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ORIGINAL ARTICLE
Valvular Heart Disease

Clinical Impact of Preoperative Symptoms of Aortic Stenosis on Prognosis After Transcatheter Aortic Valve Replacement

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Background: The prognostic significance of different presentations of aortic stenosis (AS) remains unclear. Our aim was to analyze outcomes after transcatheter aortic valve replacement (TAVR) according to preoperative AS symptoms.

Methods and Results: We retrospectively enrolled 369 consecutive patients (age 84.3±5.0 years, and 64% females) who underwent TAVR from 2014 to 2021. We divided them into 4 groups by the main preoperative symptom: asymptomatic (n=50), chest pain (n=46), heart failure (HF; n=240), and syncope (n=33). Post-TAVR rates of HF readmission, all-cause death and cardiac death were compared among the 4 groups. The 4 groups showed no significant trends in age, sex, stroke volume index, or echocardiography indices of AS severity. During a follow-up, the overall survival rate at 1 and 5 years after TAVR was 97% and 90% in the asymptomatic group, 96% and 69% in the chest pain group, 93% and 69% in the HF group, and 90% and 72% in the syncope group, respectively. HF and syncope symptom had significantly lower HF readmission or cardiac death-free survival at 5 years after TAVR (log-rank test P=0.038). In the Cox hazard multivariate analysis, preoperative syncope was an independent predictor of future HF readmission or cardiac death after TAVR (HR=9.87; 95% CI 1.67–97.2; P=0.035).

Conclusions: AS patients with preoperative syncope or HF had worse outcomes after TAVR than those with angina or no symptoms.

Key Words: Aortic stenosis; Heart failure; Syncope; Transcatheter aortic valve replacement/implantation

Aortic stenosis (AS) is one of the most common valvular heart diseases, and it has been getting more important in the aging society. The most common manifestations of AS are chest pain, shortness of breath suggesting heart failure (HF), and syncope.[1] Symptomatic patients with severe AS are known to have a poor prognosis, and previous studies have demonstrated the natural prognosis by each symptom.[2] Surgical aortic valve replacement (SAVR) and transcatheter aortic valve replacement (TAVR) are well established treatments for severe AS patients and are known to improve mortality.[3–6] While several papers have analyzed outcomes after SAVR by preoperative symptoms,[7,8] the relationship between AS symptoms and cardiac outcomes after TAVR remains unclear. Our aim was to reveal the impact of preoperative symptoms on prognosis after TAVR in severe AS patients.

Methods

Study Population and Data Collection

We retrospectively enrolled 369 consecutive patients with native severe aortic valve stenosis who underwent TAVR at Gunma Prefectural Cardiovascular Center from 2014 to 2021. Clinical data, including age, sex, body surface area, blood pressure (BP), heart rate (HR), past history, medication, laboratory data, and echocardiography parameters were retrieved from electronic medical records. In addition, we checked the patients' preoperative symptoms and divided them into 4 groups by the main symptom: asymptomatic, chest pain (Canadian Cardiovascular Society class ≥I), HF (New York Heart Association [NYHA] functional class ≥II), and syncope (or presyncope; **Figure 1**). When symptoms overlapped, the primary physicians who reviewed the medical records made a judgment on which was dominant. The reasons why asymptomatic patients

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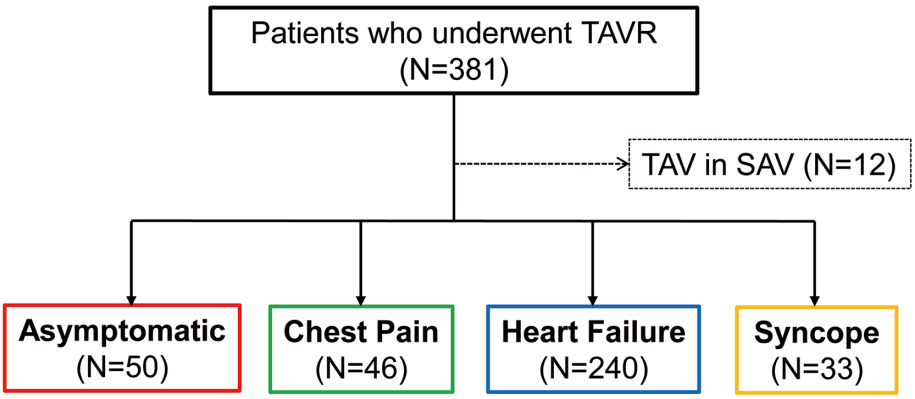

Figure 1. Study flowchart. TAVR, transcatheter aortic valve replacement; TAV in SAV, transcatheter aortic (valve in surgical) aortic valve.

underwent TAVR were LV dysfunction (LV ejection fraction [EF] <50%), very severe AS, or rapid progression according to the Japanese Circulation Society.[9] Follow-up information was obtained regularly via outpatient clinics. Patients, physicians, and next of kin were contacted by telephone if the patients were treated at a different hospital.

