Heart and Vessels (2019) 34:1031–1039
https://doi.org/10.1007/s00380-018-01329-2

ORIGINAL ARTICLE

Association between valvuloarterial impedance after transcatheter aortic valve implantation and 2-year mortality in elderly patients with severe symptomatic aortic stenosis: the OCEAN-TAVI registry

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Received: 26 September 2018 / Accepted: 21 December 2018 / Published online: 1 January 2019

Abstract

Pre-procedural valvuloarterial impedance (Zva) is considered as a useful predictor of mortality in patients diagnosed as having severe aortic stenosis (AS) who undergo transcatheter aortic valve implantation (TAVI). However, the prognostic significance of post-procedural Zva remains unclear. We aimed to evaluate the prognostic significance of Zva after TAVI. We retrospectively analyzed the clinical and echocardiographic data of 1004 consecutive elderly patients (median 84 years old, 27.5% men) who underwent TAVI for severe symptomatic AS. Zva was calculated after TAVI, and patients were divided into three groups based on tertile values: the high [> 3.33 ($n = 335$)], intermediate [2.49–3.33 ($n = 334$)], and low Zva groups [< 2.49 ($n = 335$)]. The estimated 2-year all-cause and cardiovascular mortalities using Kaplan–Meier analysis were 16.2% [95% confidence interval (CI) 11.8–20.4] and 5.9% (95% CI 3.2–8.6), respectively. There were no significant intergroup differences in each endpoint (long-rank $p = 0.518$ for all-cause mortality, $p = 0.757$ for cardiovascular mortality). Multivariable Cox regression analyzes with adjustments of patient characteristics and medications showed that the post-procedural Zva was not associated with the 2-year all-cause mortality [intermediate Zva group versus (vs.) low Zva group: adjusted hazard ratio (aHR) = 1.34, 95% CI 0.75–2.40, $p = 0.316$; high Zva group vs. low Zva group: aHR = 1.17, 95% CI 0.64–2.16, $p = 0.613$] and cardiovascular mortality (intermediate Zva group vs. low Zva group: aHR = 1.50, 95% CI 0.56–4.06, $p = 0.421$; high Zva group vs. low Zva group: aHR = 1.25, 95% CI 0.43–3.65, $p = 0.682$). Our results suggest that post-procedural Zva was not associated with 2-year all-cause or cardiovascular mortalities in patients with severe symptomatic AS who underwent TAVI.

Keywords Aortic stenosis · Stroke Volume Index · Transcatheter aortic valve implantation · Valvuloarterial impedance

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Introduction

Valvuloarterial impedance (Zva) is a useful parameter that represents the global left ventricular (LV) hemodynamic load, which is the summation of the valvular and vascular loads [1, 2]. Pre-procedural baseline Zva has been reported to be an independent predictor of overall mortality in 544 patients with asymptomatic aortic stenosis (AS) regardless of the treatments [3], or in 116 patients or in 102 patients with severe aortic stenosis who underwent transcatheter aortic valve implantation (TAVI) [4, 5] since Zva changes dramatically between before and after TAVI or surgical aortic valve replacement in patients with severe AS due to a significant release from valvular pressure load [4, 6] There are few studies considering post-procedural Zva. One single center study enrolling 198 patients with a Zva that either remained unchanged or increased post-TAVR had significantly higher mortality at 1-year post-procedure [6]. It is also of interest for physicians to evaluate whether post-procedural Zva is associated with long-term prognoses after TAVI for a long-term out-patient management or not. We aimed to assess the association between Zva after TAVI and the 2-year all-cause and cardiovascular mortalities by analyzing data from 1004 consecutive severe AS patients who enrolled in a multicenter prospective observational registry of TAVI.

Materials and methods

Study population

Study participants are severe AS patients who enrolled in the Optimized CathEter vAlvular iNtervention (OCEAN)-TAVI registry, which is a prospective, multicenter, observational registry of patients with symptomatic heart failure (New York Heart Association class II or greater), those with a high risk of heart failure, and inoperable patients with degenerative severe AS, as defined according to the guidelines, undergoing TAVI. Thirteen high-volume institutions in Japan contributed to this registry. Data were collected from October 2013 to July 2016. Patient selection flow is presented in Fig. 1. Among the 1613 patients included in the OCEAN-TAVI registry, 416 patients were excluded because of in-hospital death ($n = 50$), a second valve or conversion to open heart surgery ($n = 30$), chronic and paroxysmal atrial fibrillation ($n = 319$), moderate or severe aortic regurgitation ($n = 17$), and unavailable Zva data ($n = 193$). Finally, 1004 patients were included for final analysis in the study. Chronic and paroxysmal atrial fibrillation and severe aortic regurgitation were excluded because they affected echo-parameters such as stroke volume (SV) and pressure gradient and made it difficult to measure precisely. All participating centers had an established training and certifying program. The institutional review boards of all participating centers approved the study protocol that was developed in accordance with the Declaration of Helsinki, and all patients gave informed written consent for participating in the study. This trial was registered with the University Hospital Medical Information Network (UMIN) (registry number UMIN000020423).

