This article has an erratum available at: https://dx.doi.org/10.21037/cdt-23-477 and the article has been update on 2024-01-04 at here.

Original Article

Prognostic implication of pulmonary artery pulsatility index before transcatheter aortic valve replacement

Akira Oshima[#], Teruhiko Imamura[#], Shuhei Tanaka, Hiroshi Onoda, Ryuichi Ushijima, Mitsuo Sobajima, Nobuyuki Fukuda, Hiroshi Ueno, Koichiro Kinugawa

The Second Department of Internal Medicine, University of Toyama, Toyama, Japan

Contributions: (I) Conception and design: T Imamura; (II) Administrative support: H Ueno, K Kinugawa; (III) Provision of study materials or patients: T Imamura, N Fukuda; (IV) Collection and assembly of data: A Oshima, S Tanaka, H Onoda, R Ushijima, M Sobajima; (V) Data analysis and interpretation: T Imamura; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

[#]These authors contributed equally to this work.

Correspondence to: Teruhiko Imamura, MD, PhD, FAHA, FACC, FESC, FHFSA, FJCC, FAPSC, FACP, FICA, FASA. The Second Department of Internal Medicine, University of Toyama, 2630 Sugitani, Toyama 930-0194, Japan. Email: teimamu@med.u-toyama.ac.jp.

Background: Pulmonary artery pulsatility index (PAPI) is a recently proposed hemodynamic index that is associated with right ventricular function independently on volume status. However, its clinical implication in patients receiving transcatheter aortic valve replacement (TAVR) remains uninvestigated. Baseline PAPI might be a promising index that stratify patients' clinical outcomes following TAVR.

Methods: In this retrospective cohort study, patients with severe aortic stenosis who received TAVR and completed baseline invasive hemodynamic assessments using right heart catheterization were included. The impact of baseline PAPI on the 2-year incidence of heart failure re-hospitalizations following TAVR was investigated.

Results: A total of 227 patients (median 86 years old, 29% men) were included. Median baseline PAPI was 3.6 (2.6, 5.5). PAPI was an independent predictor of the primary endpoint with adjusted hazard ratio of 7.01 (95% confidence interval: 2.08–23.2, P=0.008) with a cutoff of 2.1, which significantly stratified the 2-year cumulative incidence of primary endpoint (lower PAPI 21% versus high PAPI 5%, P=0.003).

Conclusions: Baseline impaired right ventricular function, indicated by lower PAPI below 2.1, was associated with the occurrence of heart failure following TAVR. Further studies are warranted to clarify the mechanism underlying on our findings and the prognostic implication of aggressive intervention to improve PAPI.

Keywords: Right ventricular failure; hemodynamics; aortic valve disease

Submitted Oct 27, 2021. Accepted for publication Jan 30, 2022.
doi: 10.21037/cdt-21-682
View this article at: https://dx.doi.org/10.21037/cdt-21-682

Introduction

The prevalence of aortic stenosis increases as aging, and it reaches approximately 7% in a population aged over 80 years old. Transcatheter aortic valve replacement (TAVR) has been introduced as an alternative to surgery during the last decades (1,2).

TAVR was originally indicated for those with a high surgical risk (STS score above 8%), but has recently been indicated even for those with an intermediate or lower surgical risk (3). The strategy to risk stratify is of great importance for optimal patient selection and greater clinical outcomes.

The existence of baseline right ventricular failure is an independent risk factor of mortality following surgical/transcatheter interventions to aortic stenosis (4,5). Right ventricular function is assessed by using echocardiography parameters including tricuspid annular plane systolic

excursion and right ventricular fractional area change (6). One of the limitations to utilizing them is a requirement of expert technique for the measurement.

Another parameter to assess right ventricular function is pulmonary artery pulsatility index (PAPI), which is calculated easily by using several standard hemodynamics parameters including pulmonary artery pulse pressure and mean right atrial pressure (7). Lower PAPI is one of the useful risk factor of post-procedural morbidity including right ventricular failure and mortality following durable left ventricular assist device implantation (8) or transcatheter mitral valve repair (9), whereas little is known about its clinical implication in patients receiving TAVR. In the present study, we investigated the prognostic impact of baseline PAPI in patients receiving TAVR. We present the following article following the STROBE reporting checklist (available at https://cdt.amegroups.com/article/view/10.21037/cdt-21-682/rc).