Echocardiography Data

Comprehensive transthoracic echocardiography was performed using commercially available ultrasound equipment according to the American Society of Echocardiography (ASE) guideline.[10] Peak early and late diastolic velocity of LV inflow (E and A velocity, respectively), deceleration time of E velocity, and peak early diastolic velocity on the septal corner of the mitral annulus (e′) were measured in the apical 4-chamber view. AVA was calculated by the continuity equation using aortic valve jet velocity and SV in the LV outflow tract (LVOT).[11,12] Aortic valve jet velocity was recorded from multiple acoustic windows, and the highest velocity signal was selected. SV was determined by the velocity–time integral by pulsed wave Doppler echocardiography at LVOT×LV outflow area, which was determined with the following formula: $3.14\times(\text{LVOT diameter}/2)^2$. Follow-up transthoracic echocardiography was performed in all but 4 patients within approximately 1 week after TAVR (mean postoperative days: 5.3±1.5).

TAVR Procedure

All patients underwent TAVR using a balloon-expandable valve (Edwards Sapien XT/Sapien 3 Transcatheter Heart Valve; Edwards Lifesciences, Irvine, CA, USA) or self-expandable valve (Medtronic CoreValve/Evolut R Revalving System; Medtronic, Minneapolis, MN, USA). The indications and procedure plan for TAVR were determined based on the clinical consensus of a multidisciplinary team that included interventional cardiologists, cardiac surgeons, imaging specialists and anesthesiologists.

Study Endpoints

The primary study endpoint was all-cause death. Two secondary endpoints, the occurrence of cardiac death including periprocedural death and hospitalization for HF, were also evaluated.

Statistical Analysis

Continuous data were expressed as means±SD, and categorical data were expressed as counts and percentage. Data were compared between four groups using the Student's t-test or chi-square test, as appropriate. Probabilities of event-free survival were obtained using Kaplan-Meier analysis among the four groups and compared using the log-rank test. The impact of potential prognostic factors on event-free survival rate was assessed using Cox proportional-hazard models. Variables with P values <0.15 in univariate analysis were incorporated into the multivariable model. Variables showing collinearity were carefully considered before inclusion in the multivariate model. The group classification was entered into the model, and patients in the asymptomatic group were considered as the reference. Differences with P values <0.05 were considered statistically significant. All statistical analyses were performed using commercial statistical software (JMP version 8; SAS Institute, Cary, NC, USA). This study was approved by the institutional review board of Gunma Prefectural Cardiovascular Center (approval no. 2023001).

Results

Among the 369 enrolled patients, the mean age was 84.3±5.0 years, and 64% were female (**Table 1**). Among the 4 groups, no significant differences were found in age, sex, body surface area, or history of ischemic heart disease or stroke. In the HF group, lower BP values, greater prevalence of atrial fibrillation (AF), higher B-type natriuretic peptide (BNP) levels, and more frequent use of β-blockers and diuretics were found than in the other 3 groups. Asymptomatic patients had significantly lower clinical frailty scores and less prevalent use of anticoagulant drugs than other symptomatic groups.

Table 2 shows the preoperative and postoperative echocardiography data and details of TAVR. LVEF was significantly lower in the HF group than in the other 3 groups. Patients in the HF group had larger LV end-diastolic volume, LV end-systolic volume, and LA volume index than patients in the asymptomatic group and the chest pain group. There were no significant differences in SV index or echocardiography indices of AS severity among the 4

Table 1. Characteristics of Enrolled Patients and Comparisons Among 4 Groups Categorised by Main Preoperative Symptom: Asymptomatic, Chest Pain, HF, and Syncope