Study endpoints

We set the primary endpoint as 2-year all-cause mortality after hospital discharge and the secondary endpoint as 2-year cardiovascular mortality. The last update of the clinical events was obtained from the participating institutes in July 2016.

Echocardiographic measurement and calculation of Zva

All participants underwent a post-procedural echocardiographic evaluation after TAVI during the hospitalization. The conventional parameters were measured according to the American Society of Echocardiography guidelines [7, 8]. Systemic arterial pressure was measured with the use of an arm cuff sphygmomanometer around the echocardiography examination. Zva was obtained using the following formula: Zva = [systolic arterial pressure (SAP): SAP + transaortic mean gradient (MG)]/ Stroke Volume Index (SVI). SVI was calculated as follows: stroke volume (SV)/body surface area (BSA). SV was calculated as the product of the LV outflow tract area and velocity–time integral on pulsed-wave Doppler echocardiography.

Statistical analysis

Continuous variables were presented as median and interquartile range (IQR). Categorical variables are described as numbers and percentages. Based on the tertile value of post-procedural Zva, we divided study patients into three groups: high Zva (Zva $\geq$ 3.33, $n = 335$), intermediate Zva (2.49 < Zva < 3.33, $n = 334$), and low Zva groups (Zva $\leq$ 2.49, $n = 335$). Differences of continuous variables among the groups were tested by the Kruskal–Wallis test. Kaplan–Meier analyzes were performed for estimating event rates and the log-rank test was used to compare survival rates among the groups. Cox proportional hazard regression models were used to determine the associations between the clinical variables on 2-year all-cause mortality and cardiovascular mortality. Adjusted covariates included

