Pulmonary artery systolic pressure at 1-month predicts 1-year survival after transcatheter aortic valve implantation

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ABSTRACT

Background: Pulmonary hypertension related to left ventricle heart disease is a common finding in patients with severe aortic stenosis treated with transcatheter aortic valve implantation (TAVI) and is associated with a higher mortality rate.

Aims: The study aimed to analyze the influence of pulmonary artery systolic pressure (PASP) changes after TAVI on long-term survival.

Methods: TAVI was performed in 362 patients between January 2013 and December 2018. The study group comprised 210 patients who underwent a detailed 1-month follow-up.

Results: At 1-month, 142 had a stable or decreased PASP value (Group 1), while in 68 patients an increase was observed (Group 2). During 1-year follow-up, 20 patients died (9.5%), 9 in Group 1 and 11 in Group 2 ($P = 0.02$). The receiver operating characteristic (ROC) curve analysis (area under the curve [AUC], 0.750) revealed a significant value of 1-month measurement for 1-year mortality prediction. The cutoff for the PASP value predictive of mortality was ≤41 mm Hg. A Kaplan-Meier analysis showed significantly higher mortality in patients without a 1-month PASP decrease. In the multivariable analysis, PASP measured at 1-month after TAVI (hazard ratio, 1.040; 95% confidence interval, 1.019–1.062; P <0.001) was an independent predictor of 1-year mortality. Each 1 mm Hg increase in PASP predicts a 4% increase in the risk of death.

Conclusion: Decreased or stable value of PASP at 1-month follow-up may predict better 1-year survival after TAVI, while each 1 mm Hg increase in PASP confers a 4% greater risk of 1-year mortality.

Key words: transcatheter, aortic stenosis, pulmonary artery systolic pressure, mortality

INTRODUCTION

Transcatheter aortic valve implantation (TAVI) is currently a standard of care in a selected group of patients [1–4]. Commonly used risk score calculators [5], including the EuroSCORE II and Society of Thoracic Surgeons (STS) score, have been designed for peri-operative risk assessment in cardiac surgery but have been shown to underperform in the TAVI population. Impaired left ventricular ejection fraction [6], acute kidney injury [7], anemia [8], pulmonary hypertension (PH) [9–11], peri-procedural complications, including the need for blood transfusion [12, 13], and several other factors have established influence on the mortality rate. Several attempts have been undertaken to construct new or use already known prognostic risk scores [14, 15] and to determine prognostic markers for long-term survival after both cardiac surgery and transcatheter procedures.

Pulmonary hypertension, secondary to left ventricular failure, is present in even 65% of patients with symptomatic aortic stenosis [16]. According to the current guidelines, it is classified as pulmonary hypertension in left heart disease (PH-LHD) [16]. PH related to left ventricle heart disease is more common than other types of pulmonary hypertension. Aortic valve calcific degeneration causes

WHAT'S NEW?

Increased pulmonary artery systolic pressure (PASP) is related to left ventricular dysfunction in course of severe aortic stenosis and is associated with a worse prognosis. Transcatheter or surgical aortic valve replacement enables a decrease in PASP. Changes in PASP, 1-month after the procedure, have significant prognostic value for long-term survival. An increase in PASP compared to discharge value is an independent predictor of 1-year mortality. Each 1 mm Hg increase in PASP confers a 4% greater risk of 1-year mortality. Therefore, a 1-month assessment is crucial for evaluating the need for modification of therapy to prevent worse outcomes.

a continuous increase in the left atrial pressure, which is reflected in the pulmonary venous and arterial bed with vasoconstriction and a secondary increase in pulmonary venous and arterial pressures [17]. Pulmonary hypertension further depends on and influences right ventricular function and dilatation. Right ventricular systolic pressure rises, and tricuspid regurgitation increases. Additionally, worsening of the left ventricular function with impairment in systolic function, moderate to severe mitral regurgitation, and concomitant coronary artery disease, further increase PH. Right ventricular dysfunction is common in patients undergoing TAVI [18].

PH has been recognized as an important risk factor for mortality in patients with aortic stenosis and worse survival after surgical aortic valve replacement (SAVR) or TAVI [9, 10, 19, 20]. Importantly, an increase in PH has been proposed as one of the most influencing factors for decision-making in patients with asymptomatic aortic stenosis [21] since it reflects a worsening of heart failure status and long-term prognosis.