Methods

Patient selection

In this retrospective cohort study, consecutive patients with severe aortic stenosis who received TAVR at University of Toyama between May 2015 and November 2020 were prospectively listed in our registry. The initial date of this time range is when our institutional registry was started. The final date of this time range is when prospective listing on the institutional registry was temporarily terminated. Of them, data of patients who received invasive hemodynamic assessments within one week before TAVR were retrospectively retrieved. Patients were followed for two years or until Jun 2021 according to the standard follow-up manner by the board-certified cardiologists. Written informed consents were obtained from participants before listing. Institutional review board of University of Toyama approved the study protocol (R2015154, April 11 2016). This study was conducted according to the Declaration of Helsinki (as revised in 2013).

TAVR procedure

Patients with severe aortic stenosis with peak velocity >4.0 m/s or mean pressure gradient above 40 mmHg were considered to receive TAVR. The indication of TAVR was determined by the heart-valve team conference involving multiple types of clinicians. Patients received TAVR according to the standard procedure. Patients received self-expandable valves or balloon-expandable valves under general or local anesthesia.

Hemodynamic assessment

Invasive hemodynamic assessments were performed within one week before TAVR in the catheter laboratory room by the board-certified cardiologists in clinically stable conditions. Standard hemodynamic data were measured successively. As a primary independent variable, PAPI was calculated as follows: [pulmonary artery pressure difference between systole and diastole (mmHg)]/[central venous pressure (mmHg)].

Other clinical variables

Demographics, laboratory, and echocardiography data obtained within one week before TAVR were obtained as baseline characteristics. Death or heart failure readmissions requiring IV diuretics therapy were counted. A cumulative incidence of heart failure readmission during the two-year observational period following TAVR were defined as a primary outcome.

Statistical analysis

Continuous variables were shown as median and interquartile irrespective of their distribution and compared between the two groups using Mann-Whitney U test, given the small sample size. Categorical variables were shown as number and percentage and compared between the two groups using Fisher's exact test. A 2-tailed value of $P<0.05$ was considered statistically significant. Statistical analyses were performed using SPSS Statistics 22 (SPSS Inc., Armonk, IL, USA).

The primary endpoint was a cumulative incidence of two-year heart failure readmission. The impact of baseline PAPI on the primary endpoint was the major concern in this study.

A cutoff of PAPI was calculated by time-dependent receiver operating characteristics analysis for the primary endpoint. Kaplan-Meier analyses were performed to assess the cumulative incidence of the primary endpoints. Cox proportional hazard ratio regression analyses were performed to investigate the impact of baseline PAPI on the primary endpoint. The impact of baseline PAPI was adjusted for other variables that were assumed to

have potential impacts on the primary endpoint: age, hemoglobin, estimated glomerular filtration ratio, plasma B-type natriuretic peptide level, and left ventricular ejection fraction at baseline.

Results

Pre-TAVR baseline characteristics

Among 294 patients, 67 patients without invasive hemodynamic data were excluded. A total of 227 patients {median 86 [83, 88] years old, 29% men} were finally included (*Table 1*). All patients had severe aortic stenosis with 4.5 [4.0, 4.9] m/s of peak velocity and 47 [38, 57] mmHg of mean pressure gradient. Baseline plasma B-type natriuretic peptide level was 262 [123, 498] pg/mL and PAPI was 3.6 [2.2, 5.5]. Baseline PAPI distributed widely between 0.8 and 15.0 (*Figure 1*).

Post-TAVR clinical characteristics

All TAVR were successfully performed without any critical complications. Plasma B-type natriuretic peptide improved significantly following TAVR (P<0.05; *Table 2*).

Prognostic impact of baseline PAPI

A cutoff of baseline PAPI to predict the primary endpoint was calculated as 2.1 (sensitivity 0.87, specificity 0.42, area under the curve 0.68; Figure S1). Area under the curves of central venous pressure and pulmonary artery pulsatility were 0.62 and 0.57, respectively. We stratified patients into two groups according to this cutoff (i.e., lower PAPI versus higher PAPI).

Incidences of 30-day complications including pacemaker implantation, stroke, vascular complication, and heart failure were statistically not different between the two groups (P>0.05 for all; *Table 3*).