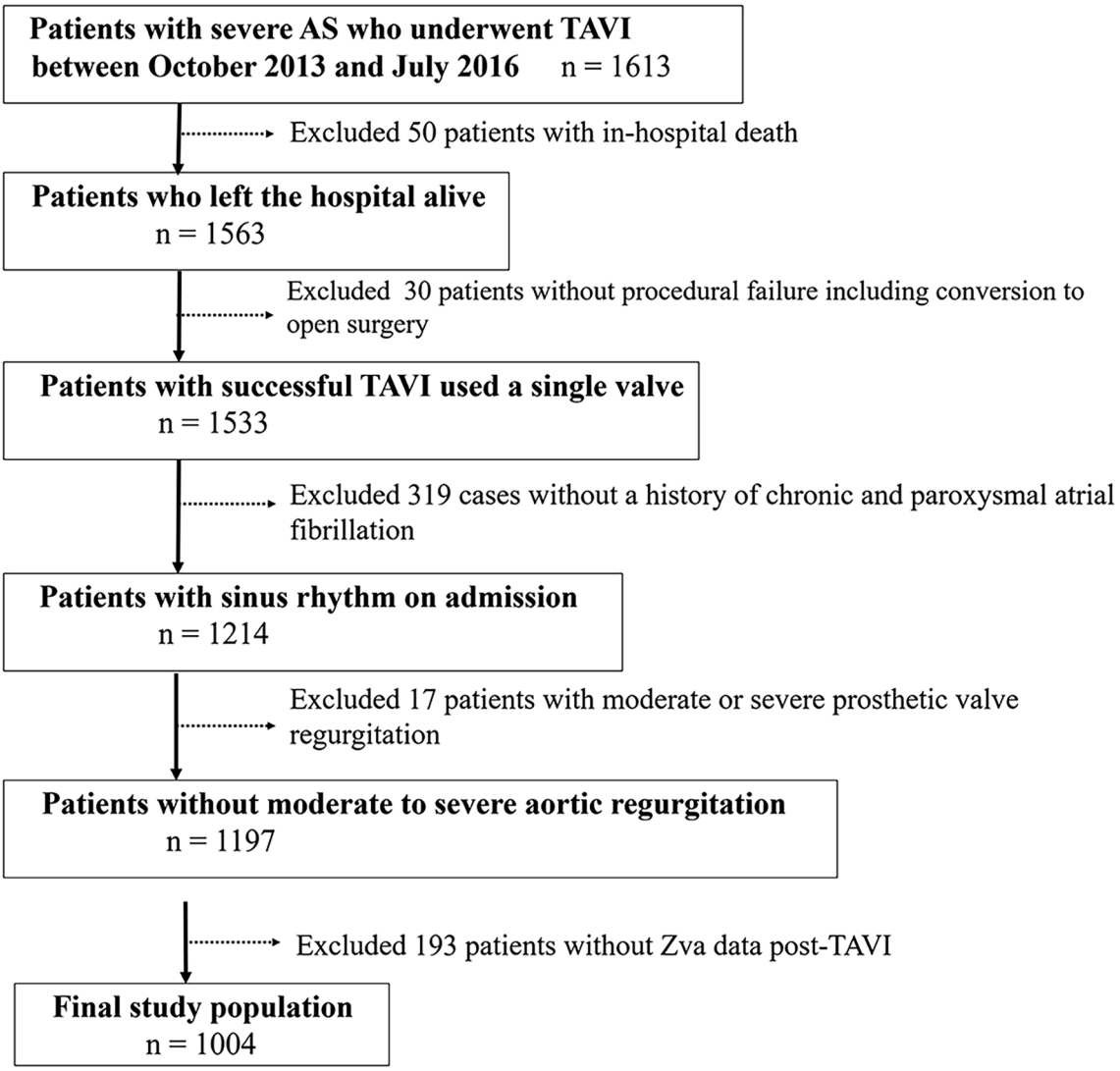

Fig. 1 Patient selection flow chart. AS, aortic stenosis; TAVI, transcatheter aortic valve implantation. *Zva* valvuloarterial impedance

oral administration of an angiotensin-converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB), oral administration of a beta-blocker, LV ejection fraction (LVEF), and Society of Thoracic Surgeons' (STS) surgical mortality risk score. These were selected clinically considering previous reports and multicollinearity. All statistical analyzes were performed using R software package (version 3.1.1; R Development Core Team). A p value < 0.05 was considered statistically significant.

Results

Figure 2 shows the distribution of Zva. The median level of Zva after TAVI was 2.87 (IQR 2.34–3.58). Table 1 shows baseline characteristics of the study patients.

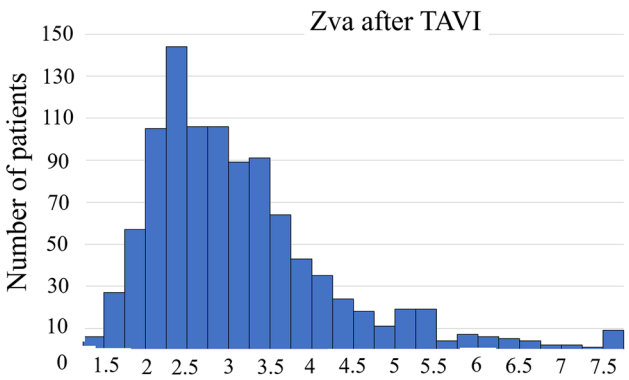

Fig. 2 Distribution of Zva after TAVI. The median level of Zva after TAVI is 2.87 (interquartile range 2.34–3.58). *TAVI* transcatheter aortic valve implantation, *Zva* valvuloarterial impedance

