.23 to 5.07, p = 0.012), and the presence of an aortic regurgitation ≥2 at discharge (adjusted odds ratio 3.33; 95% CI 1.43 to 7.73, p <0.01). There were no differences in IE timing and causative microorganisms between groups, but surgical management was significantly lower in patients with MV-IE (6.0%, vs 21.6% in patients with THV-IE, p = 0.001). All-cause mortality rates at 2-year follow-up were high and similar between patients with MV-IE (51.4%, 95% CI 39.8 to 64.1) and patients with THV-IE (51.5%, 95% CI 45.4 to 58.0) (log-rank p = 0.295). The factors independently associated with increased mortality risk in patients with MV-IE were the occurrence of heart failure (adjusted p <0.001) and septic shock (adjusted p <0.01) during the index hospitalization. One of 6 IE episodes after TAVI is localized on the MV. The implantation of a self-expanding THV and the presence of an aortic regurgitation ≥2 at discharge were associated with MV-IE. Patients with MV-IE were rarely operated on and had a poor prognosis at 2-year follow-up.**  **(Am J Cardiol 2022;172:90−97)**

**Abbreviations:** HF, heart failure; IE, infective endocarditis; MV, mitral valve; PVL, paravalvular leaks; TAVI, transcatheter aortic valve implantation; THV, transcatheter heart valve

Infective endocarditis (IE) after transcatheter aortic valve implantation (TAVI) is a relatively rare (5-year incidence of ∼6%) but life-threatening (mortality of 66% at 1 year) complication.[1,2] Numerous studies have investigated the clinical characteristics and main outcomes of IE after TAVI.[3,4] Unexpectedly, it has been shown that mitral valve (MV) IE is common after TAVI as it occurs in about 1 in 5 cases.[2] The extension of an aortic IE to the MV, related to the anatomic adjacency of the aortic ring, the intervalvular fibrosa, and the anterior mitral leaflet, has already been reported before the transcatheter era.[5,6] Some reports have also described mitral perforation and endocardial lesions because of the friction of the valve stent frame leading to mitral IE after TAVI.[7−10] However, the mechanisms, risk factors, and prognosis of MV-IE after TAVI remain unknown. Thus, we sought to evaluate the incidence, clinical characteristics, and outcomes of MV-IE after TAVI.

## Methods

Data were collected from The Infectious Endocarditis after TAVI International Registry. Details on the design of this observational, multicenter, international registry have been previously reported.[11] At the time of this analysis, this registry included data from 579 patients with definite IE after TAVI from 59 centers in 11 countries across Europe, North America, and South America between June 2005 and November 2020. Only patients presenting with definite IE on the transcatheter heart valve (THV) or the MV were included in the statistical analysis regardless of the cardiac structure affected (native or prosthetic valve).

Patients were identified by each center according to the modified Duke criteria.[12] A dedicated uniform case report form (database) was used at all sites for data collection (191 variables). Informed consent was obtained from all patients before the procedure, and the individual anonymized data sharing was performed according to the local ethics committee of each center. The research was performed without patient or public involvement.

The definition of definite IE was based on the modified Duke criteria.[12] Type of valve involvement was defined as the presence of any of the following findings: vegetation, periannular abscess, fistula or pseudoaneurysm, valvular abscess or aneurysm, or new partial dehiscence the of prosthetic valve on the THV or the MV. Clinical events were defined according to the Valve Academic Research Consortium-2 criteria.[13] Healthcare-associated IE was defined as previously reported.[14] Persistent bacteremia was defined as positive blood cultures despite appropriate antibiotic therapy for >7 days. IE complications with an indication for surgery were defined according to the European guidelines[12] as follows: (1) in-hospital episode of heart failure (HF) attributed to a severe aortic or MV dysfunction, (2) locally uncontrolled perivalvular extension of the infection, (3) aortic or mitral vegetation >10 mm after 1 or more embolic episode.

