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Prognostic Role of Right Ventricular-Pulmonary Artery Coupling Assessed by TAPSE/PASP Ratio in Patients With Acute Heart Failure

Youngnam Bok (iD), MD, **Ji-Yeon Kim** (iD), MD, and **Jae-Hyeong Park** (iD), MD, PhD

Department of Cardiology in Internal Medicine, Chungnam National University Hospital, Chungnam National University College of Medicine, Daejeon, Korea

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Address for Correspondence:
Jae-Hyeong Park, MD, PhD
Department of Cardiology in Internal Medicine, Chungnam National University Hospital, Chungnam National University College of Medicine, 282 Munhwa-ro, Jung-gu, Daejeon 35015, Korea.
Email: jaehpark@cnu.ac.kr

ABSTRACT

BACKGROUND: Right ventricular (RV) dysfunction is a significant risk of major adverse cardiac events in patients with acute heart failure (AHF). In this study, we evaluated RV-pulmonary artery (PA) coupling, assessed by tricuspid annular plane systolic excursion (TAPSE)/pulmonary artery systolic pressure (PASP) and assessed its prognostic significance, in AHF patients.

METHODS: We measured the TAPSE/PASP ratio and analyzed its correlations with other echocardiographic parameters. Additionally, we assessed its prognostic role in AHF patients.

RESULTS: A total of 1147 patients were included in the analysis (575 men, aged 70.81 ± 13.56 years). TAPSE/PASP ratio exhibited significant correlations with left ventricular (LV) ejection fraction($r = 0.243$, $p < 0.001$), left atrial (LA) diameter($r = -0.320$, $p < 0.001$), left atrial global longitudinal strain (LAGLS, $r = 0.496$, $p < 0.001$), mitral E/E′ ratio($r = -0.337$, $p < 0.001$), and right ventricular fractional area change (RVFAC, $r = 0.496$, $p < 0.001$). During the median follow-up duration of 29.0 months, a total of 387 patients (33.7%) died. In the univariate analysis, PASP, TAPSE, and TAPSE/PASP ratio were significant predictors of mortality. After the multivariate analysis, TAPSE/PASP ratio remained a statistically significant parameter for all-cause mortality (hazard ratio [HR], 0.453; $p = 0.037$) after adjusting for other parameters. In the receiver operating curve analysis, the optimal cut-off level of TAPSE/PASP ratio for predicting mortality was 0.33 (area under the curve = 0.576, $p < 0.001$), with a sensitivity of 65% and a specificity of 47%. TAPSE/PASP ratio < 0.33 was associated with an increased risk of mortality after adjusting for other variables (HR, 1.306; $p = 0.025$).

CONCLUSIONS: In AHF patients, TAPSE/PASP ratio demonstrated significant associations with RVFAC, LA diameter and LAGLS. Moreover, a decreased TAPSE/PASP ratio < 0.33 was identified as a poor prognostic factor for mortality.

INTRODUCTION

The coupling between the right ventricle (RV) and the pulmonary artery (PA) reflects the ability of the RV to adapt to the pulmonary vascular load and maintain sufficient RV cardiac output.[1] This RV-PA coupling is typically assessed through invasive right heart catheterization (RHC). Unlike the systemic circulation, the pulmonary circulation receives the same cardiac output (CO) at a much lower pressure. The pressure-volume loop of the RV exhibits distinct characteristics compared to the left ventricle (LV), with a trapezoidal or triangular shape and differing amplitude of pressure change.[2] In a healthy individual, the slope of the RV end-systolic pressure-volume relationship, known as end-systolic elastance (Ees), should be equivalent to the effective arterial elastance (Ea), resulting in an Ees/Ea ratio greater than 1.0.[3]

Patients with acute heart failure (AHF) may experience complications such as pulmonary hypertension (PH), which can contribute to RV dysfunction.[4] However, over time, PH patients may progress to RV decompensation, characterized by a decline in RV Ees relative to Ea, RV dilation to sustain stroke volume (SV), and a decrease in RV ejection fraction. RV dysfunction in these patients represents a significant risk factor for major adverse cardiac events.[5][6]