Table 1 Patient demographic characteristics

Parameter	Total patients $n = 1004$	Low Zva group $n = 335$	Intermediate Zva group $n = 334$	High Zva group $n = 335$	p value
Zva after TAVI	4.10 (3.19–4.83)	2.14 (1.98–2.34)	2.89 (2.70–3.08)	4.38 (3.58–4.94)	< 0.001
Age (years)	84 (81–88)	84 (81–87)	84 (81–88)	85 (81–88)	0.207
Male sex	276 (27.5)	75 (22.4)	108 (32.3)	93 (26.2)	0.016
BSA (m^2)	1.42 (1.30–1.53)	1.39 (1.27–1.50)	1.44 (1.32–1.54)	1.42 (1.30–1.54)	< 0.001
Atherosclerotic risks					
Hypertension	791 (78.8)	252 (75.2)	267 (80.0)	272 (81.2)	0.138
Dyslipidemia	436 (43.4)	138 (41.2)	151 (45.2)	147 (43.9)	0.566
Diabetes mellitus	262 (26.1)	67 (20.0)	98 (29.3)	97 (29.0)	0.008
Current smoker	26 (2.6)	5 (1.5)	8 (2.4)	13 (3.9)	0.145
Chronic kidney disease	580 (57.8)	167 (50.0)	188 (56.3)	225 (67.2)	< 0.001
Previous ischemic stroke	133 (13.2)	41 (12.2)	57 (17.1)	35 (10.4)	0.033
STS surgical mortality risk score	7.55 (4.57–9.00)	6.91 (4.28–8.24)	7.39 (4.61–8.96)	8.37 (4.72–9.80)	0.003
Treatment					
Valve type					0.462
Edwards SAPIEN XT	829 (82.5)	281 (83.9)	269 (80.5)	278 (83.0)	
Edwards SAPIEN 3	93 (9.3)	26 (7.8)	31 (9.3)	36 (10.7)	
Medtronic core valve	83 (8.3)	28 (8.4)	34 (10.2)	21 (6.3)	
Valve size (mm)	23 (23–26)	23 (23–26)	23 (23–26)	23 (23–26)	0.576
Medication					
ACEI or ARB	550 (54.8)	189 (56.4)	179 (53.6)	183 (54.3)	0.748
Beta-blocker	309 (30.8)	81 (24.2)	105 (31.4)	123 (36.7)	0.002
Calcium blocker	442 (44.0)	150 (44.8)	141 (42.2)	151 (45.1)	0.716
Diuretic	493 (49.1)	156 (46.6)	176 (52.7)	161 (48.1)	0.256
Statin	439 (43.7)	144 (43.0)	152 (45.5)	143 (42.7)	0.722

Categorical variables are shown as numbers (percentages), and continuous variables are shown as medians (25–75th percentiles)

ACEI angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *BSA* body surface area, *STS* Society of Thoracic Surgeons', *TAVI* transcatheter aortic valve implantation, *Zva* valvuloarterial impedance

Median age was 84 (IQR 80–88) and 27.5% were men. Several patient characteristics were different among groups. For example, patients with intermediate Zva were more likely to be males and have past medical history of ischemic stroke, patients with low Zva had a significantly smaller BSA and a lower prevalence of diabetes mellitus, and patients with high Zva had a higher prevalence of chronic kidney disease, STS score, and the most oral administration of βblocker than the other 2 groups. Table 2 shows the post-procedural echocardiographic data and blood pressure (BP) data. Patients with higher Zva had a smaller effective orifice area, higher systolic and diastolic arterial pressures, and smaller SV. There was no significant difference in transvalvular gradient. And patients with higher Zva also had a higher prevalence of MR and TR which grade were 3 or 4 though their overall prevalence were only 5.3% and 4.5%, respectively.

Clinical outcomes

Overall, 69 patients died post-TAVI during the 2-year follow-up. Among them, 23 deaths were due to a cardiovascular cause. Forty-six deaths were caused by non-cardiac conditions such as pneumonia ($n = 10$), infections including septic shock but excluding pneumonia ($n = 9$), some types of cancer ($n = 8$), and traumatic cerebral hemorrhage ($n = 5$).

Figure 3 demonstrates the Kaplan–Meier survival analysis for all-cause mortality and cardiovascular mortality. The estimated 2-year all-cause and cardiovascular mortalities were 16.2% [95% confidence interval (CI) 11.8–20.4] and 5.9% (95% CI 3.2–8.6), respectively (Fig. 3a, c). There were no significant intergroup differences in each endpoint (long-rank $p = 0.518$ for all-cause mortality; $p = 0.757$ for cardiovascular mortality; Fig. 3b, d).