Both SAVR and TAVI cause an immediate and long-lasting persistent decrease in pulmonary artery systolic pressure (PASP) [22]. Conversely, aortic balloon valvuloplasty enables a post-procedural PASP decrease, but the effect is not sustained, and the pressure returns to the baseline values during one-year observation [22] due to aortic valve re-stenosis and progressive worsening in left ventricular function.

Though several reports are showing a worse prognosis in patients with aortic stenosis with PH and a decrease in PASP after TAVI and SAVR, little is known about the long-term influence of post-procedural changes in PASP.

The aim was to analyze the influence of pulmonary artery systolic pressure (PASP) changes 1-month after TAVI on long-term survival.

METHODS

Study patients

Three hundred and sixty-two patients underwent TAVI between January 2013 and December 2018 in our hospital after careful clinical assessment by the heart team. Out of this group, 210 patients who underwent detailed echocardiographic and laboratory evaluation 1-month after TAVI were included in the final analysis and followed for up to 12 months (Figure 1). Fifteen patients who died in the hospital or during a 30-day follow-up were not included in the study group. Six of them died during the procedure, one due to stroke as a TAVI complication, and 8 due to multi-organ failure.

Clinical data

Demographic and clinical data were collected during TAVI-related hospitalization and at the 1-month visit.

Follow-up

All 210 patients included in the final analysis were followed up to one year after TAVI. One-year follow-up outcomes, including all-cause death, were assessed based on hospital or ambulatory clinic visits and the national database.

Echocardiographic analysis

Transthoracic echocardiography (TTE) was performed in all patients during the qualification process, before and after the procedure, at discharge, and during 1-year follow-up. The exam was performed on each patient by an experienced echocardiographer according to the same protocol and in compliance with echocardiographic recommendation on PH assessment, after 10–15 minutes of rest before the exam. Aortic valve stenosis was assessed according to the current guidelines on the management of valvular heart disease [1]. Pulmonary artery systolic pressure was evaluated according to the guidelines on the management of pulmonary artery hypertension [16] and was calculated based on the peak tricuspid regurgitation velocity and taking into account pressure in the right atrium (RAP). RAP was estimated based on the diameter and respiratory variation of the inferior vena cava as defined in the guidelines on PH management [16]. The severity of suspected pulmonary hypertension based on the baseline PASP value was evaluated according to criteria used in EuroScore II, which is widely used during TAVI qualification by heart teams as moderate or severe if PASP was 31–55 mm Hg or >55 mm Hg, respectively. The discharge PASP was compared to a 1-month measurement.

Laboratory tests

Blood samples were collected at baseline, after the procedure, and at the 1-month visit. The simple whole blood counts, creatinine, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels were analyzed.