While the invasive measurement of Ees/Ea using pressure-volume loops is costly and requires hospitalization, echocardiographic assessment of RV-PA coupling offers a non-invasive and convenient alternative. Tricuspid annular plane systolic excursion (TAPSE) over pulmonary artery systolic pressure (PASP), measured through Doppler echocardiography, serves as a surrogate marker and an independent echocardiographic predictor in patients with PH.[7-9] However, the role of RV-PA coupling assessed by the TAPSE/PASP ratio remains understudied in patients with AHF. Therefore, our study aimed to investigate the prognostic significance of RV-PA coupling, as assessed by the TAPSE/PASP ratio, in patients with AHF.

METHODS

Study population

We included all consecutive patients with AHF who were admitted to Chungnam National University Hospital in Daejeon, Korea, between January 2009 and December 2016. AHF was diagnosed based on symptoms, elevated levels of N-terminal pro-B-type natriuretic peptide, and evidence of either pulmonary edema or objective findings of structural heart disease. Patients with severe valvular disease requiring surgery or those with acute coronary syndrome were excluded from the study. The study protocol received approval from the Institutional Review Board (IRB) of the hospital (IRB No. 2017-04-005). Due to the retrospective nature of the study, the IRB waived the requirement for informed consent from the participants. The research was conducted in accordance with the principles outlined in the Helsinki Declaration.

Study variables and definitions

Baseline demographic data and past medical history were extracted from the patients' medical records. Body mass index (BMI) was calculated using the recorded height and weight measurements. We used the blood test results on the day of admission. Hypertension was defined as the use of antihypertensive medication for a duration of more than 6 months. Additionally, patients who had a diagnosis of hypertension and were solely on lifestyle modification treatment were also classified as hypertensive.

Patients with diabetes mellitus (DM) were identified as those who were actively receiving treatment with oral hypoglycemic agents or insulin. Furthermore, individuals with an abnormal fasting glucose level ($\geq$ 126 mg/dL) or an abnormal 2-hour postprandial glucose level ($\geq$ 200 mg/dL) who were being treated with dietary modification only were also diagnosed with DM. The presence of atrial fibrillation (AF) was determined by documentation of AF on electrocardiograms or by a previous diagnosis of AF using the International Classification of Diseases 10th Revision (ICD-10) code I48. Ischemic heart disease (IHD) was ascertained if patients had a history of coronary artery intervention and a corresponding diagnosis of IHD using the ICD-10 codes I20, I21, and I25. Based on the left ventricular ejection fraction (LVEF) derived from echocardiographic findings during AHF admission, patients were categorized into 3 groups: heart failure with reduced ejection fraction (HFrEF) if LVEF was $\leq$ 40%, heart failure with mildly reduced ejection fraction (HFmrEF) if 40% < LVEF < 50%, and heart failure with preserved ejection fraction (HFpEF) if LVEF was $\geq$ 50%.

Echocardiographic measurement

All echocardiographic examinations were performed with echocardiographic machines and a 2.5 MHz probe using standard echocardiographic techniques, including M-mode, 2-dimensional, and Doppler modalities, as recommended by the American Society of Echocardiography.[10] We selected the most recent echocardiogram if there were multiple echocardiograms during admission, and the mean time

between the admission and the echocardiographic examination was 2.5 ± 4.9 days.