Table 2 Post-procedural data

Parameter	Total patients $n=1004$	Low Zva group $n=335$	Intermediate Zva group $n=334$	High Zva group $n=335$	p value
TTE data after TAVI					
iEOA	1.16 (1.0–1.36)	1.37 (1.19–1.53)	1.16 (0.98–1.30)	1.06 (0.88–1.21)	< 0.001
Mean AVP gradient (mmHg)	10 (8–13)	11 (8–13)	10 (8–12)	10 (7–13)	0.154
SVI (ml)	47 (39–57)	60 (55–66)	48 (43–52)	35 (29–40)	< 0.001
LVEF (%)	61 (55–68)	62 (55–70)	60 (54–68)	61 (56–68)	0.114
E/e'	21.5 (15.3–26.2)	22.5 (16.4–26.7)	21.3 (15.2–26.3)	20.8 (14.7–25.5)	0.176
LV diameter	41 (37–45)	41 (37–45)	42 (38–46)	41 (37–45)	0.356
MR grades 3, 4	53 (5.3)	13 (3.9)	15 (4.5)	25 (7.5)	< 0.001
TR grades 3, 4	45 (4.8)	14 (4.6)	9 (2.8)	22 (6.7)	0.013
Peak TVP gradient (mmHg)	31 (25–36)	31 (24–36)	31 (24–37)	32 (25–38)	0.152
Systolic blood pressure (mmHg)	127 (115–137)	118 (108–128)	127 (118–136)	134 (122–144)	<0.001
Diastolic blood pressure (mmHg)	61 (54–69)	59 (51–66)	60 (52–67)	65 (58–71)	< 0.001

Categorical variables are shown as numbers (percentages), and continuous variables are shown as medians (25–75th percentiles)

*AVP*aortic valve pressure, *LVEF*left ventricular ejection fraction by modified Simpson method, *E/e'*peak early diastolic left ventricular filling velocity/early diastolic mitral annular velocity, *iEOA* indexed effective orifice area, *LV* left ventricular, *MR* mitral regurgitation, *SVI* Stroke Volume Index, *TAVI* transcatheter aortic valve implantation, *TTE* transthoracic echocardiography, *TR* tricuspid regurgitation, *TVP* tricuspid valve pressure, *Zva* valvuloarterial impedance

Table 3 shows results from univariable and multivariable Cox regression analyzes. Multivariate Cox regression analyzes after adjusting for baseline characteristics revealed that post-procedural Zva was not associated with the 2-year all-cause mortality [intermediate Zva group versus (vs.) low Zva group: adjusted hazard ratio (aHR) = 1.34, 95% CI 0.75–2.40, $p = 0.316$; high Zva group vs. low Zva group: aHR = 1.17, 95% CI 0.64–2.16, $p = 0.613$] nor cardiovascular mortality (intermediate Zva group vs. low Zva group: aHR = 1.50, 95% CI 0.56–4.06, $p = 0.421$; high Zva group vs. low Zva group: aHR = 0.25, 95% CI 0.43–3.65, $p = 0.682$). These results were confirmed by analyzing the association of Zva value itself as continuous explanatory variable with setting prognoses as independent variable.

Discussion

In the present study enrolling the largest study population ever ($n = 1004$), Zva after TAVI did not have a significant association with the 2-year all-cause and cardiovascular mortalities in patients with severe AS.

Zva represents the sum of valvular and vascular load. In Zva's formula, transvalvular gradient represents valvular load, systolic atrial pressure represents vascular load, and these are indexed by SVI [1]. Zva significantly decreased after TAVI [6, 9, 10]. After aortic valve implantation, transaortic pressure gradients reduced immediately, left ventricular ejection fraction increased which leads stroke volume to increase [5]. Previous studies have reported the value of Zva in patients with AS. Pre-procedural Zva has been shown to be an independent prognostic marker in patients with asymptomatic AS with preserved LVEF [3] and in those who underwent TAVI [4, 5]. In contrast, other studies have reported that Zva does not improve risk stratification in low LVEF, low-gradient AS [11, 12], and in those after surgical aortic valve replacement [13]. All of these studies discussed pre-procedural Zva (baseline Zva), not Zva after treatment. One study discussed the change in Zva post-TAVI and suggested that Zva either remained unchanged or increased post-TAVI, and had a significantly higher mortality at 1 year post-procedure [6]. Here, we focused on post-procedural Zva, but unexpectedly, post-procedural Zva was not associated with patient prognosis.

After successful TAVI, the valvular load caused by the stenotic valve is released, SVI increases because typically LVEF improves, which leads to an increase SV, and then Zva decreases [5]. However, most previous studies evaluated prognosis using preoperative Zva and that may be because the most reflective point of the advanced state of disease was considered the time of the measurement of preoperative Zva. After invasive treatment, Zva usually improves, but its immediate improvement may not directly indicate the effectiveness of the treatment. Additionally, in this study, we evaluated only a one-time value of postoperative Zva in the hospital. The amount of myocardial damage is affected by the length of the load to which the myocardium is exposed, and it does not recovery immediately, as shown in some studies [14, 15]. In these studies, which were based on cardiac magnetic resonance imaging, it was reported that significant left ventricular reverse remodeling occurred 6 months after TAVI [14]; in a later study, LV hypertrophy