Continuous variables were expressed as mean ± SD or median (interquartile range) depending on the variable distribution, which was assessed using the Kolmorogov-Smirnov test. Categorical variables were expressed as number (%). Comparisons between groups were performed using the student's *t* test or Wilcoxon rank sum test for continuous

variables and chi-square or Fisher's exact test for categorical variables. A multivariable model was performed to determine the factors associated with MV-IE. Baseline, TAVI-procedure, and IE episode-related variables (excluding IE complications) considered a priori to contribute to MV-IE with a p value <0.20 were included in the multivariable model. A multivariable Cox proportional hazard model was performed to determine the factors independently associated with cumulative time to all-cause mortality in patients with MV-IE. Likewise, this model included all baseline (excluding immediate TAVI complications) and IE episode-related variables considered a priori to contribute to 2-year mortality in this group of patients. Given the limited sample size and to avoid over-fitting, only variables with a p value <0.05 in the univariable analysis were included in the time-to-event multivariable models. Both models were built by backward stepwise (likelihood ratio) selection. Missing data were assumed to be random and were handled by multiple imputations using chained equations. Ten data sets were created, and the results were pooled. All analyses were performed using a hierarchical method to account for between-center variability. The Kaplan-Meier method was used to provide survival estimates, which were assessed with a log-rank test. Event times were measured from the date of initial IE symptoms to the date of death or last follow-up. Differences in the incidence of mortality were determined using the log-rank test. A 2-sided p value of <0.05 was considered statistically significant. Data analyses were performed using the STATA Statistical Software Version 14.0 (StataCorp, College Station, Texas).

## Results

In the 579 patients, IE was located on the THV in 284 patients (49.1%) and on the MV in 86 patients (14.9%). The other patients had either multiple IE locations (177 patients, 30.6%) or right-sided cardiac structure involvement (32 patients, 5.5%). Only patients with THV-IE or MV-IE were included in the analysis (Figure 1).

The MV was the only structure affected in 86 patients. In them, 11 patients (12.8%) had a mechanical or biological prosthesis in the mitral position. The baseline and TAVI characteristics of patients with MV-IE versus THV-IE are shown in Table 1. Patients with MV-IE were more frequently women (44.2% vs 31.7%, p = 0.033), more likely to have had a new pacemaker implantation after TAVI (22.4% vs 10.6%, p = 0.005), and exhibited a higher rate of significant (grade ≥ 2) paravalvular leak (PVL) (25.9% vs 10.3%, p <0.001). There were no differences in the presence of baseline mitral regurgitation ≥2 between groups (MV-IE: 25%, THV-IE: 29%, p = 0.480).

In the multivariable analysis (Table 2), the 2 factors associated with an increased risk of MV-IE were the use of a self-expanding THV (adjusted odds ratio 2.49, 95% confidence interval 1.23 to 5.07, p = 0.012), and the presence of significant PVL after TAVI (adjusted odds ratio 3.33, 95% CI 1.43 to 7.73, p <0.01).

Clinical outcomes after the IE episode according to the type of valve involvement (MV vs THV) are shown in Table 3. The IE causative organisms were similar between groups. Patients with MV-IE had less in-hospital complications compared with THV-IE including a lower rate of septic shock episodes (14.5% vs 27.7%, p = 0.014), persistent bacteriemia (20.3% vs 31.8%, p = 0.050), and perivalvular infection extension (11.6% vs 22.9%, p = 0.023). Surgical management at index IE hospitalization was also substantially lower in patients with MV-IE (6% vs 21.6%, p = 0.001). Up to 48.8% of patients with MV-IE had at least 1 IE complication with an indication for surgery (vs 56.0% in patients with THV-IE, p = 0.244).

In-hospital mortality was higher in patients with THV involvement (30.7%) compared with MV-IE (19.3%), p = 0.042. The Kaplan-Meier survival curves at 2-year follow-up stratified by the valve involved are shown in Figure 2. There were no differences between groups in 2-year mortality rates after IE (MV-IE: 51.4%, 95% CI 39.8 to 64.1; THV-IE: 51.5%, 95% CI 45.4 to 58.0; log-rank p=0.295). The analysis of the factors associated with increased mortality risk in patients with MV-IE is shown in Table 4. In the multivariable analysis, HF (adjusted hazard ratio 3.23, 95% CI 2.10 to 4.97, p <0.001), and septic shock (adjusted hazard ratio 3.71, 95% CI 1.59 to 8.67, p <0.01) at index IE hospitalization were associated with an increased mortality risk.