During the observational period, 12 patients had heart failure admissions. A lower PAPI was associated with the primary endpoint with unadjusted hazard ratio of 4.84 (95% confidence interval: 1.54–15.3, P=0.007) and adjusted hazard ratio 7.01 (95% confidence interval: 2.08–23.2, P=0.008) accounting for age, hemoglobin, estimated glomerular filtration ratio, plasma B-type natriuretic peptide level, and left ventricular ejection fraction as potential confounders (*Table 4*). A lower PAPI was associated with higher 2-year cumulative incidence of the primary endpoint versus higher PAPI (21% versus 5%, P=0.003; *Figure 2*).

During the observational period, 14 patients died. The 2-year survival was not significantly stratified between the lower PAPI group and the higher PAPI group (87% versus 97%, P=0.37).

Patient characteristics stratified by baseline PAPI

Among baseline characteristics, there were no statistically significant difference except for higher hemoglobin and central venous pressure in the lower PAPI group (P<0.05 for both; *Table 1*). Pulmonary capillary wedge pressure tended to be higher in the lower PAPI group (P=0.062). Following TAVR, plasma B-type natriuretic peptide level was lower in the lower PAPI group (P=0.016; *Table 2*).

Discussion

In the present study, we investigated the impact of baseline lower PAPI, indicating impaired right ventricular function, on the two-year heart failure readmission. Major findings are followings: (I) approximately 15% of the patients had lower PAPI at baseline defined as PAPI <2.1; (II) a lower PAPI at baseline was associated with higher incidence of heart failure recurrence following TAVR.

Lower PAPI in patients with aortic stenosis

There is no gold standard to accurately quantify the severity of right ventricular failure. Classical tool to assess the severity of right ventricular failure is echocardiography (6), although one of their limitations is requirement of expert technique. We in this study focused on PAPI, which is a recently-introduced novel parameter to quantify right ventricular function by representing the pulse pressure of pulmonary artery per central venous pressure unit, which is created by systolic function of right ventricle (7). Impaired right ventricle cannot create pulse pressure of pulmonary artery despite incremental preload on right ventricle (i.e., central venous pressure).

Association between right ventricular function and aortic stenosis is reported. Previous studies found that some of the severe aortic stenosis accompanied right ventricular failure (4,10). Left ventricular remodeling due to restricted forward flow though aortic valve increases intra-cardiac pressure and develops secondary pulmonary hypertension, which increases afterload on right ventricle and impairs right ventricular function, as observed also in this study.

Table 1 Baseline characteristics

	Total (n=227)	Lower PAPI (n=33)	Higher PAPI (n=194)	P value
Demographics				
Age, years, median [range]	86 [83, 88]	85 [83, 88]	86 [82, 89]	0.26
Men	66 (29%)	13 (39%)	53 (27%)	0.21
Body surface area, m^2, median [range]	1.38 [1.29, 1.52]	1.43 [1.31, 1.61]	1.38 [1.28, 1.50]	0.15
Approach				0.22
Trans-femoral	214 (94%)	30 (91%)	189 (97%)	–
Trans-apex	5 (2%)	1 (3%)	3 (2%)	–
Trans-subclavian	7 (3%)	1 (3%)	2 (1%)	–
Direct aorta	1 (1%)	1 (3%)	0 (0%)	–
Comorbidity				
History of stroke	46 (20%)	2 (6%)	33 (17%)	0.12
Diabetes mellitus	52 (23%)	8 (24%)	31 (16%)	0.32
Hyperlipidemia	136 (60%)	17 (52%)	85 (44%)	0.44
Peripheral artery disease	83 (37%)	12 (36%)	47 (24%)	0.20
Chronic obstructive pulmonary disease	19 (8%)	2 (6%)	13 (7%)	1.0
History of cardiac surgery	17 (7%)	3 (9%)	6 (3%)	0.13
Atrial fibrillation	23 (10%)	4 (12%)	10 (5%)	0.91
Laboratory data, median [range]				
Hemoglobin, g/dL	10.9 [9.9, 12.1]	11.7 [10.7, 12.6]	10.8 [9.8, 11.9]	0.007*
Serum albumin, g/dL	3.8 [3.5, 4]	3.8 [3.5, 4.0]	3.8 [3.5, 4.0]	0.82
Serum sodium, mEq/L	140 [139, 142]	140 [139, 142]	140.5 [139, 142]	0.92
eGFR, mL/min/1.73 m^2	49.1 [37.4, 61.7]	50.1 [33.5, 60.6]	49.1 [37.9, 61.9]	0.88
Plasma B-type natriuretic peptide, pg/mL	262 [123, 498]	191 [114, 420]	270 [123, 506]	0.51
Echocardiography				
Peak velocity at aortic valve, m/sec, median [range]	4.5 [4.0, 4.9]	4.5 [4.2, 4.7]	4.5 [4.0, 4.9]	0.61
Mean pressure gradient at aortic valve, mmHg, median [range]	47 [38, 57]	47 [41, 54]	47 [38, 57]	0.63
Left ventricular end-diastolic diameter, mm, median [range]	46 [42, 51]	46 [41, 52]	46 [42, 51]	0.56
Left ventricular ejection fraction, %, median [range]	65 [54, 70]	65 [50, 69]	65 [54, 70]	0.80
Moderate or greater MR	16 (7%)	6 (18%)	10 (5%)	0.081
Moderate or greater TR	6 (3%)	0 (0%)	6 (3%)	0.33
Hemodynamics, median [range]				
Systolic blood pressure, mmHg	114 [105, 124]	110 [105, 123]	115 [105, 125]	0.30
Heart rate, bpm	69 [60, 77]	71.5 [61, 87.25]	68 [59.75, 75.25]	0.15
Central venous pressure, mmHg	5 [3, 7]	8 [6, 9]	5 [3, 6]	<0.001*
Systolic pulmonary artery pressure, mmHg	31 [26, 36]	28 [24, 31]	31 [26, 37]	0.021*
Diastolic pulmonary artery pressure, mmHg	12 [9, 15]	15 [12, 18]	11 [9, 15]	<0.001*
Pulmonary artery pulsatility index	3.6 [2.6, 5.5]	1.7 [1.3, 1.9]	4.3 [3.0, 6.0]	<0.001*
Pulmonary capillary wedge pressure, mmHg	11 [9, 15]	13 [10, 16]	11 [8, 15]	0.062
Cardiac index, L/min/m^2	2.71 [2.39, 3.03]	2.53 [2.18, 2.85]	2.75 [2.40, 3.05]	0.37