	Overall (N=369)	Asymptomatic (N=50)	Chest pain (N=46)	HF (N=240)	Syncope (N=33)	P value
Age (years)	84.3±5.0	82.7±6.3	84.0±5.3	84.5±4.7	85.3±4.6	0.070
Female sex (%)	63.7	64.0	63.0	63.3	66.7	0.99
BSA (m^2)	1.44±0.17	1.45±0.18	1.45±0.15	1.44±0.17	1.44±0.14	0.93
BMI (kg/m^2)	22.1±3.5	22.0±2.9	22.8±4.1	22.1±3.6	22.2±2.7	0.57
Heart rate (beat/min)	74.3±14.2	72.4±10.5	74.5±11.5	75.1±15.7	71.0±10.6	0.31
Systolic BP (mmHg)	130.9±23.2	138.7±19.0	138.1±25.3	127.3±23.0	134.8±22.0	<0.001
Diastolic BP (mmHg)	71.9±13.4	74.0±10.5	75.8±13.0	70.5±13.9	73.3±12.9	0.04
Hypertension (%)	78.9	84.0	76.1	77.5	84.9	0.56
Dyslipidemia (%)	46.9	54.0	54.4	44.2	45.5	0.43
Diabetes (%)	24.7	28.0	32.6	23.8	15.2	0.30
History of smoking (%)	22.5	22.0	26.1	22.5	18.2	0.87
Prior MI or CABG (%)	14.5	8.0	21.7	15.0	15.2	0.30
Prior PCI (%)	30.0	24.0	37.0	30.7	24.2	0.47
CKD (%)	67.8	50.0	67.4	72.5	60.6	0.018
Atrial fibrillation (%)	25.7	8.0	10.9	32.5	24.2	<0.001
Cerebrovascular event (%)	11.9	14.0	10.9	10.8	18.2	0.66
Malignancy (%)	10.8	16.0	6.5	10.4	12.1	0.50
Chronic lung disease (%)	15.4	8.0	15.2	17.1	15.2	0.39
Prior PMI (%)	5.4	4.0	4.4	6.3	3.0	0.88
Clinical frailty score	4.2±1.2	3.7±1.2	4.3±1.3	4.3±1.1	4.4±1.1	0.003
NYHA classification (%)						<0.001
I	21.1	100.0	34.8	0.0	36.4	
II	44.2	0.0	43.5	52.9	48.5	
III	28.2	0.0	21.7	37.5	12.1	
IV	6.5	0.0	0.0	9.6	3.0	
Hemoglobin (mg/L)	11.7±1.6	12.2±1.6	11.8±1.4	11.6±1.6	11.6±1.8	0.14
BNP (pg/mL)	434±782	163±110	287±115	531±51	340±139	0.009
eGFR ($mL/min/1.73\,m^2$)	51.0±18.0	56.4±17.5	52.4±18.2	49.2±18.3	53.4±15.1	0.053
ARB or ACEI (%)	55.3	52.0	52.2	56.7	54.6	0.90
β-blocker (%)	30.1	20.0	21.7	35.8	15.2	0.008
CCB (%)	48.8	54.0	56.5	47.5	39.4	0.39
Diuretics (%)	52.0	18.0	34.8	63.8	42.4	<0.001
Stain (%)	43.4	44.0	52.2	40.8	48.5	0.49
Anticoagulation (%)						0.001
DOAC	19.2	2.0	10.9	24.6	18.2	
Warfarin	6.2	6.0	4.4	6.7	6.1	
None	74.5	92.0	84.8	68.8	75.8	
Antiplatelet (%)						0.30
DAPT	21.1	22.0	30.4	19.6	18.2	
SAPT	25.7	18.0	32.6	25.8	27.3	
None	53.1	60.0	37.0	54.6	54.6	

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; BMI, body mass index; BNP, B-type natriuretic peptide; BP, blood pressure; BSA, body surface area; CABG, coronary artery bypass grafting; CCB, calcium channel blocker; CKD, chronic kidney disease; DOAC, direct oral anticoagulant; DAPT, dual antiplatelet therapy; eGFR, estimated glomerular filtration rate; HF, heart failure; MI, myocardial infraction; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; PMI, pacemaker implantation; SAPT, single antiplatelet therapy.

groups. Overall, the majority (95%) of TAVR procedures were performed using the transfemoral approach, and balloon-expandable valves were selected in 73%. Regarding procedural complications, cardiac rupture or tamponade occurred in 4 patients. Atrioventricular block requiring new pacemaker implantation after TAVR was found in 21 (5.7%) cases, and 8 (2.2%) cases experienced a stroke after TAVR. No significant differences among the 4 groups were shown in the perioperative complications of TAVR. Overall, mild aortic regurgitation (AR) after TAVR, which means paravalvular leakage or transvalvular leakage of the implanted valve, was shown in 18.7%, and moderate AR was shown in 0.3%. Postoperatively, the mean pressure gradient of the aortic valve was 9.9±5.8 mmHg, with no significant differences among the 4 groups.