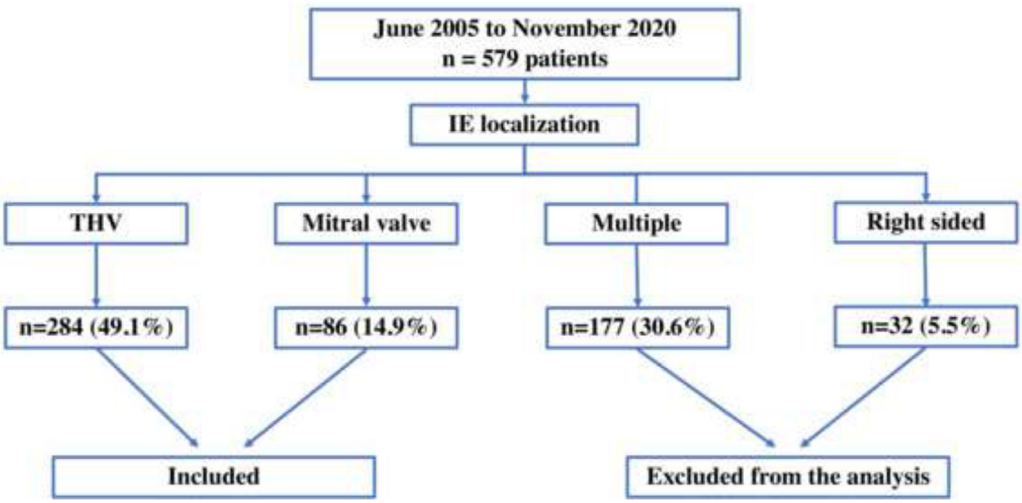


Figure 1. Flowchart of the study.

Table 1
Baseline characteristics, comparison between patients with THV or MV involvement

| Variable | | | Unadjusted | |
|---|---|---|---|---|
| | THV-IE (n=284) | MV-IE (n=86) | *p-Value* | n |
| **Baseline characteristics** | | | | |
| Age (years) ± SD | 78.5 ± 7.7 | 79.7 ± 6.8 | 0.195 | 370 |
| Male, n (%) | 194 (68.3%) | 48 (55.8%) | **0.033** | 370 |
| Body mass index (kg/m$^2$) | 27.9 ± 5.9 | 27.5 ± 4.9 | 0.448 | 368 |
| Diabetes mellitus, n (%) | 98 (34.5%) | 32 (38.4%) | 0.511 | 370 |
| COPD, n (%) | 82 (28.9%) | 25 (29.1%) | 0.972 | 370 |
| Atrial fibrillation, n (%) | 124 (43.7%) | 38 (44.2%) | 0.932 | 370 |
| Chronic kidney disease, n (%) | 127 (45.2%) | 30 (35.7%) | 0.124 | 365 |
| Previous Stroke, n (%) | 38 (13.4%) | 8 (9.3%) | 0.315 | 370 |
| Previous valve surgery, n (%) | 32 (11.3%) | 12 (13.9%) | 0.507 | 370 |
| Previous infectious endocarditis, n (%) | 2 (0.7%) | 2 (2.3%) | 0.233 | 369 |
| Logistic EuroSCORE, % (SD) | 16.7 ± 11.7 | 16.2 ± 11.3 | 0.765 | 329 |
| Left ventricular ejection fraction, % ± SD | 53.2 ± 13.0 | 56.2 ± 11.8 | 0.065 | 363 |
| Mitral regurgitation ≥ 2, n (%) | 81 (29.0%) | 20 (25.0%) | 0.480 | 361 |
| Mean transaortic gradient, mean ± SD, mmHg | 45.1 ± 15.8 | 48.8 ± 15.7 | 0.066 | 348 |
| **Periprocedural characteristics** | | | | |
| Implantation site | | | | |
| Catheterization laboratory, n (%) | 118 (41.6%) | 45 (52.3%) | | 370 |
| Operating hybrid room, n (%) | 20 (7.0%) | 8 (9.3%) | 0.105 | |
| Hybrid room, n (%) | 146 (51.4%) | 33 (38.4%) | | |
| Approach | | | | |
| Transfemoral, n (%) | 238 (84.1%) | 77 (89.5%) | 0.212 | 369 |
| Prosthesis type | | | | |
| Balloon-expanding, n (%) | 162 (57.8%) | 41 (48.2%) | 0.118 | 365 |
| Self-expanding, n (%) | 118 (42.1%) | 44 (51.8%) | | |
| Antibiotic prophylaxis | | | | |
| B-Lactam alone, n (%) | 226 (93.0%) | 73 (92.4%) | | 322 |
| Vancomycin alone or in combination, n (%) | 11 (4.5%) | 6 (7.6%) | 0.246 | |
| Other, n (%) | 6 (2.5%) | 0 (0%) | | |
| **In-hospital Outcomes (TAVR)** | | | | |
| Acute renal failure, n (%) | 30 (10.6%) | 10 (12.4%) | 0.665 | 363 |
| Stroke, n (%) | 10 (3.6%) | 3 (3.7%) | 0.999 | 363 |
| Major vascular complication, n (%) | 16 (5.7%) | 6 (6.1%) | 0.598 | 363 |
| Major bleeding, n (%) | 22 (7.8%) | 6 (7.4%) | 0.907 | 363 |
| Sepsis, n (%) | 25 (9.3%) | 12 (14.8%) | 0.157 | 350 |
| New pacemaker implantation, n (%) | 30 (10.6%) | 19 (22.4%) | **0.005** | 369 |
| Residual aortic regurgitation ≥ 2 at discharge, n (%) | 29 (10.3%) | 22 (25.9%) | **<0.001** | 366 |
| Mean residual transaortic gradient, mean ± SD, mmHg | 11.3 ± 6.8 | 10.1 ± 5.0 | 0.089 | 332 |
| Length of hospital stay, median (IQR), days | 8.0[6.0-14.0] | 9.0[5.0-14.0] | 0.853 | 352 |