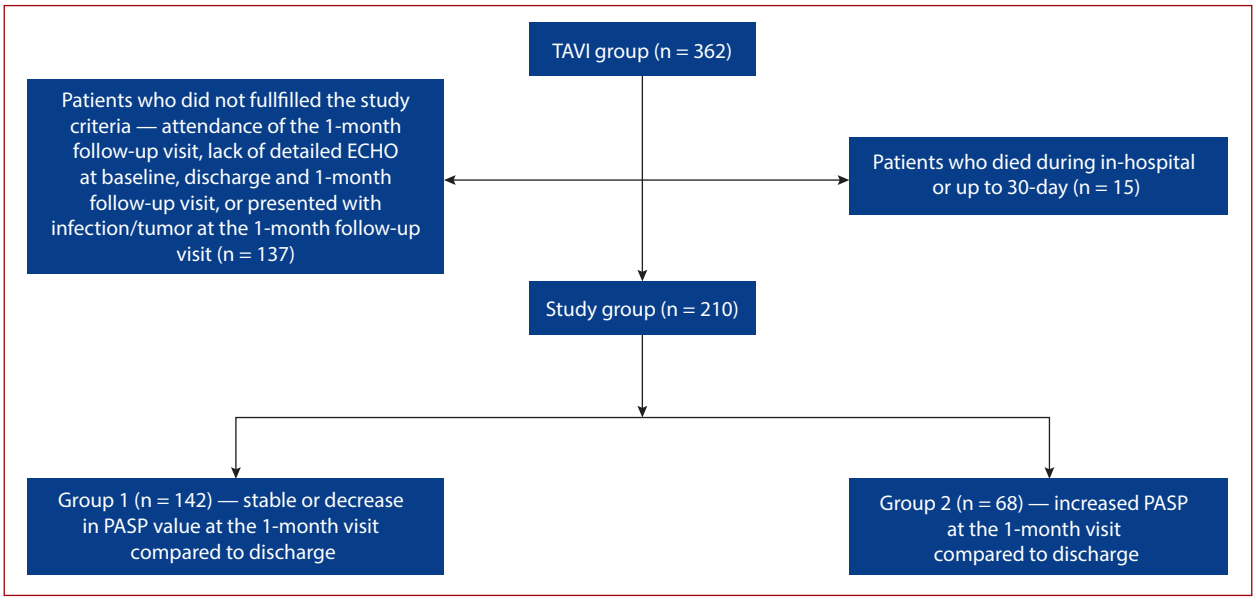

Figure 1. Flowchart of the study inclusion and explanation of exclusion criteria

Abbreviations: ECHO, echocardiography; PASP, pulmonary artery systolic pressure, TAVI, transcatheter aortic valve implantation

Details of TAVI

TAVI procedures were performed by the same experienced team in the hybrid room under fluoroscopic and echocardiographic guidance. The majority of patients (97.6%) had transfemoral access with the use of the Prostar™ system or two Proglides™, and five others including one transcarotid and four direct aorta routes. One-third (65.7%, n = 138) of procedures were performed under general anesthesia, while 72 patients had local anesthesia with sedation. Only self-expandable prostheses were implanted, including Medtronic CoreValve and CoreValve Evolut R, Lotus, Portico, and Symetis Accurate (Table 1). The procedural details were previously described [7].

The study was approved by the institutional Ethics Committee (no. 971/15) and respected the principles outlined in the Declaration of Helsinki.

Statistical analysis

Continuous variables were checked for normality using the Shapiro-Wilk test. Normally distributed data were presented as mean and standard deviation (SD) and compared using the unpaired t-test. If continuous data were not normally distributed, they were expressed as median with interquartile range (IQR) and compared with the nonparametric Mann-Whitney u test. Categorical variables were reported as numbers (n) and percentages (%) and compared with the use of Fisher exact test. A Wilcoxon signed-rank test was used to compare within-subject differences in PASP. The performance of PASP measured at the different time points was assessed using receiver operating characteristic analysis, and the area-under-the-curve values were compared using the DeLong test. We selected the optimal cutoff values of PASP for predicting 1- year mortality by identifying the receiver operating characteristic values that maximized Youden's J statistic (sensitivity + specificity – 1). Kaplan-Meier curves were used to compare 1-year survival in patients with and without a decrease in PASP at 1-month after TAVI and compared with a long-rank test. A Cox proportional-hazards analysis was performed to evaluate predictors of one-year mortality. The input model was adjusted for clinical, echocardiographic, and biochemical variables previously shown to associate with mid-term mortality (sex, age, body mass index [BMI], EuroSCORE II, STS score, diabetes, arterial hypertension, chronic obstructive pulmonary disease [COPD], atrial fibrillation, history of myocardial infarction, NT-proBNP). The final multivariable model was based on the backward stepwise selection of variables at a Wald *P*-value of 0.1. The *P*-values of less than 0.05 were considered statistically significant. Statistical analysis was performed using JASP statistical software and SPSS ver. 23.

RESULTS

The final study group comprised 210 patients (112 females, 53.3%) with a median (IQR) age of 80 (75–83) years. Out of them, 142 had a stable or decreased PASP value (Group 1 — decreased 1-month PASP) while in 68 patients an increase was observed (Group 2 — increased 1-month PASP). These subgroups did not differ in clinical variables (Table 1), but the STS score was higher in Group 2.

Demographic and clinical data are presented in Table 1. In the study group, 34 patients had normal baseline PASP (<30 mm Hg), 126 patients (60%) presented moderate, and 50 patients (23.8%) had a severe increase in preprocedural PASP. In 160 (76.2%) patients, PASP decreased or did not change at discharge measurement. In 102 patients, a stable PASP or a decrease were observed at 1-month follow-up, but in 58 patients PASP increased, and over 10 mm Hg increase was shown in 21 of them. At 1-month