LV end-diastolic and end-systolic volumes were calculated from the apical 4- and 2-chamber views using the 2-dimensional Simpson's method, and LVEF was calculated from these values. Mitral inflow velocities (mitral E and A velocities, E/A ratio, deceleration time) were measured by pulsed-wave Doppler at the point of mitral valve coaptation, and mitral annular velocities (mitral annular E′ velocity) were estimated by the tissue Doppler of the mitral annular septum. Anteroposterior LA diameter was measured from the parasternal long-axis view. PASP was estimated from the peak tricuspid regurgitation jet velocity (TR Vmax) derived from continuous wave Doppler, and right atrial (RA) pressure was estimated from the size and collapsibility of the inferior vena cava. Right ventricular fractional area change (RVFAC), one of the global parameters of RV systolic function, was calculated from the areas of RV with RV-focused apical 4-chamber view. TAPSE was measured as the length between the end-diastolic and peak systolic points of the lateral tricuspid annulus.

We measured left atrial (LA) strain from the stored echocardiographic images using TomTec-Arena version 4.6 (TomTec, Munich, Germany), a vendor-independent strain measurement software.[11] The software automatically tracked speckles along the endocardial border and myocardium throughout the cardiac cycle after manually tracing the endocardial border on the end-systolic frame in the selected image. The left atrial global longitudinal strain (LAGLS) was defined as the first peak of positive deflection demonstrating LA reservoir function, using the R-R gating as the zero reference point. The LAGLS was calculated as the mean of the four segments of each apical view, averaged from the global longitudinal strain values obtained from the apical 4- and 2-chamber views. Due to poor delineation and contamination of the pulmonary veins, the roof of the LA was not analyzed. All LAGLS values were measured on a single cardiac cycle and all LAGLS values were calculated independently by an echocardiography specialist who was blinded to the clinical data.

Statistical analyses

Continuous variables are presented as means ± standard deviations, categorical variables as frequencies. For comparisons between groups, we used the Student's t-test for continuous variables and the χ^2 test for categorical variables. Pearson's correlation coefficient was used to calculate the correlation between TAPSE/PASP and other echocardiographic variables. We used multivariate Cox proportional hazards

analysis to determine the independent predictors of all-cause death and included all significant variables in the univariate analysis as covariates in the multivariate analysis because we had a sufficient number of death events in our study. However, in the multivariate analysis, we excluded variables that had multicollinearity with other variables. SPSS version 25 (IBM Corp., Armonk, NY, USA) was used for data analysis. A 2-tailed p-value of < 0.05 was considered statistically significant.

RESULTS

Patients characteristics

After screening all patients with AHF admitted to our hospital, we analyzed a total of 1,147 patients (575 men, aged 70.81 ± 13.56 years) after excluding patients with exclusion criteria. Baseline characteristics are summarized in **Table 1**. Hypertension was the most common associated cardiovascular risk factor (56.5%), and 385 patients (33.6%) had DM. The mean LVEF was 39.0 ± 15.4%, and 638 (55.6%), 175 (15.3%) and 334 (29%) patients were classified as having HFrEF, HFmrEF and HFpEF, respectively. The mean value of TAPSE was 14.8 ± 4.7 mm, PASP was 42.3 ± 13.3 mmHg, and TAPSE/PASP ratio was 0.37 ± 0.17. TAPSE/PASP ratio showed significant correlations with LVEF (r = 0.243, p < 0.001), LA diameter (r = −0.320, p < 0.001), LAGLS (r = 0.496, p < 0.001), mitral E/E′ ratio (r = −0.337, p < 0.001), and RVFAC (r = 0.496, p < 0.001).

Patients with a decreased TAPSE/PASP ratio (< 0.33) exhibited several notable differences compared to those with a preserved TAPSE/PASP ratio (**Table 1**). Specifically, the decreased TAPSE/PASP ratio group had a significantly lower BMI, lower systolic blood pressure, and higher heart rate. AF was also more frequently observed in patients with a decreased TAPSE/PASP ratio. Furthermore, the decreased TAPSE/PASP ratio group demonstrated significantly higher LV dimensions and volumes, lower LVEF, increased LA diameter, and decreased LAGLS. PASP was notably elevated in the decreased TAPSE/PASP ratio group. Moreover, HFrEF was more prevalent in the decreased TAPSE/PASP ratio group.