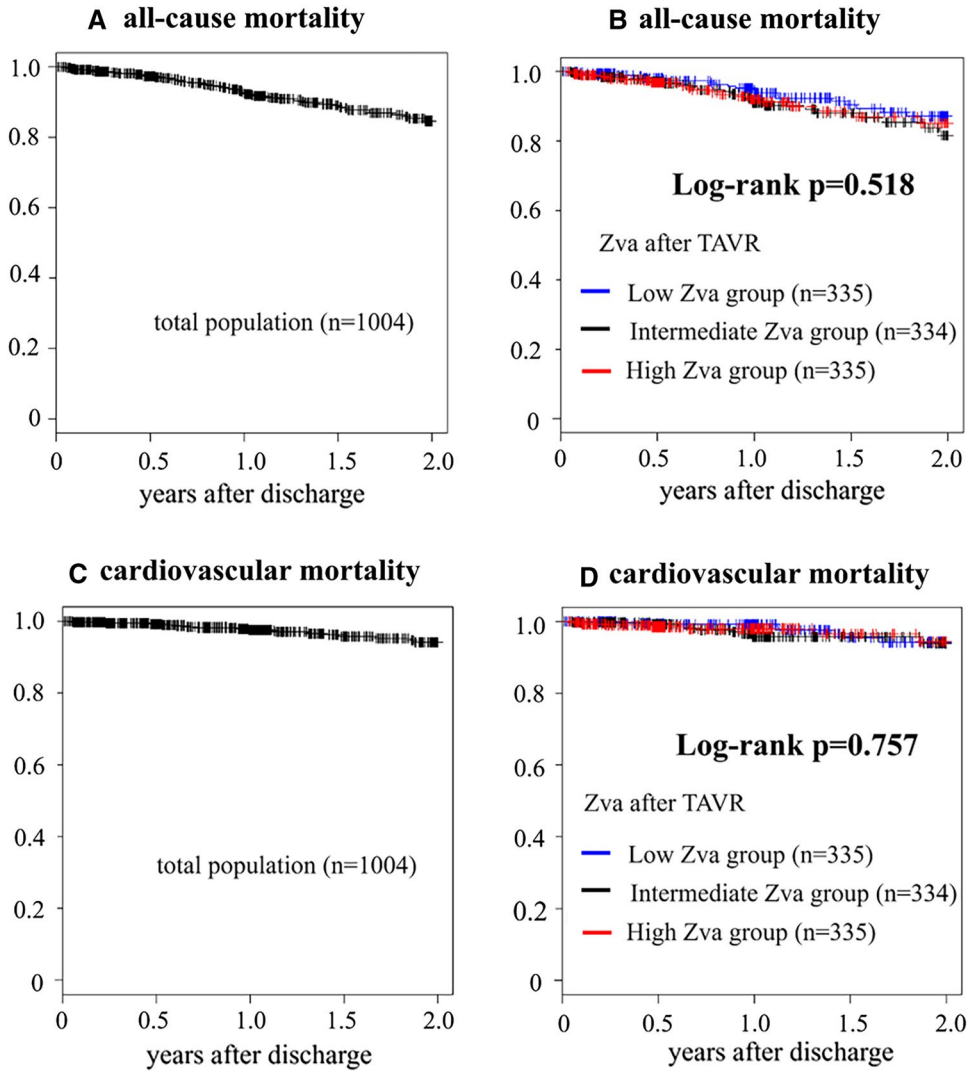

Fig. 3 Kaplan–Meier estimates of each endpoint. The estimated 2-year all-cause and cardiovascular mortalities using Kaplan–Meier analysis are 16.2% [95% confidence interval (CI) 11.8–20.4] and 5.9% (95% CI 3.2–8.6), respectively (**a** all-cause mortality; **c** cardiovascular mortality). There are no significant intergroup differences in each endpoint (long-rank $p = 0.518$ for all-cause mortality; **b**, $p = 0.757$ for cardiovascular mortality; **d** cardiovascular mortality). *TAVI* transcatheter aortic valve implantation, *Zva* valvuloarterial impedance