Median follow-up duration was 1.5 years (interquartile

Table 2. Preoperative Echocardiography Data and Details of TAVR in Enrolled Patients

	Overall (N=369)	Asymptomatic (N=50)	Chest pain (N=46)	HF (N=240)	Syncope (N=33)	P value
LVEDV (mL)	77.7±32.1	68.5±21.2	70.1±22.1	81.1±34.6	76.9±35.6	0.024
LVESV (mL)	30.6±25.4	18.9±11.0	24.3±16.2	34.6±27.5	28.7±28.8	<0.001
LVEF (%)	64.2±15.9	72.9±11.1	67.5±14.4	61.3±16.4	67.0±14.7	<0.001
SVI (mL/m^2)	32.5±10.2	33.8±9.2	31.5±9.4	32.3±10.6	33.6±9.9	0.64
LAVI (mL/m^2)	59.6±26.9	50.9±18.2	49.5±20.0	63.5±29.0	58.9±25.2	<0.001
E/A	0.91±0.66	0.75±0.42	0.84±0.46	0.99±0.78	0.79±0.31	0.074
E/e'	20.7±10.0	18.1±7.7	20.2±10.6	21.8±10.5	18.3±6.9	0.055
LVMI (g/m^2)	131.0±35.1	125.1±29.3	127.1±37.4	134.3±36.4	121.1±27.0	0.078
Peak aortic jet velocity (m/s)	4.3±0.8	4.5±0.6	4.2±0.8	4.2±0.8	4.4±0.8	0.19
Mean PG (mmHg)	46.7±17.8	49.3±12.8	45.2±19.7	46.1±18.1	48.3±19.7	0.59
AVA (cm^2)	0.67±0.21	0.71±0.20	0.68±0.19	0.65±0.20	0.67±0.26	0.21
AVA index (cm^2/m^2)	0.47±0.14	0.50±0.13	0.47±0.14	0.46±0.14	0.46±0.18	0.33
AR degree (%)						0.14
No or trivial	53.4	54.0	63.0	49.6	66.7	
Mild	39.0	40.0	37.0	30.3	30.3	
≥Moderate	7.6	6.0	0.0	3.4	3.0	
MR degree (%)						0.001
No or trivial	68.6	90.0	76.1	62.5	69.7	
Mild	21.4	10.0	21.7	23.3	24.2	
≥Moderate	10.0	0.0	2.2	14.1	6.1	
TR degree (%)						0.001
No or trivial	70.5	90.0	76.1	62.5	69.7	
Mild	24.5	10.0	21.7	23.3	24.2	
≥Moderate	4.9	0.0	2.2	14.1	6.1	
TAVR procedure						
Transfemoral approach (%)	94.9	100.0	93.5	94.6	90.9	0.25
Balloon-expandable valve (%)	72.6	80.0	76.1	70.4	72.7	0.80
Perioperative complication						
Rupture/tamponade	1.1	0.0	2.2	1.3	0.0	0.52
New PMI	5.7	8.0	4.4	4.2	15.2	0.13
Stroke	2.2	2.0	0.0	2.9	0.0	0.26
Postoperative AR (%)						0.92
No or trivial	81.0	82.0	76.1	82.1	78.8	
Mild	18.7	18.0	23.9	17.5	21.2	
≥Moderate	0.3	0.0	0.0	0.4	0.0	
Postoperative mean PG (mmHg)	9.9±5.8	10.3±4.2	9.5±4.1	9.9±6.5	9.7±3.9	0.92

AR, aortic regurgitation; AVA, aortic valve area; BAV, balloon aortic valvuloplasty; HF, heart failure; LAVI, left atrial volume index; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; LVMI, left ventricular mass index; SVI, stroke volume index; MR, mitral regurgitation; PG, pressure gradient; PMI, pacemaker implantation; TAVR, transcatheter aortic valve replacement; TR, tricuspid regurgitation.