*, P<0.05. PAPI, pulmonary artery pulsatility index; eGFR, estimated glomerular filtration ratio; MR, mitral regurgitation; TR, tricuspid regurgitation. Continuous variables are presented as median and interquartile. Categorical variables are presented as numbers and percentages.

Left ventricular remodeling with systolic and diastolic dysfunction, which are often observed in patients with severe aortic stenosis, would also affect geometrically and functionally on right ventricle (11).

Prognostic implication of baseline PAPI following TAVR

There are several conflicting studies investigating the impact of TAVR on right ventricular function. Kempny and colleagues observed persistently unchanged right ventricular function assessed by echocardiography following TAVR (12). Poch and colleagues observed that approximately half of the patients with baseline impaired right ventricular function enjoyed their improvement following TAVR (13). TAVR would considerably reduce afterload on left ventricle and ameliorate secondary pulmonary hypertension. Geometrical improvement in left ventricle would also positively affect right ventricle. Nevertheless, too remodeled right ventricle might be refractory to such improvement. Furthermore, incremental systemic circulation following TAVR might rather increase preload on right ventricle and impair right ventricular function. Given all together, at least some of the impaired right ventricle with lower PAPI at baseline might persist even after successful TAVR.

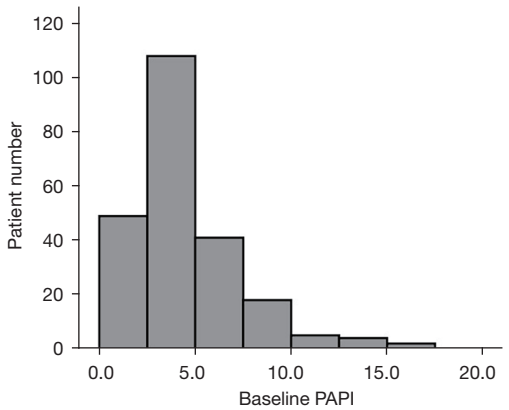

Figure 1 Distribution of baseline PAPI. PAPI, pulmonary artery pulsatility index.

Following TAVR, plasma B-type natriuretic peptide level was lower in patients with lower PAPI, although their levels were relatively lower in both groups. Such patients might have predominant right ventricular failure, which in general paradoxically does not show extremely high B-type natriuretic peptide levels irrespective of the stage of heart failure. PAPI consistently had no significant correlation with plasma B-type natriuretic peptide levels (P=0.86).