Table 2
Univariable and multivariable analysis of risks factors associated with MV involvement

| Variable | Univariable Analysis Odds ratios (95% CI) | Unadjusted *p-Value* | Multivariable Analysis Odds Ratio (95% CI) | Adjusted *p-Value* |
|---|---|---|---|---|
| **Baseline characteristics** | | | | |
| Male | 0.62 [0.36-1.07] | 0.084 | | |
| Chronic kidney disease | 0.68 [0.39-1.21] | 0.189 | | |
| Left ventricular ejection fraction | 1.02 [1.00-1.04] | 0.081 | | |
| Mean transaortic gradient | 1.01 [1.00-1.03] | 0.146 | | |
| **Periprocedural characteristics** | | | | |
| Self-expanding valves | 1.83 [0.98-3.40] | 0.056 | 2.49 [1.23-5.07] | **0.012** |
| **In-hospital Outcomes (TAVI)** | | | | |
| Sepsis | 1.98 [0.87-4.47] | 0.100 | | |
| New pacemaker implantation | 2.27 [1.14-4.54] | 0.020 | | |
| Aortic regurgitation ≥ 2 at discharge | 3.26 [1.58-6.74] | <0.01 | 3.33 [1.43-7.73] | **<0.01** |

Table 3
Main clinical characteristics, management, and outcomes of IE after THV or MV involvement