Table 3 Effect of Zva

Study endpoints	Univariable analysis		Multivariable analysis	
	HR (95% CI)	*p* value	Adjusted HR (95% CI)	*p* value
All-cause mortality				
Zva				
Intermediate group vs. low group	1.39 (0.78–2.47)	0.266	1.34 (0.75–2.40)	0.316
High group vs. low group	1.28 (0.70–2.33)	0.418	1.17 (0.64–2.16)	0.613
Per 1 increase	1.20 (0.99–1.46)	0.065	1.16 (0.95–1.42)	0.153
CV mortality				
Zva				
Intermediate group vs. low group	1.45 (0.54–3.91)	0.457	1.50 (0.56–4.06)	0.421
High group vs. low group	1.25 (0.44–3.56)	0.682	1.25 (0.43–3.65)	0.682
Per 1 increase	1.30 (0.95–1.78)	0.107	1.30 (0.94–1.79)	0.109

In the multivariable model, the STS score, administration of an ACEI or ARB on admission, administration of a beta-blocker on admission, and LVEF were included as covariates for CV mortality and total mortality

ACE angiotensin-converting enzyme, *ARB* angiotensin receptor blocker, *CV* cardiovascular, *HR* hazard ratio, *LVEF* left ventricular ejection fraction, *STS* Society of Thoracic Surgeons', *Zva* valvuloarterial impedance

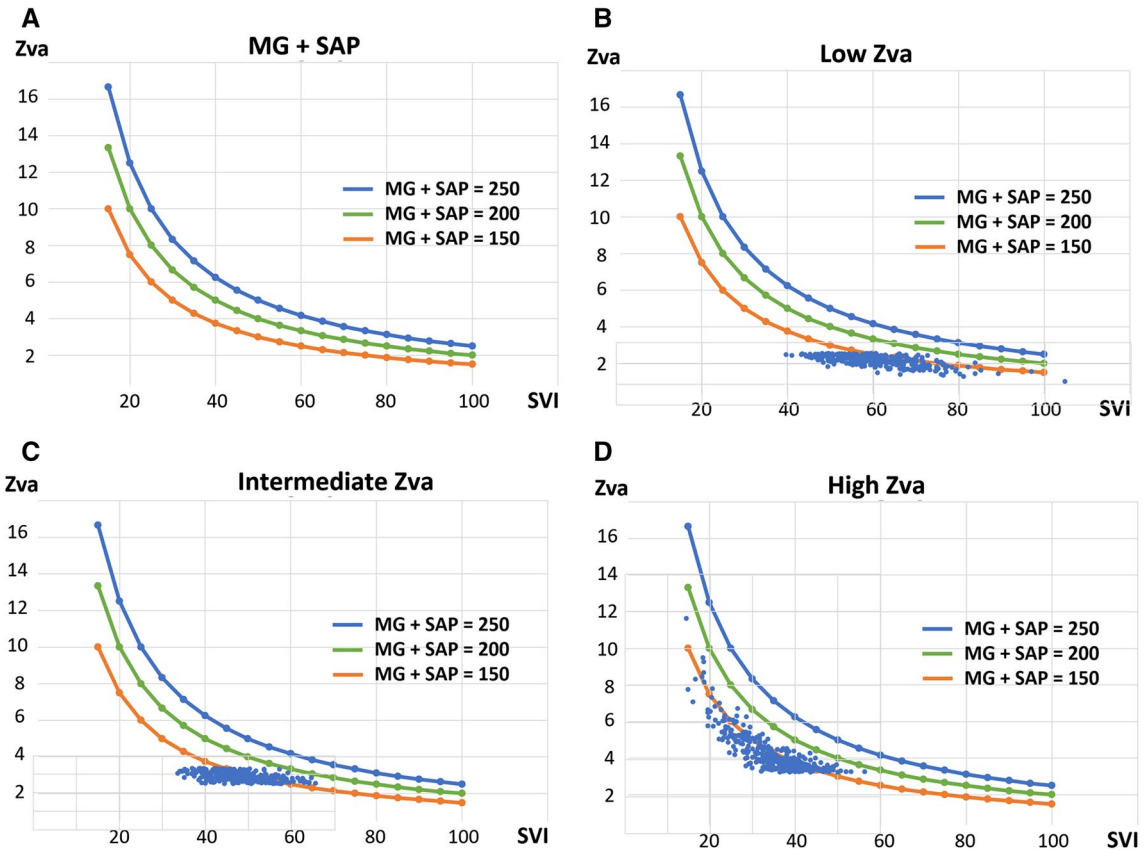

Fig. 4 Association between Zva and SVI in three patterns of MG+SAP. The distribution of Zva (**a** MG + SAP; **b** low Zva; **c** intermediate Zva; and **d** high Zva) is added in the graphs of the association between Zva and SVI in three patterns of MG + SAP. *MG* transaortic mean gradient, *SAP* systolic arterial pressure, *SVI* Stroke Volume Index, *Zva* valvuloarterial impedance

regression occurred, but diffuse fibrosis was not reversible 6 months after AVR [15]. Zva immediately after TAVI does not include the length of recovery of myocardial damage in severe AS. Therefore, it may be important to assess the value of Zva after reversible remodeling.