All-cause death and its determinants

A total of 387 patients (33.7%) died during the median follow-up of 29.0 months. The comparison of variables by death is shown in **Supplementary Table 1**. Patients who died were older (76.4 ± 10.6 years old vs. 68.0 ± 14.0 years old, p < 0.001), had a lower BMI (21.9 ± 3.6 kg/m² vs. 23.8 ± 4.4 kg/m², p < 0.001), and a higher proportion of patients with severe symptoms of New York Heart Association (NYHA) functional class IV (12.7%

Table 1. Baseline characteristics according to tricuspid annular plane systolic excursion/pulmonary artery systolic pressure ratio (> 0.33)

Variable	Total (n = 1,147)	TAPSE/PASP ≥ 0.33 (n = 551)	TAPSE/PASP < 0.33 (n = 596)	p-value
Baseline clinical characteristics				
Male sex	575 (50.1)	280 (50.8)	295 (49.5)	0.679
Age (year)	70.8 ± 13.6	70.8 ± 13.7	70.9 ± 13.4	0.877
BMI (kg/m²)	23.2 ± 4.2	23.5 ± 3.8	22.9 ± 4.5	0.017
NYHA Fc IV	89 (8.0)	36 (6.8)	53 (9.1)	0.184
Physical examination				
SBP (mmHg)	123.2 ± 23.6	124.7 ± 23.0	121.8 ± 24.0	0.040
DBP (mmHg)	72.3 ± 14.0	72.8 ± 13.8	71.9 ± 14.2	0.247
Heart rate (/min)	84.4 ± 22.3	78.8 ± 18.8	89.6 ± 24.0	< 0.001
Past medical history				
Atrial fibrillation	359 (31.5)	121 (22.2)	238 (40.2)	< 0.001
Hypertension	648 (56.5)	314 (57.0)	334 (56.0)	0.766
Diabetes mellitus	385 (33.6)	182 (33.0)	203 (34.1)	0.754
Ischemic heart disease	395 (34.4)	208 (37.7)	187 (31.4)	0.025
Laboratory findings				
Hemoglobin (g/dL)	12.3 ± 2.3	12.3 ± 2.3	12.2 ± 2.4	0.497
BUN (mg/dL)	24.1 ± 14.4	21.7 ± 12.2	26.3 ± 15.8	< 0.001
Creatinine (mg/dL)	1.35 ± 1.43	1.30 ± 1.71	1.40 ± 1.10	0.227
Total cholesterol (mg/dL)	151.3 ± 44.2	156.3 ± 44.5	146.7 ± 43.5	< 0.001
Echocardiographic findings				
LVEDD (mm)	51.5 ± 9.2	50.6 ± 8.8	52.4 ± 9.4	0.001
LVESD (mm)	40.7 ± 11.5	38.9 ± 11.0	42.3 ± 11.6	< 0.001
LVEDV (mL)	115.5 ± 56.5	109.2 ± 52.1	121.1 ± 59.7	< 0.001
LVESV (mL)	75.3 ± 50.5	67.4 ± 45.7	82.5 ± 53.7	< 0.001
LVEF (%)	39.0 ± 15.4	42.3 ± 14.8	36.0 ± 15.4	< 0.001
LA diameter (mm)	43.7 ± 8.7	41.2 ± 7.5	46.2 ± 8.9	< 0.001
LAGLS (%)	12.6 ± 9.3	16.5 ± 10.4	8.9 ± 6.3	< 0.001
Mitral E-velocity (m/sec)	0.89 ± 0.35	0.76 ± 0.29	1.01 ± 0.35	< 0.001
Mitral A-velocity (m/sec)	0.78 ± 0.30	0.84 ± 0.29	0.70 ± 0.29	< 0.001
E′ velocity (cm/sec)	5.10 ± 2.07	5.21 ± 2.03	4.99 ± 2.10	< 0.001
E/E′ ratio	18.4 ± 9.3	15.6 ± 7.1	21.7 ± 10.6	0.144
PASP (mmHg)	42.3 ± 13.3	34.7 ± 7.9	49.4 ± 13.3	< 0.001
RVFAC (%)	44.2 ± 12.2	49.1 ± 10.8	39.8 ± 11.7	< 0.001
TAPSE (mm)	14.8 ± 4.7	17.7 ± 4.0	12.1 ± 3.6	< 0.001
Definition of HF				< 0.001
HFrEF	638 (55.6)	252 (45.7)	386 (64.8)	
HFmrEF	175 (15.3)	110 (20.0)	65 (10.9)	
HFpEF	334 (29.1)	189 (34.3)	145 (24.3)	
Medication at discharge				
RAS-inhibitor	865 (75.5)	408 (74.0)	457 (76.8)	0.303
Beta-blocker	919 (80.2)	429 (77.9)	490 (82.4)	0.064
MRA	631 (55.1)	280 (50.8)	351 (59.0)	0.006