| Variable | THV-IE (n=284) | MV-IE (n=86) | Unadjusted *p-Value* | n |
|---|---|---|---|---|
| Time from TAVI, median (IQR), days | 191 [73-421] | 175[52-423] | 0.363 | 326 |
| **Initial symptoms** | | | | |
| Fever, n (%) | 212 (76.0%) | 68 (80.0%) | 0.442 | 364 |
| New-onset heart failure, n (%) | 109 (39.4%) | 32 (37.7%) | 0.778 | 362 |
| Neurological, n (%) | 50 (18.1%) | 17 (20.5%) | 0.618 | 360 |
| Systemic embolism, n (%) | 33 (11.9%) | 14 (16.9%) | 0.240 | 360 |
| Skin lesions, n (%) | 12 (4.4%) | 2 (2.4%) | 0.424 | 359 |
| Health care−associated infection, n (%) | 115 (40.5%) | 41 (47.7%) | 0.237 | 370 |
| **Echocardiographic findings, No./total (%)** | | | | |
| Perivalvular extension, n (%) | 65 (22.9%) | 10 (11.6%) | **0.023** | 370 |
| Vegetation size, median (IQR), mm | 10.0 [6.0-14.5] | 11.5 [7.0-17.0] | 0.180 | 230 |
| **Causative microorganisms, No./total (%)** | | | | |
| *Staphylococcus aureus*, n (%) | 56/272 (20.6%) | 20/83 (24.1%) | 0.495 | 355 |
| Coagulase-negative staphylococci, n (%) | 51/272 (18.8%) | 16/83 (19.3%) | 0.914 | 355 |
| Enterococci, n (%) | 75/272 (27.6%) | 19/83 (22.9%) | 0.397 | 355 |
| Streptococci | | | | |
| *S. viridans*, n (%) | 34/272 (12.5%) | 12/83 (14.5%) | 0.642 | 355 |
| *S. gallolyticus (S. bovis),* n (%) | 10/272 (3.7%) | 6/83 (7.2%) | 0.172 | 355 |
| Others, n (%) | 13/272 (4.8%) | 3/83 (3.6%) | 0.999 | 355 |
| Culture negative, n (%) | 23/272 (8.5%) | 4/83 (4.8%) | 0.274 | 355 |
| **Presumed source of entry, n (%)** | | | | |
| Unknown, n (%) | 99 (36.0%) | 30 (36.1%) | N/A | |
| Procedural TAVR related, n (%) | 5 (1.9%) | 2 (3.0%) | | |
| Urological, n (%) | 25 (9.1%) | 7 (8.4%) | | |
| Odontological, n (%) | 9 (3.3%) | 1 (1.2%) | | |
| Pacemaker implantation, n (%) | 4 (1.5%) | 2 (2.4%) | | 358 |
| Skin/soft tissue infection, n (%) | 13 (4.7%) | 1 (1.2%) | | |
| Digestive, n (%) | 28 (10.2%) | 3 (3.6%) | | |
| Cancer, n (%) | 1 (0.4%) | 0 (0%) | | |
| **Complications during IE hospitalization No./total (%)** | | | | |
| Heart failure, n (%) | 116/269 (43.1%) | 30/83 (36.1%) | 0.259 | 352 |
| Acute renal failure, n (%) | 100/265 (37.7%) | 27/70 (38.6%) | 0.898 | 335 |
| Septic shock, n (%) | 74/267 (27.7%) | 12/83 (14.5%) | **0.014** | 350 |
| Stroke, n (%) | 27/269 (10.0%) | 14/83 (16.9%) | 0.090 | 352 |
| Systemic embolization, n (%) | 33/268 (12.3%) | 10/83 (12.1%) | 0.949 | 351 |
| Persistent bacteremia, n (%) | 80/252 (31.8%) | 16/79 (20.3%) | **0.050** | 331 |
| **Management and Outcomes, n (%)** | | | | |
| Antibiotic treatment alone, n (%) | 218 (78.4%) | 78 (94.0%) | **0.001** | |
| Antibiotic + Surgery during IE hospitalization, n (%) | 60/278 (21.6%) | 5/83 (6.0%) | | 361 |
| Time to surgery, median (IQR), days | 20.0 [8-47] | 28 [6.0-380] | 0.824 | 64 |
| Transcatheter valve in valve, n (%) | 3 (2.2%) | 0 (0%) | 0.452 | 160 |
| Isolated pacemaker extraction, n (%) | 0 (0%) | 0 (0%) | N/A | 160 |
| In-hospital mortality, n (%) | 85 (30.7%) | 16 (19.3%) | **0.042** | **370** |
| 30-day mortality rate, (95% CI), % | 20.1 [15.9-25.2] | 11.7 [6.5-20.6] | 0.076* | 370 |
| 1-year mortality rate, (95% CI), % | 46.1 [40.2-52.4] | 35.1 [25.7-46.8] | 0.055* | 370 |
| 2-year mortality rate, (95% CI), %) | 51.5 [45.4-58.0] | 51.4 [39.8-64.1] | 0.295* | 370 |
| Follow-up, median (IQR), months | 6.3 [1.4-26.0] | 12.0 [2.7-24.9] | 0.152 | 370 |

* by log-rank test.

## Discussion

MV involvement was common in the setting of IE after TAVI, and this should be considered when evaluating patients with a suspicion of IE after TAVI. To the best of our knowledge, no data exist regarding this complication after surgical aortic valve replacement (SAVR), suggesting that this is a particular feature of IE after TAVI. Unlike SAVR recipients, a significant proportion of patients with TAVI exhibit significant concomitant MV disease that is not fixed at the time of the intervention,[15] and this may play a role in the discrepancies in MV involvement between patients with IE after TAVI and after SAVR. However, the presence of significant mitral regurgitation was not a factor determining a higher likelihood of MV involvement in our study. Interestingly, 2 factors specific to the TAVI field, such as the use of a self-expanding valve system and the presence of significant PVL, determined an increased risk of MV involvement in patients with IE after TAVI.