Table 1. Demographic and clinical data

	Whole group (n = 210)	Group 1 (n = 142)	Group 2 (n = 68)	*P*-value
Female sex, n (%)	112 (53.3)	78 (54.9)	34 (50)	0.50
Age, years, median (IQR)	80 (75–83)	81 (75–83)	78.5 (74.75–83)	0.42
BMI, kg/m^2, median (IQR)	27.3 (24.43–30.38)	27.2 (24.53–30.38)	27.5 (24.1–30.13)	1.00
EuroSCORE II, %, median (IQR)	4.82 (2.97–7.96)	4.61 (2.94–7.63)	5.6 (3.04–9.1)	0.20
STS score, %, median (IQR)	4.68 (3.09–9.9)	4.21 (3.08–7.18)	7.05 (3.39–14.31)	0.01
Diabetes, n (%)	75 (35.7)	48 (33.8)	27 (39.7)	0.40
Arterial hypertension, n (%)	154 (73.3)	102 (71.8)	52(76.5)	0.48
COPD, n (%)	43 (20.5)	27 (19)	16 (23.5)	0.45
Atrial fibrillation, n (%)	80 (38.1)	51 (35.9)	29 (42.7)	0.35
MI in history, n (%)	75 (35.7)	44 (31)	31 (45.6)	0.04
Stroke or TIA in history, n (%)	21 (10)	15 (10.6)	6 (8.8)	0.69
New York Heart Association class III/IV, n (%)	194 (92.4)	133 (93.7)	61 (89.7)	0.31
Pacemaker, n (%)	31 (14.8)	18 (12.7)	13 (19.1)	0.22
CABG in history, n (%)	38 (18.1)	26 (18.3)	12 (17.6)	0.91
PCI in history, n (%)	76 (36.2)	51 (35.9)	25 (36.8)	0.91
Baseline AVA, cm^2, median (IQR)	0.7 (0.6–0.8)	0.7 (0.575–0.8)	0.6 (0.6–0.7)	0.95
Baseline MPG, mm Hg, median (IQR)	55 (45–66)	56 (46.5–66.75)	52 (42–63)	0.07
Baseline PPG, mm Hg, median (IQR)	89 (76–108)	89.5 (78.25–109.5)	85.5 (70–104.25)	0.22
Left ventricular ejection fraction, %, median (IQR)	55 (45–60)	55 (45–60)	50 (40–60)	0.18
Baseline PASP, mm Hg, median (IQR)	42 (35–53.75)	42 (35–51.5)	45 (35–54.25)	0.41
NT-proBNP, pg/ml, median (IQR)	2480.05 (1041.5–6540)	2174.5 (1000–6751)	2995 (1678.5–5850.25)	0.37
Femoral artery access, n (%)	205 (97.6)	140 (98.6)	65 (95.6)	0.18
Other access, n (%)	5 (2.4)	2 (1.4)	3 (4.4)	
CoreValve/EvolutR, n (%)	152 (72.4)	96 (67.6)	56 (82.4)	0.13
Symetis, n (%)	16 (7.6)	14 (9.9)	2 (2.9)	
Lotus, n (%)	34 (16.2)	26 (18.3)	8 (11.8)	
Portico, n (%)	8 (3.8)	6 (4.2)	2 (2.9)	
Anesthesia general, n (%)	138 (65.7)	88 (62)	50 (73.5)	0.1
Deaths in 1 to 12 months follow up, n (%)	20 (16.7)	9 (6.3)	11 (16.2)	0.02

Abbreviations: AVA, aortic valve area; BMI, body mass index; CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; MI, myocardial infarction; MPG, mean pressure gradient; NYHA, New York Heart Association; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PASP, pulmonary artery systolic pressure; PCI, percutaneous coronary intervention; PPG, peak pressure gradient; STS, Society of Thoracic Surgeons; TIA, transient ischemic attack

follow-up, all patients presented improvement in clinical status. During 1-year follow-up, 20 patients died (9.5%), 9 in Group 1 and 11 in Group 2 (P = 0.02), irrespective of the death cause. The follow-up was carried out for up to 12 months, with mean (SD, min-max) 348.762 (59.8; 63–365) days.

PASP values' comparison between the survivor and deceased groups in the studied periods: at baseline, discharge, and 1-month visit are presented in Figure 2A–C.

A ROC curve analysis for discharge PASP and 30-day PASP was performed and revealed a significant value of 1-month measurement for 1-year mortality prediction (Figure 3). The cutoff for the PASP value predictive of mortality was ≤41 mm Hg, yielding a sensitivity of 67.4% and a specificity of 75%. Baseline NT-proBNP decreased significantly (P <0.001) at 1-month assessment from the median (IQR) value of 2480.05 (1041.5–6540) pg/ml to 1248 (622.5–2307) pg/ml.