range: 1.0–2.0 years). During follow-up, 31 HF readmissions, 46 all-cause deaths, and 17 cardiac deaths (7 sudden cardiac deaths, 5 deaths due to perioperative complications, 4 to HF and 1 to myocardial infarction) occurred. **Figure 2** shows the Kaplan-Meier analysis of all-cause death-free, cardiac death-free, HF readmission-free, and HF readmission or cardiac death-free survival among the 4 groups. There were similar 1- and 5-year all-cause death-free survival rates among the 4 groups (97±3% and 90±5% for the asymptomatic group, 96±3% and 69±15% for chest pain group, 93±2% and 69±6% for the HF group, and 90±5% and 72±11% for the syncope group, respectively; log-rank test P=0.25). The cardiac death-free survival rates at 1 and 5 years were also similar among the 4 groups (100±0% and 97±3% for the asymptomatic group, 98±2% and 98±2% for the chest pain group, 96±1% and 89±4% for the HF group, and 97±3% and 81±11% for the syncope group, respectively; log-rank test P=0.33). There were no significant differences in the 1- and 5-year HF readmission-free survival rates (98±2% and 98±2% for the asymptomatic group, 95±3% and 95±3% for the chest pain group, 95±2% and 74±7% for the HF group, and 85±7% and 85±7% for the syncope group, respectively; log-rank test P=0.12). Patients in the HF group and the syncope group had significantly lower HF readmission or cardiac death-free survival at their 1- and 5-year follow-ups (92±2% and 69±7% for the HF group, and 82±7% and 67±12% for the syncope group vs. 98±2% and 94±4% for the asymptomatic group, and 93±4% and 93±4% in the chest pain group; log rank test P=0.038).