Self-expanding valves are deployed from their ventricular side, exerting high radial forces within the left ventricular outflow tract (often deeper than balloon-expanding

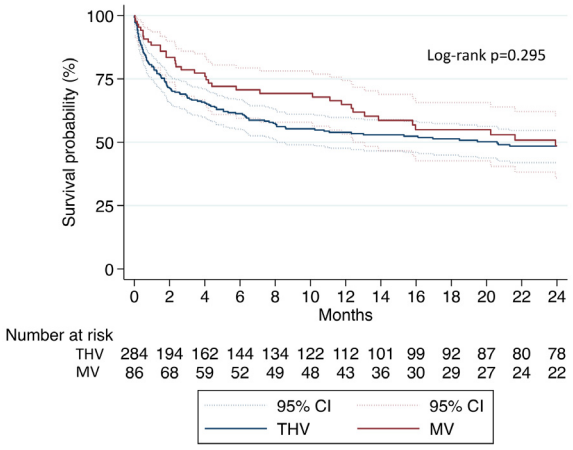


Figure 2. Kaplan-Meier curves comparing survival stratified into THV-IE and MV-IE. Test comparing the 2 groups was based on the log-rank test.

valves, particularly at the beginning of the TAVI experience). Thus, friction between the THV stent frame with the anterior mitral leaflet may occur not only during the TAVI procedure but also over time if the valve is implanted too ventricular. This pathophysiological hypothesis is supported by the high rate of pacemaker permanent implantation after TAVI in our population with MV-IE (22.4%), an indirect marker of implantation depth.[16] In addition to the well-known association between a lower THV implantation and a higher risk of conduction disturbances[17] and PVL,[18] our data suggest that a too ventricular positioning of the THV may increase the risk of MV involvement in the context of IE after TAVI.

The presence of PVL was also associated with an increased risk of MV-IE. Historical studies with previous generation THV systems showed a high rate of significant PVL after TAVI.[19] Although the introduction of newer generation THV systems, along with improvements in valve sizing and the increasing experience of the centers/operators have translated into a major reduction in the rate and severity of PVL after TAVI,[20] its incidence remains higher than that observed after SAVR.[21] Endocardial damage of the MV because of "jet lesion" has already been described in non-TAVI recipients as a rare phenomenon.[22,23] These lesions occur at the point where the jet stream hits the heart structure (leaflets, papillary muscle, or chordae tendinae). The turbulence between the THV and the native valve can also promote platelet aggregation and thrombus formation serving as a nidus to the infection more prone to produce the matrix of vegetation in an adhesive platelet-fibrine environment.[24]

The clinical characteristics of MV-IE were similar to those of THV-IE, with a median time between TAVI and the index IE episode of about 6 months. Moreover, the enterococci and *Staphylococcus aureus* were the most frequent microorganisms involved. However, patients with MV-IE exhibited a lower incidence of complications, including septic shock, persistent bacteremia, and perivalvular infection extension. Previous studies in patients with non-TAVI IE also showed a higher rate of perivalvular infection extension but a lower incidence of embolic events in aortic patients with IE compared with their mitral IE counterparts.[25,26] Despite the lower incidence of IE complications, up to 48.8% of patients with MV-IE exhibited at least 1 complication that would have required surgical management. However, surgical intervention in patients with MV-IE was very low (6%), much lower than that reported in patients with non-TAVI MV-IE (49%),[27] and also lower than patients with THV-IE. Although surgical management was not associated with improved outcomes, the low number of patients who had surgery precluded an appropriate analysis of this factor.

IE after TAVI has been associated with very high early and late mortality rates.[2] In accordance with these results, patients with MV-IE also had poor outcomes, and up to one-third and half of the patients died at index hospitalization and at 2-year follow-up, respectively. As previously described in patients with MV-IE, HF along with septic shock were found to be independent predictive factors of death.[25–27] HF after IE is a well-described prognostic factor[28] and is the most common indication for surgery in left-sided IE.[29] Gelsomino et al[30] compared the outcomes of 379 patients with no shock, cardiogenic shock, and septic shock after non-TAVI MV-IE. Patients with septic shock exhibited a fourfold higher risk of complications and had a higher risk of postoperative complications and death after surgery. Thus, surgery of the MV seems associated with prohibitive risk in this subpopulation with an unfavorable prognosis.