It is plausible that the existence of baseline right ventricular failure, indicated by lower PAPI, was associated with

Table 2 Post-TAVR laboratory data

	Total (n=227)	Lower PAPI (n=33)	Normal PAPI (n=194)	P value
Hemoglobin, g/dL	10.2 [9.6, 11.0]	10.4 [10.0, 11.4]	10.1 [9.5, 10.9]	0.069
Serum albumin, g/dL	3.4 [3.1, 3.6]	3.6 [3.2, 3.7]	3.6 [3.4, 4.3]	0.066
Serum sodium, mEq/L	139 [138, 141]	139 [137, 140]	140 [138, 141]	0.068
eGFR, mL/min/1.73 m^2	52.2 [39.1, 66.4]	51 [43.5, 61.6]	52.2 [38.8, 66.7]	0.97
Plasma B-type natriuretic peptide, pg/mL	101 [57, 193]	77 [34, 143]	106 [64, 207]	0.016*

Data presented as median [range]. *, P<0.05. TAVR, transcatheter aortic valve replacement; PAPI, pulmonary artery pulsatility index; eGFR, estimated glomerular filtration ratio. Variables are presented as median and interquartile.

Table 3 Thirty-day major complications following TAVR

	Lower PAPI (n=33)	Higher PAPI (n=194)	P value
Pacemaker implantation	2 (6%)	21 (11%)	0.40
Stroke	0	2 (1%)	0.56
Vascular complication	0	4 (2%)	0.41
Heart failure	1 (3%)	1 (1%)	0.15

TAVR, transcatheter aortic valve replacement; PAPI, pulmonary artery pulsatility index.

Table 4 Association between baseline PAPI and the primary endpoint

	Hazard ratio	95% confidence interval	P value
Unadjusted PAPI	4.84	1.54–15.3	0.007*
Adjusted PAPI	7.01	2.08–23.2	0.008*
Age, years	1.14	0.97–1.33	0.11
Hemoglobin, g/dL	1.21	0.77–1.87	0.41
eGFR, mL/min/1.73 m^2	0.95	0.90–0.99	0.017*
Plasma B-type natriuretic peptide, pg/mL	0.99	0.99–1.01	0.62
Left ventricular ejection fraction, %	1.02	0.97–1.08	0.43

*, P<0.05 by Cox proportional hazard ratio regression analysis. PAPI, pulmonary artery pulsatility index; eGFR, estimated glomerular filtration ratio. In the multivariate analysis, PAPI was adjusted for age, hemoglobin, estimated glomerular filtration ratio, and plasma B-type natriuretic peptide level.

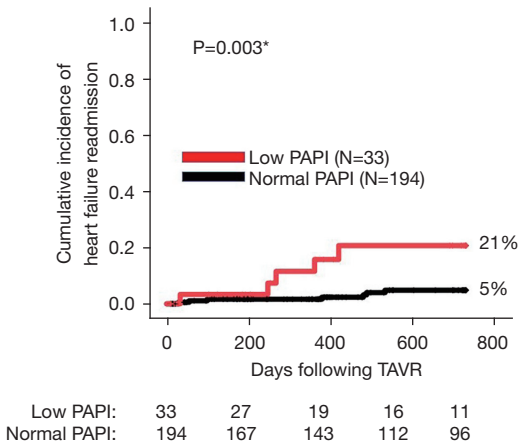

Figure 2 Cumulative incidence of the primary endpoint stratified by the level of baseline PAPI. *, P<0.05 by log-rank test. TAVR, transcatheter aortic valve replacement; PAPI, pulmonary artery pulsatility index.

heart failure readmission following TAVR, given above hypothesized mechanisms and other previous studies (5,14).

Predictability of PAPI was superior to central venous pressure and pulmonary artery pulsatility alone. Central venous pressure is highly dependent on the hemodynamics. On the contrary, PAPI indicates patient's own right heart function, i.e., how the right heart can make pulmonary artery pulse per unit of preload, which is relatively independent on the hemodynamics.

One of the strengths of this study is the use of PAPI instead of echocardiographic parameters. PAPI does not require expert technique. One of the limitations to obtain PAPI data is a requirement of invasive right heart catheterization. However, such a procedure would be generally essential and routinely performed to assess hemodynamics status and construct therapeutic strategy before TAVR.