Survival was assessed using the Kaplan–Meier analysis and showed significantly higher mortality in patients without a 1-month PASP decrease (Figure 4). The analysis of survivors' and deceased patients' characteristics is presented in Table 2. Non-survivors were characterized by higher values of discharge and 1-month PASP, though the baseline PASP values did not differ significantly between subjects.

The results of the univariable Cox proportional-hazards analysis are summarized in Table 3. In the multivariable analysis, PASP measured at 1-month after TAVI (hazard ratio [HR], 1.040; 95% confidence interval [CI], 1.019–1.062; P <0.001) was independent predictor of 1-year mortality, as was BMI (HR, 0.861; 95% CI, 0.760–0.976; P = 0.02) and diabetes (HR, 3.433; 95% CI, 1.205–9.781; P = 0.02). Each 1 mm Hg increase in PASP predicts a 4% increase in the risk of death. Changes in echocardiographic parameters between discharge and 1-month examination in survivors and deceased are presented in Table 4. The observation was confirmed by the sensitivity analysis testing the multivariable model (Supplementary material, *Table S1*) adjusting for both clinical and echocardiographic parameters (PASP, left ventricular ejection fraction, peak transaortic gradient) measured at 1-month, where PASP at 1-month follow-up was the strongest echocardiographic predictor of 1-year mortality.

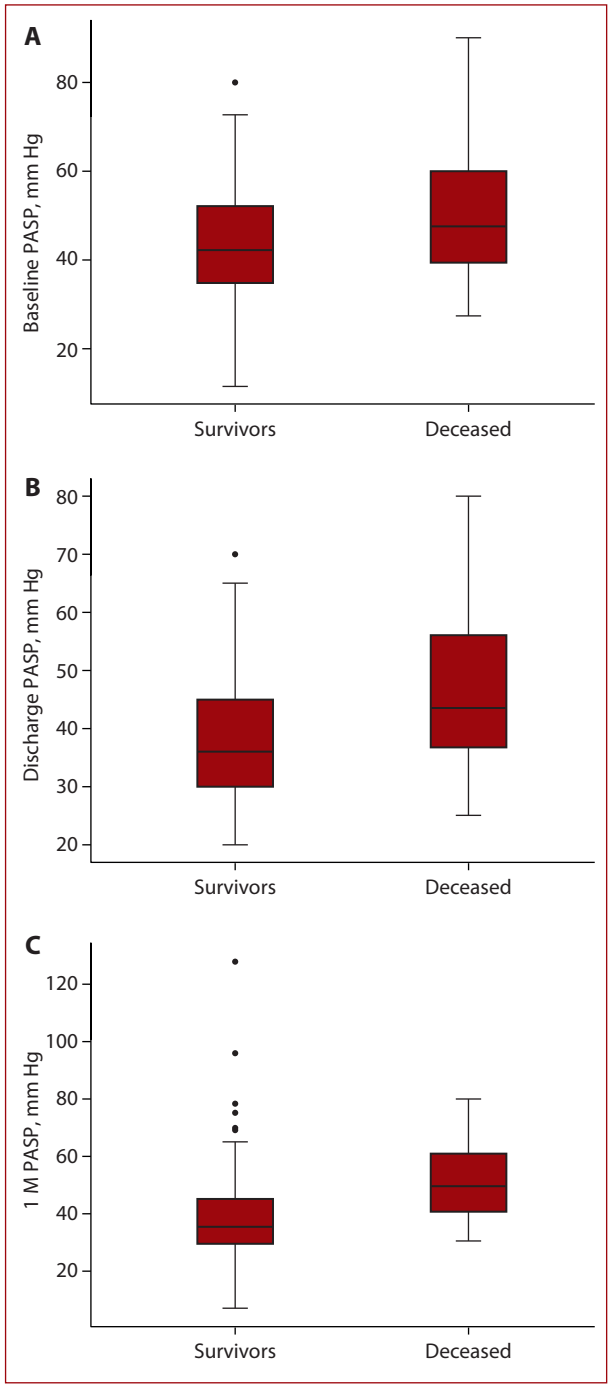

Figure 2. Pulmonary artery systolic pressure values comparison between survivors and deceased groups: **A.** at baseline; **B.** at discharge; **C.** at the 30-day visit