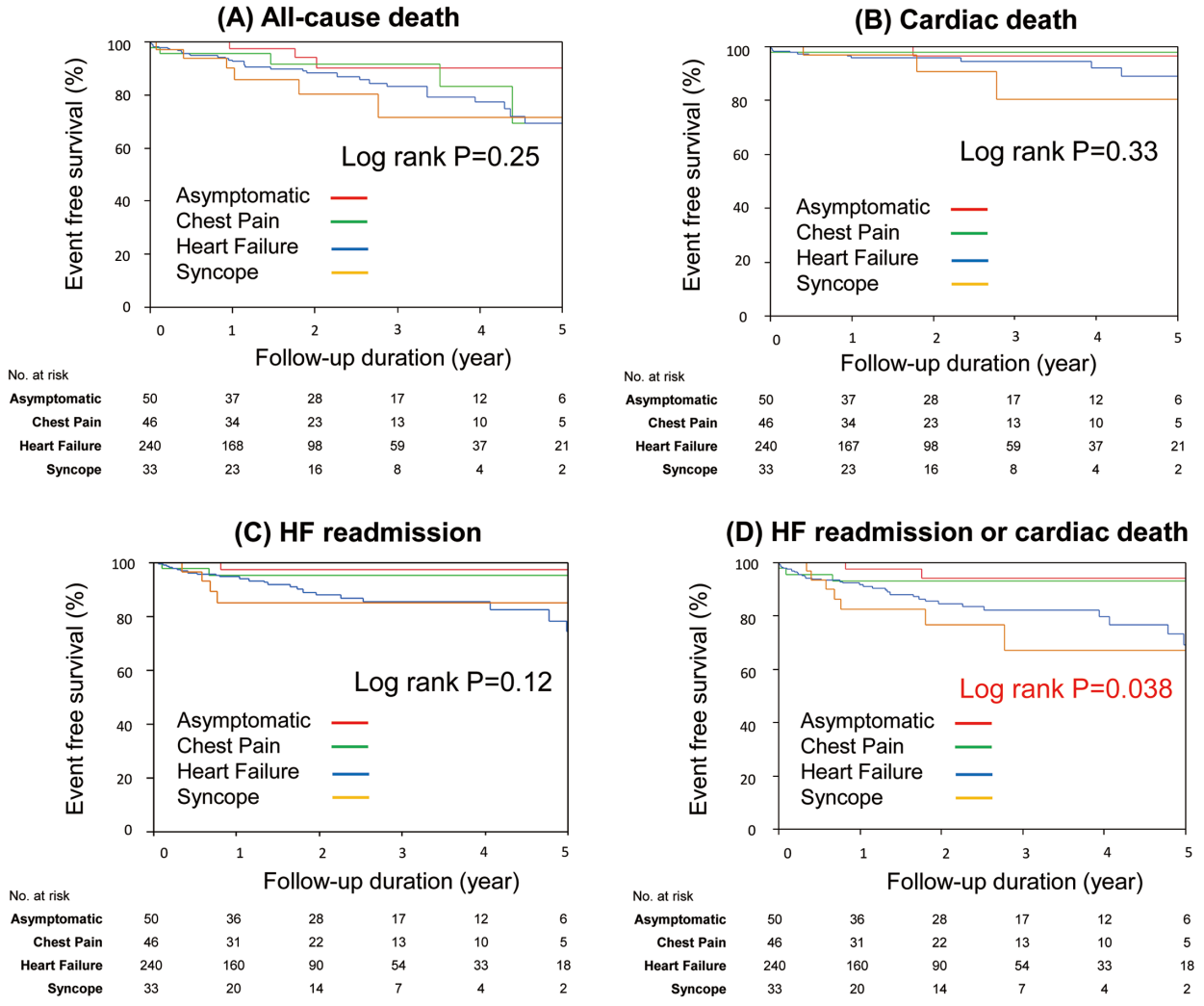

Figure 2. Kaplan-Meier analysis of (**A**) all cause death-free survival, (**B**) cardiac death-free survival, (**C**) heart failure (HF) readmission-free survival, and (**D**) HF readmission or cardiac death-free survival after transcatheter aortic valve replacement among 4 groups categorised by the main preoperative symptom: asymptomatic, chest pain, HF, and syncope.

As shown in **Table 3**, the Cox-Hazard univariate analysis of the secondary endpoint demonstrated that AF, BNP level, LVEF <40%, LA volume index, HF, and syncope were predictors of future HF readmission or cardiac death after TAVR. The Cox-Hazard multivariate analysis showed that AF, LA volume index, and syncope were independent predictors. The hazard ratio of syncope was 9.867 (95% CI 1.67–97.2; P=0.035), when asymptomatic status was set as the reference.

Discussion

This was the first observational study to find that preoperative syncope in AS patients is an independent significant predictor of cardiac events after TAVR. However, Goliasch et al showed similar results in patients undergoing SAVR.[7] In that study, preoperative syncope in severe AS patients was an independent determinant for long-term mortality after SAVR, whereas preoperative HF (NYHA ≥II) or chest pain were not significantly associated with prognosis. They suggested that worse outcomes in patients experiencing syncope might be due to pathophysiological characteristics such as a smaller valve area, smaller cardiac cavities, and lower SV compared with patients without syncope. Additionally, another paper reported that AS patients with syncope were likely to have smaller LV size and reduced SV than patients with other clinical presentations.[13] However, in the present study, there were no significant differences in LV volume, SV index, and AVA between patients in the syncope group and patients in the other 3 groups. Therefore, another pathophysiology might be related to poor outcomes in syncopal patients with AS.

Mechanism of Syncope in AS Patients

Syncope occurs from BP depression resulting in cerebral hypoperfusion, and syncopal episodes in AS are usually observed during exercise. While several papers have reported that syncope with AS is associated with arryth-

Table 3. Univariate and Multivariate Analysis of HF Readmission or Cardiac Death in Enrolled Patients

	Univariate analysis			Multivariate analysis		
	HR	95% CI	P value	HR	95% CI	P value
Age (years)	0.997	0.942–1.060	0.93	0.970	0.905–1.045	0.42
Female	1.281	0.695–2.484	0.44	1.312	0.498–3.426	0.58
BSA (m^2)	0.259	0.040–1.541	0.14	0.291	0.017–4.385	0.38
Heart rate (beat/min)	1.003	0.980–1.025	0.78			
Systolic BP (mmHg)	0.991	0.977–1.004	0.16			
CKD	1.613	0.844–3.340	0.15	1.074	0.505–2.445	0.85
Atrial fibrillation	3.939	2.180–7.135	<0.001	3.428	1.565–7.550	0.002
Coronary artery disease	0.868	0.459–1.583	0.65			
Chronic lung disease	1.165	0.476–2.451	0.72			
Anemia	1.230	0.684–2.229	0.49			
Clinical frailty score	1.095	0.837–1.428	0.51			
BNP (μg/mL)	1.323	1.017–1.567	0.040	1.213	0.836–1.569	0.26
β-blocker use	1.607	0.862–2.909	0.13	1.055	0.499–2.144	0.89
ARB or ACEi use	0.989	0.549–1.814	0.97			
LVEF <40%	2.518	1.022–5.353	0.045	0.922	0.292–2.655	0.88
LVEDV (mL)	1.006	0.998–1.014	0.15	1.010	0.998–1.022	0.11
LVESV (mL)	1.007	0.996–1.016	0.18			
SVI (mL/m^2)	1.019	0.990–1.046	0.20			
LAVI (mL/m^2)	1.017	1.009–1.023	<0.001	1.011	1.000–1.021	0.048
Preoperative mean PG (mmHg)	0.999	0.982–1.016	0.91			
AVA index (cm^2/m^2)	1.312	0.168–9.174	0.79			
Balloon expandable valve	1.258	0.632–2.787	0.53			
Postoperative AR ≥mild	1.166	0.503–2.380	0.69			
Postoperative mean PG (mmHg)	0.936	0.864–1.004	0.067	0.953	0.869–1.032	0.26
New PMI	0.667	0.108–2.155	0.55			
Asymptomatic	Ref.			Ref.		
Chest pain	1.714	0.284–13.025	0.55	1.070	0.045–22.498	0.50
Heart failure	4.090	1.242–25.234	0.017	1.947	0.526–12.626	0.73
Syncope	6.384	1.537–42.935	0.010	9.867	1.674–97.180	0.035