Values are presented as mean ± standard deviation or number (%).
BMI: body mass index, BUN: blood urea nitrogen, DBP: diastolic blood pressure, HF: heart failure, HFmrEF: heart failure with mildly reduced ejection fraction, HFpEF: heart failure with preserved ejection fraction, HFrEF: heart failure with reduced ejection fraction, LA: left atrium, LAGLS: left atrial global longitudinal strain, LVEDD: left ventricular end-diastolic dimension, LVEDV: left ventricular end-diastolic volume, LVEF: left ventricular ejection fraction, LVESD: left ventricular end-systolic dimension, LVESV: left ventricular end-systolic volume, MRA: mineralocorticoid receptor antagonist, NYHA Fc: New York Heart Association functional class, PASP: pulmonary arterial systolic pressure, RAS: renin-angiotensin system, RVFAC: right ventricular fractional area change, SBP: systolic blood pressure, TAPSE: tricuspid annular plane systolic excursion.

vs. 5.6%, p < 0.001). The incidence of hypertension (61.2% vs. 54.1%, p = 0.023), DM (38.8% vs. 30.9%, p = 0.008), and IHD (39.3% vs. 32.0%, p = 0.015) was higher in the deceased group than in the survivors. Blood tests also showed higher BUN (28.6 ± 16.5 mg/dL vs. 21.8 ± 12.6 mg/dL, p < 0.001) and creatinine (1.58 ± 1.28 mg/dL vs. 1.24 ± 1.49 mg/dL, p < 0.001) in the death group. Total cholesterol was significantly lower in the deceased group (145.4 ± 42.5 mg/dL vs. 154.3 ± 44.8 mg/dL, p = 0.001).

Echocardiography showed that LV volumes were significantly lower in the death group, but LVEF did not differ between the two groups (39.5 ± 15.2% vs. 38.7 ± 15.5%, p = 0.415). In terms of LV diastolic parameters, LA diameter (44.6 ± 10.1 mm vs. 43.2 ± 7.9 mm, p = 0.022) and mitral E/E′ ratio (19.6 ± 9.3 vs. 17.9 ± 9.3, p = 0.030) were higher in the death group. While RV systolic function as assessed by TAPSE and RVFAC was similar in both groups, PASP was significantly higher in the death group (44.9