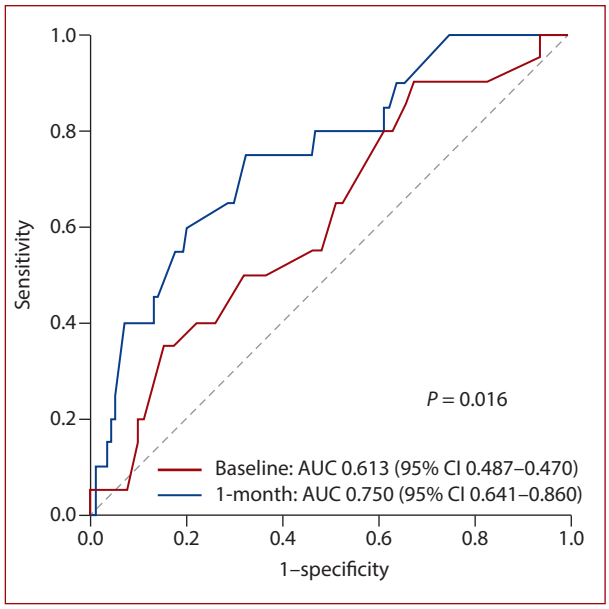

Figure 3. Comparison of pulmonary arterial systolic pressure at baseline (red) and 1-month (blue) after transcatheter aortic valve implantation for prediction of 1-year mortality

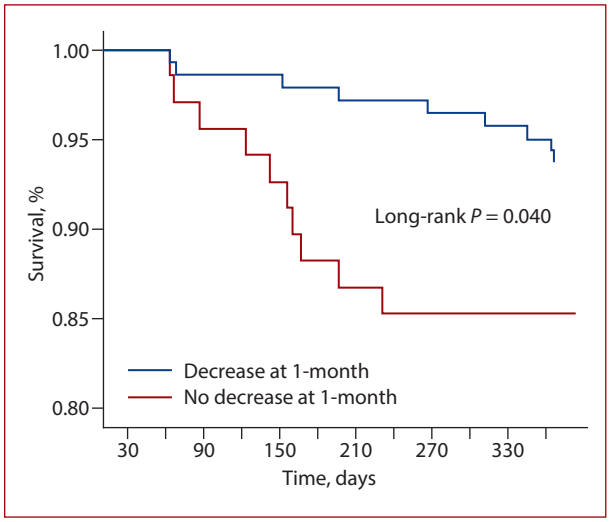

Figure 4. Comparison of 1-year survival in patients with (blue) and without (red) a decrease in pulmonary artery systolic pressure at 1-month after transcatheter aortic valve implantation

DISCUSSION

The main finding of our study is the high prognostic value of PASP measured one month after TAVI for one-year mortality. PASP was the strongest echocardiographic predictor, and the lack of its decrease at 1-month follow-up was associated with worse outcomes.

Several studies [19, 23, 24] proved that PH secondary to aortic stenosis impairs mortality in patients treated with TAVI. Though right heart catheterization (RHC) is currently an established tool for PH diagnosis, still echocardiography remains the best and easiest screening method for the PH probability evaluation. The most precise assessment of PH-LVD according to the guidelines on PH management [16] includes measurement of tricuspid regurgitation velocity (TRV) and presence of other PH echocardiographic signs: right ventricular/left ventricular basal diameter, left ventricular eccentricity index, right ventricular outflow acceleration time, midsystolic notch, early diastolic pulmonary regurgitation velocity, the diameter of the inferior vena cava, and right atrial area. However, the majority of studies use PASP as a key component of PH evaluation. O'Sullivan et al. [25] tested computed tomography for PH evaluation