Future perspective

One of the purposes of TAVR is improvement in exercise capacity and quality of life. Impact of lower PAPI on these outcomes remains the next concern. Also, implication and methodology of aggressive intervention to lower PAPI is the future concern (13). Aggressive diuretic therapy before TAVR would rather reduce forward flow to left ventricle and deteriorate hemodynamics. Post-TAVR anti-pulmonary hypertension agents might be one of the promising tools to unload right ventricle.

Limitations

This is a retrospective study including a moderate sample size, although we completed comprehensive invasive hemodynamics data before TAVR. We could not follow PAPI because we do not routinely perform right heart catheterization following TAVR as other institutions. Also, we do not have comprehensive echocardiographic data associating with right ventricular function such as tricuspid annular plane excursion, and could not compare the prognostic impact between PAPI and echocardiography parameters. In the multivariate analyses, we might have ignored several potential confounders.

Conclusions

Baseline impaired right ventricular function, indicated by lower PAPI below 2.1, was associated with the occurrence of heart failure following TAVR. Further studies are warranted to clarify the detailed mechanism of our findings and prognostic implication of aggressive intervention to PAPI.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://cdt.amegroups.com/article/view/10.21037/cdt-21-682/rc

Data Sharing Statement: Available at https://cdt.amegroups.com/article/view/10.21037/cdt-21-682/dss

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://cdt.amegroups.com/article/view/10.21037/cdt-21-682/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consents were obtained from all participants before the listing. Institutional review board of University of Toyama approved the study protocol (R2015154, April 11 2016). This study was conducted according to the Declaration of Helsinki (as revised in 2013).

References

1. Hamm CW, Arsalan M, Mack MJ. The future of transcatheter aortic valve implantation. Eur Heart J 2016;37:803-10.
2. Baumgartner H, Falk V, Bax JJ, et al. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J 2017;38:2739-91.
3. Mack MJ, Leon MB, Thourani VH, et al. Transcatheter Aortic-Valve Replacement with a Balloon-Expandable Valve in Low-Risk Patients. N Engl J Med 2019;380:1695-705.
4. Koifman E, Didier R, Patel N, et al. Impact of right ventricular function on outcome of severe aortic stenosis patients undergoing transcatheter aortic valve replacement. Am Heart J 2017;184:141-7.
5. Testa L, Latib A, De Marco F, et al. The failing right heart: implications and evolution in high-risk patients undergoing transcatheter aortic valve implantation. EuroIntervention 2016;12:1542-9.
6. Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010;23:685-713; quiz 786-8.
7. Lim HS, Gustafsson F. Pulmonary artery pulsatility index: physiological basis and clinical application. Eur J Heart Fail 2020;22:32-8.
8. Kang G, Ha R, Banerjee D. Pulmonary artery pulsatility index predicts right ventricular failure after left ventricular assist device implantation. J Heart Lung Transplant 2016;35:67-73.
9. Osteresch R, Diehl K, Schmucker J, et al. Prognostic Impact of the Pulmonary Artery Pulsatility Index in Patients with Chronic Heart Failure and Severe Mitral Regurgitation Undergoing Percutaneous Edge-to-Edge Repair. Cardiology 2021;146:74-84.
10. Galli E, Guirette Y, Feneon D, et al. Prevalence and prognostic value of right ventricular dysfunction in severe aortic stenosis. Eur Heart J Cardiovasc Imaging 2015;16:531-8.
11. Santamore WP, Dell'Italia LJ. Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. Prog Cardiovasc Dis

1998;40:289-308.
12. Kempny A, Diller GP, Kaleschke G, et al. Impact of transcatheter aortic valve implantation or surgical aortic valve replacement on right ventricular function. Heart 2012;98:1299-304.
13. Poch F, Thalmann R, Olbrich I, et al. Changes of Right Ventricular Function After Transcatheter Aortic Valve Replacement and Association With Outcomes. J Card Fail 2021;27:1337-44.
14. Lindman BR, Maniar HS, Jaber WA, et al. Effect of tricuspid regurgitation and the right heart on survival after transcatheter aortic valve replacement: insights from the Placement of Aortic Transcatheter Valves II inoperable cohort. Circ Cardiovasc Interv 2015;8:e002073.

Cite this article as: Oshima A, Imamura T, Tanaka S, Onoda H, Ushijima R, Sobajima M, Fukuda N, Ueno H, Kinugawa K. Prognostic implication of pulmonary artery pulsatility index before transcatheter aortic valve replacement. Cardiovasc Diagn Ther 2022;12(2):188-195. doi: 10.21037/cdt-21-682