Zva may not be an accurate parameter because subtle changes in SVI may cause significant variations in Zva. Figure 4a shows the association between Zva and SVI with three different patterns of MG+SAP. The figure suggests that the smaller SVI is, the higher Zva is likely to become. SVI is the only denominator so it has the strongest effect among the three parameters: SAP, MG, and SVI. Figure 4b–d shows the distribution of Zva along with the association between Zva and SVI from Fig. 4a. The numerator is MG + SAP, and no significant difference in MG was found in this study. Patients with a higher Zva had a higher SAP. However, Fig. 4 shows that SAP had no strong effect on Zva in the three groups. In these figures, it is easily to see that the high Zva group tended to have low SVI. In other words, the impact of minor error during echocardiography in the measurement of the SV on the calculation of Zva may lead to a large error. The calculation of SVI is slightly complex. The SV represents the product of the velocity–time integral and the cross-sectional area. It is easily affected by the heart rate; thus, it is recommended that data from 3 to 5 cardiac cycles are averaged, and the cross-sectional area is calculated using the annulus diameter, assuming that the aortic annulus is a circle, although it is not perfectly circular [16]. Therefore, it is possible that single-center studies [2, 3, 5, 6] in specialized facilities for echocardiography have more accurate values of Zva in contrast to multicenter studies like ours, in which 13 centers participated. Furthermore, the systolic BP as well as the patient's physical and mental conditions change by the time Zva is measured. Although Zva is a non-invasive and easy-to-use marker, it is difficult to calculate accurately and interpret it correctly.

SVI is closely related to prosthesis patient mismatch (PPM), which is a well-known strong predictor of short-term mortality [17]. PPM was defined by indexed EOA (iEOA); iEOA, which is EOA indexed by BSA, was determined using the following formula derived from the equation of continuity: left ventricular out flow tract (LVOT) × velocity time integral (VTI) at LVOT / VTI at transcatheter heart valve (THV) × BSA. Therefore, iEOA contains SVI

in the formula, which is equal to SVI/VTITHV. Therefore, it is easy to infer that Zva has a prognostic value. PPM was defined as moderate if iEOA was > 0.65 and ≤ 0.85 cm^2/m^2 and severe if it was ≤ 0.65 cm^2/m^2, and patients with severe PPM had a poor prognosis.

In this study, the median iEOA was 1.06 (IQR 0.88–1.21) even in the high Zva group. We hardly had any patients with severe PPM in this study. Therefore, a smaller EOA had an effect on hemodynamics but was not attributed to prognosis in our population. In a previous study of a group of Japanese patients who had undergone TAVI in our TAVI registry, we already reported a low prevalence of PPM and observed that it was not a risk factor of mid-term mortality in Ref. [18].

Study limitations

Several limitations of this study should be acknowledged. First, we only assessed change of Zva in the short term. The follow-up period after TAVI was only 2 years. Second, this was a retrospective analysis of prospectively collected data from the OCEAN-TAVI registry. Third, data collected were measured in individual OCEAN registry institutions using different methods rather than at a single core laboratory. Especially, the technical inaccuracy and facility disparity of the Doppler echocardiographic measurement for SVI and the mean gradient cannot be ruled out.

Conclusion

Post-procedural Zva after TAVI was not associated with 2-year all-cause and cardiovascular mortalities in patients with severe symptomatic AS.

Acknowledgements The authors thank all the investigators and institutions for collecting the data, and thank the Japan Society of Clinical Research for their dedicated support for study completion.

Funding None.

Compliance with ethical standards

Conflict of interest Drs. Watanabe, Shirai, Tada, Araki, Naganuma, Yamamoto, and Hayashida are proctors for transfemoral-TAVI, and Dr. Tabata is a proctor of transapical TAVI for Edwards Lifesciences. Drs. Watanabe, Shirai, Naganuma, and Yamamoto are proctors of transfemoral TAVI for Medtronic. The other authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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