Table 2. The analysis of survivors' and deceased patients' characteristics

	Survivors (n = 190)	Deceased (n = 20)	*P*-value
Female sex, n (%)	106 (55.8)	6 (30)	0.03
Age, years, median (IQR)	80 (75–83)	77 (73.25–79.5)	0.045
BMI, kg/m^2, median (IQR)	27.35 (24.53–30.73)	25.45 (23.95–27.6)	0.08
EuroSCORE II, %, median (IQR)	4.84 (3.03–8.05)	4.32 (2.67–6.47)	0.47
STS score, %, median (IQR)	4.54 (3.08–10.21)	5.17 (3.28–8.47)	0.62
Diabetes mellitus, n (%)	63 (33.2)	12 (60)	0.02
Arterial hypertension, n (%)	139 (73.2)	15 (75)	0.86
COPD, n (%)	38 (20)	5 (25)	0.60
Atrial fibrillation, n (%)	70 (36.8)	10 (50)	0.25
MI in history, n (%)	69 (36.3)	6 (30)	0.58
NT-proBNP, pg/ml, median (IQR)	2454.8 (1000–6500)	5222 (1500–8604)	0.20
NT-proBNP 1-month, pg/ml, median (IQR)	1206 (605–2212)	1913 (1009.5–3661)	0.11
Baseline LVEF, %	55 (45–60)	55 (38.75–60)	0.91
Discharge LVEF, %	55 (45–60)	55 (43.75–60)	0.79
1-month LVEF, %	58 (50–60)	60 (48.75–60)	0.94
Baseline MPG, mm Hg, median (IQR)	56 (45.5–66)	47.5 (41.75–64)	0.28
Discharge MPG, mm Hg, median (IQR)	9 (6–11)	7.9 (5.48–11.5)	0.53
1-month MPG, mm Hg, median (IQR)	8 (6–11)	7.5 (4.98–10.13)	0.35
Baseline PPG, mm Hg, median (IQR)	90 (78–108)	83.5 (61.7–103.5)	0.31
Discharge PPG, mm Hg, median (IQR)	17 (12–21)	17 (11–23)	0.83
1-month PPG, mm Hg, median (IQR)	15 (10–20)	18 (10.68–20.5)	0.66
PASP baseline, mm Hg, median (IQR)	42 (35–52)	47.5 (40–60)	0.10
PASP discharge, mm Hg, median (IQR)	36 (30–45)	43.5 (36.5–56.25)	0.02
PASP 1 month, mm Hg, median (IQR)	35 (29.25–45)	50 (40.75–60.5)	<0.001

Abbreviations: grad, gradient; other — see Table 1

Table 3. Association of clinical parameters and pulmonary artery systolic pressure at 1-month post-TAVI with the risk of mortality during 1-year follow-up

	Univariable analysis		Multivariable analysis[a]	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Female sex	0.384 (0.146–1.009)	0.05	—	—
Age, per 1 year increase	0.955 (0.902–1.011)	0.12	—	—
BMI, per 1 kg/m^2 increase	0.941 (0.849–1.042)	0.24	0.861 (0.760–0.976)	0.02
EuroSCORE II	1.023 (0.971–1.079)	0.39	—	—
STS score	1.023 (0.969–1.081)	0.41	—	—
Diabetes	3.255 (1.281–8.269)	0.01	3.433 (1.205–9.781)	0.02
Arterial hypertension	1.376 (0.457–4.145)	0.57	—	—
COPD	1.406 (0.507–3.905)	0.51	—	—
Atrial fibrillation	1.853 (0.753–4.561)	0.18	—	—
MI in history	0.646 (0.233–1.793)	0.40	—	—
NT-proBNP[b]	1.662 (0.634–4.362)	0.30	—	—
PASP at 1-month follow-up, per 1 mm Hg increase	1.033 (1.015–1.050)	<0.001	1.040 (1.019–1.062)	<0.001

[a]Final models based on the backward stepwise selection of variables at a Wald *P*-value of 0.1. [b]Per doubling of variable

Abbreviations: see Table 1

Table 4. Changes in echocardiographic parameters between discharge and 1-month examination in survivors and deceased

	Discharge	One-month	*P*-value
Whole group — EF, %	55 (45–60)	60 (50–60)	<0.001
Survivors — EF, %	55 (45–60)	58 (50–60)	<0.001
Deceased — EF, %	55 (43.75–60)	60 (48.75–60)	0.12
Whole group — MPG, mm Hg	8.9 (6–11)	8 (5.875–11)	0.045
Survivors — MPG, mm Hg	9 (6–11)	8 (6–11)	0.10
Deceased — MPG, mm Hg	7.9 (5.5–11.5)	7.5 (5–10.1)	0.21
Whole group — PPG, mm Hg	17 (12–21.33)	15 (10–20)	0.002
Survivors — PPG, mm Hg	17 (12–21)	15 (10–20)	0.003
Deceased — PPG, mm Hg	17 (11–23)	18 (10.7–20.5)	0.34

Continuous variables are expressed as the medians (interquartile range [IQR])