CI, confidence interval; HR, hazard ratio. Other abbreviations as in Tables 1,2.

mia,[14,15] Richards et al used continuous BP monitoring during exercise and found sudden BP depression without arrythmia in all presyncopal episodes.[16] The current assumption regarding syncope in AS is that it is caused by an imbalance of cardiac output (CO) and peripheral resistance during exercise.[16–18] In general, exercise increases sympathetic nerve activity and decreases parasympathetic nerve activity, which leads to an increase in CO and a decrease in total peripheral resistance due to vasodilation in the skeletal muscles.[19–21] In particular, AS patients with syncope cannot increase CO to compensate for exercise-induced decreases in peripheral resistance, probably due to reduced CO by the stenotic aortic valve and/or sympathetic nerve dysfunction.

However, the reasons why patients who have AS with preoperative syncope are at high risk of cardiac death or HF hospitalization even after TAVR remain unclear. Reduced exercise capacity and sympathetic nerve dysfunction associated with AS with syncope are possible mechanisms. Obviously, reduced exercise capacity has been known as a strong predicter for mortality or HF hospitalization,[22–24] and several papers reported that the frailty in patients who underwent TAVR independently predicted poor outcomes.[25–27] Exercise capacity has been associated with multiple factors, such as SV, HR, peripheral vascular resistance, autonomic balance, and skeletal muscles.[28] Regarding the autonomic nerve system, a previous study indicated that cardiac sympathetic nerve dysfunction assessed using MIBG imaging was associated with major adverse cardiovascular events after TAVR.[29,30] Therefore, prolonged cardiac sympathetic nerve dysfunction even after TAVR might be partly responsible for reduced exercise capacity and poor outcomes.

Study Limitations

The present study has several limitations. First, this was a single-center and non-randomized retrospective study, which could have produced a selection bias. Moreover, the number of syncope patients might be too small to assess the clinical outcomes. Thus, prospective multicenter studies are required to validate our results. Second, the symptomatic status assessed in this study may not have been accurate due to the lack of standardization in history taking and the absence of exercise tests to confirm symptomatic status. Moreover, the classification of 4 symptoms might be ambiguous and contain information bias because some patients had overlapping symptoms. Third, 2 of 33 patients with preoperative syncope had recurrent syncope even after TAVR, both of which were due to unknown cause. Fourth, we had no data about medication after

TAVR, which is thought to be important for the development of HF and improvement of prognosis. Last, the syncope status of patients was determined by reviewing medical records alone. Therefore, we could not obtain further details to clarify the cause of syncope in these patients or rigorously eliminate transient arrhythmia or myocardial ischemia attack as causes.

Conclusions

We demonstrated that preoperative syncope in severe AS patients undergoing TAVR was associated with future cardiac death or HF readmission; to a lesser extent, the same was true for preoperative HF. We should carefully follow patients who have undergone TAVR, especially those presenting with syncopal episodes, because various factors other than AS may contribute to difficulty maintaining hemodynamics, even after TAVR.

Sources of Funding

The authors declare that they have no potential conflicts of interest.

IRB Information

The present study was approved by the institutional review board of Gunma Prefectural Cardiovascular Center (reference number: 2023001).

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