Our study has some limitations. First, because of its retrospective observational design, some data were not available. For instance, we did not gather information about the implantation depth of each THV or other anatomic data

Table 4
Univariable and multivariable analysis of parameters associated with mortality in patients with mitral involvement

| Variable | Univariable Analysis Hazard Ratio (95% CI) | Unadjusted *p-Value* | Multivariable Analysis Hazard Ratio (95% CI) | Adjusted *p-Value* |
|---|---|---|---|---|
| **In-hospital complication during IE** | | | | |
| Heart failure | 3.92 [2.47-6.23] | <0.001 | 3.23 [2.10-4.97] | <0.001 |
| Acute renal failure | 2.91 [1.31-6.44] | <0.01 | | |
| Septic Shock | 4.45 [1.59-12.43] | <0.01 | 3.71 [1.59-8.67] | <0.01 |
| Systemic embolization | 2.70 [1.11-6.56] | 0.029 | | |
| Persistent bacteremia | 3.10 [1.74-5.51] | <0.001 | | |
| **Management** | | | | |
| Surgery at index IE hospitalization* | 0.79 [0.29-2.13] | 0.639 | | |

* Not included in the multivariable analysis.

such as mitral leaflet-specific involvement. Centers participated voluntarily, and there was no external monitoring committee to verify the accuracy of data reported by each center. Finally, because of its multicenter design, diagnosis and treatment modalities of patients may have been different between participating centers.

The MV was affected in 1 of 6 patients diagnosed with IE after TAVI, and this should be considered when evaluating patients with TAVI with suspicion of IE. MV involvement was not determined by the presence of significant mitral regurgitation before TAVI but by the presence of 2 TAVI-specific factors such as implantation of self-expanding THVs and significant residual PVL after the procedure. Patients with MV-IE after TAVI were rarely managed surgically and had a dismal prognosis, particularly those presenting with HF and/or septic shock at index hospitalization. Future studies should further evaluate the mechanisms associated with MV-IE after TAVI and determine the most appropriate therapeutic approach in such patients.

## Disclosures

Josep Rodés-Cabau has received institutional research grants from Edwards Lifesciences, Medtronic, and Boston Scientific. Vassili Panagides has received institutional research grants from Medtronic, Boston Scientific, and Microport. Didier Tchetche has reported consulting fees from Abbott Vascular, Boston Scientific, Edwards Lifesciences, and Medtronic. Howard C. Herrmann has received institutional research grants from Abbott, Boston Scientific, Edwards Lifesciences, and Medtronic and consulting fees from Edwards Lifesciences and Medtronic. John G. Webb has reported that he has received consulting fees from Edwards Lifesciences and St. Jude Medical. Raj Makkar has reported that he has received research grants from Edwards Lifesciences, Medtronic, Abbott, Capricor Therapeutics, and St. Jude Medical; has served as a proctor for Edwards Lifesciences; and has received consulting fees from Medtronic. Fabio Sandoli de Brito Jr. has reported that he has received honoraria from Medtronic and Edwards Lifesciences for symposium speeches and proctoring cases. Stamatios Lerakis has reported that he has received consulting fees from Edwards Lifesciences. Hervé Le Breton reports lecture fees from Edwards Lifesciences, outside the submitted work. Jan Malte Sinning reports speaker honoraria from Abbott, Boston Scientific, Edwards Lifesciences, and Medtronic and research grants from Boston Scientific, Edwards Lifesciences, and Medtronic, outside the submitted work. Kim Won-Keun reports personal fees from Boston Scientific, Edwards Lifesciences, Abbott, Medtronic, and Meril, outside the submitted work. Stefan Stortecky reports grants to the institution from Edwards Lifesciences, Medtronic, Boston Scientific, and Abbott and personal fees from Boston Scientific, BTG, and Teleflex, outside the submitted work. Oliver Husser reports personal fees from Boston Scientific and payments from Abbott. Norman Mangner reports personal fees from Edwards Lifesciences, Medtronic, Biotronik, Novartis, Sanofi Genzyme, AstraZeneca, Pfizer, and Bayer, outside the submitted work. The other authors have no conflicts of interest to declare.

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