Abbreviations: EF, ejection fraction; other — see Table 1

and proposed the ratio of the diameter of the main pulmonary artery to the diameter of the ascending aorta as best correlating with PASP. In patients with PA/AA ratio >0.80, PH is more likely, however, PH cannot be reliably excluded among patients with lower PA/AA ratio values. Kleczynski et al. [19] showed that a simple assessment of TRV is beneficial. They showed that TRV >3.4 m/s indicates worse impaired clinical outcomes after TAVI. Several authors, including Kokkinidis et al. [26] underlined heterogeneity in PH definitions reported in different studies. The majority of studies commonly use the cutoff values available from the EuroSCORE II calculator. This choice seems reasonable as it enables easy comparison with previous and current surgical analyses commonly using surgical scores. It should be mentioned, however, that only EuroSCORE II, and not STS, contains PASP evaluation. Nevertheless, the significance of PH for survival seems crucial. In the meta-analysis by Tang et al. [27] of 16 studies enrolling 9204 patients with AS who underwent TAVI, independent of different methods of PH assessment, baseline PH increased overall 30-day, 1-year, and 2-year all-cause mortality, the risk of acute kidney injury, and the risk of stroke. The presence of post-TAVI PH was also related to higher 2-year mortality. Nijenhuis et al. [28], who divided patients into subgroups based on PASP according to cutoff values recommended in current PH guidelines [16], still showed worse outcomes in patients with "high PH probability". They also claimed the importance of additional PH signs. Different cutoff values have been presented to determine the most critical value of PASP. Kokkinidis et al. [26] in their meta-analysis of 22 studies showed PASP >60 mm Hg as most likely to be associated with the worst outcomes among the different endpoints. Luçon et al. [29] found that PASP ≥40 mm Hg was associated with increased 1-year mortality but not with increased 30-day mortality. The functional status was significantly improved regardless of the PASP value.

Similarly, PH worsened early results and long-term mortality after SAVR. Roselli et al. [20] reported prolonged hospitalization, renal and respiratory failure, and sepsis in patients with PH. Interestingly, not a sustained decrease in PH, but an even increase, was observed in the long-term observation after SAVR in their analysis.

Lindman et al. [23] underlined that not hemodynamic indices but clinical factors should be taken into consideration in the long-term prediction of survival after TAVI. The more comorbidities, including kidney dysfunction, pulmonary disease, and left ventricular performance are present, the higher the risk of PH influences survival. In our analysis of clinical factors, BMI and diabetes correlated with a worse 1-year prognosis.

TAVI leads to an immediate improvement in echocardiographic parameters [30] and functional status. A significant decrease in PASP is observed in most studies immediately after the procedure and in the midterm follow-up after TAVI [27, 31]. The observation of a worse prognosis was confirmed in our analysis with the decrease in PASP in the echocardiographic exams at discharge and 1-month follow-up. However, there are some patients with sustained or increased PH after the procedure, at the short term-observation. Medvedofsky et al. [32] reported that 57% of the patients with prior PH presented a reduction in PASP to below 50 mm Hg and identified COPD as the only independent predictor of persistent PH. O'Sullivan et al. [33] showed that PASP immediately improved after TAVI in patients with postcapillary combined PH, but not in those with precapillary PH. Also, Sultan et al. [34] presented worse results in the group of patients with combined pre- and post-capillary PH. These observations may partially explain the lack of improvement in some subjects with PH.

We showed that the group of patients without a decrease in PASP present a considerably worse 1-year prognosis. Each 1 mm Hg increase in PASP predicts a 4% increase in the risk of death. We also found that in survivors ejection fraction increased and peak gradient decreased at 1-month evaluation in comparison with discharge values. Conversely, these parameters did not change significantly in non-survivors. However, in the sensitivity analysis, only PASP showed the prognostic value for 1-year mortality.

TAVI has developed sufficiently during the last years and is a common method of aortic stenosis treatment. Therefore, with an increase in the TAVI-treated population, the diagnostic vigilance in the follow-up may decrease. Based on the results of our study we strongly believe that a 1-month follow-up with even a simple echocardiographic assessment is crucial for the improvement of long-term results. Patients with sustained or increased PH should undergo scrutiny and clinical investigation to identify potential clinical factors which may be modified. Therefore, a one-month follow-up should not be omitted. Moreover, these patients would probably need more frequent follow-up visits than those with a decrease in PASP 1-month after TAVI.

The use of echocardiography as the only method of PH evaluation, without RHC assessment, may be treated as a limitation of our study. PAPS evaluated by echocardiography is burdened with an inherent error. However, echocardiography remains a standard tool in everyday practice and is easier, non-invasive, and widely available than RHC. No other commonly used methods of PH evaluation (RHC) are available in ambulatory settings after TAVI.

CONCLUSION

The decreased or stable value of PASP at 1-month follow-up may predict better one-year survival after TAVI, while each 1 mm Hg increase in PASP confers a 4% greater risk of 1-year mortality.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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