Table 2. Univariate analysis in the prediction of all-cause death within 5 years

Variable	HR	95% CI	p-value
Age (per 1 year)	1.058	1.047–1.068	< 0.001
Male sex	0.983	0.805–1.199	0.863
BMI (per 1 Kg/m²)	0.909	0.883–0.937	< 0.001
DBP (per 1 mmHg)	0.987	0.979–0.994	0.001
NYHA Fc IV	1.796	1.327–2.432	< 0.001
Hypertension	1.338	1.090–1.642	0.005
Diabetes mellitus	1.401	1.142–1.720	0.001
Ischemic heart disease	1.344	1.088–1.661	0.006
Creatinine (per 1 mg/dL)	1.052	1.019–1.085	0.001
Total cholesterol (per 1 mg/dL)	0.995	0.993–0.998	< 0.001
LVEDD (per 1 mm)	0.985	0.974–0.997	0.011
LVEDV (per 1 mL)	0.998	0.996–1.000	0.063
LA diameter (per 1 mm)	1.020	1.008–1.031	0.001
LAGLS (per 1%)	0.979	0.968–0.991	0.001
E/E′ ratio (per 1)	1.017	1.003–1.030	0.014
PASP (per 1mmHg)	1.019	1.012–1.026	< 0.001
TAPSE (per 1mm increase)	0.977	0.957–0.999	0.037
TAPSE/PASP ratio	0.219	0.118–0.406	< 0.001
Use of RAS-inhibitors at discharge	0.690	0.554–0.861	0.001
Use of beta-blockers at discharge	0.728	0.578–0.916	0.007

BMI: body mass index, CI: confidence interval, DBP: diastolic blood pressure, HR: hazard ratio, NYHA Fc: New York Heart Association functional class, LA: left atrium, LAGLS: left atrial global longitudinal strain, LVEDD: left ventricular end-diastolic dimension, LVEDV: left ventricular end-diastolic volume, PASP: pulmonary artery systolic pressure, RAS: renin-angiotensin system, TAPSE: tricuspid annular plane systolic excursion.

Table 3. Multivariate analysis in the prediction of all-cause death within 5 years

Variable	HR	95% CI	p-value
Analysis A			
Age (per 1 year)	1.058	1.044–1.072	< 0.001
BMI (per 1 Kg/m²)	0.961	0.929–0.993	0.019
DBP (per 1 mmHg)	0.993	0.984–1.002	0.137
NYHA Fc IV	1.276	0.889–1.832	0.186
Hypertension	0.983	0.775–1.247	0.888
Diabetes mellitus	1.496	1.182–1.892	0.001
Ischemic heart disease	1.122	0.890–1.413	0.329
Creatinine (per 1 mg/dL)	1.110	1.059–1.163	< 0.001
Total cholesterol (per 1 mg/dL)	0.999	0.996–1.002	0.416
LVEDV (per 1 mL)	1.002	1.000–1.005	0.049
LA diameter (per 1 mm)	1.018	1.004–1.031	0.009
Use of RAS-inhibitor at discharge	0.761	0.589–0.983	0.036
Use of beta-blocker at discharge	0.884	0.678–1.152	0.362
TAPSE/PASP ratio	0.453	0.216–0.952	0.037
Analysis B			
Age (per 1 year)	1.058	1.044–1.072	< 0.001
BMI (per 1 Kg/m²)	0.960	0.928–0.992	0.016
DBP (per 1 mmHg)	0.993	0.985–1.002	0.149
NYHA Fc IV	1.280	0.891–1.838	0.182
Hypertension	0.976	0.768–1.239	0.839
Diabetes mellitus	1.507	1.191–1.907	0.001
Ischemic heart disease	1.127	0.894–1.420	0.311
Creatinine (per 1 mg/dL)	1.111	1.061–1.164	< 0.001
Total cholesterol (per 1 mg/dL)	0.999	0.996–1.002	0.406
LVEDV (per 1 mL)	1.002	1.000–1.005	0.045
LA diameter (per 1 mm)	1.018	1.005–1.031	0.008
Use of RAS-inhibitor at discharge	0.757	0.587–0.978	0.033
Use of beta-blocker at discharge	0.885	0.679–1.153	0.365
TAPSE/PASP ratio < 0.33	1.306	1.035–1.648	0.025

BMI: body mass index, CI: confidence interval, DBP: diastolic blood pressure, HR: hazard ratio, NYHA Fc: New York Heart Association functional class, LA: left atrium, LVEDV: left ventricular end-diastolic volume, PASP: pulmonary artery systolic pressure, RAS: renin-angiotensin system, TAPSE: tricuspid annular plane systolic excursion.
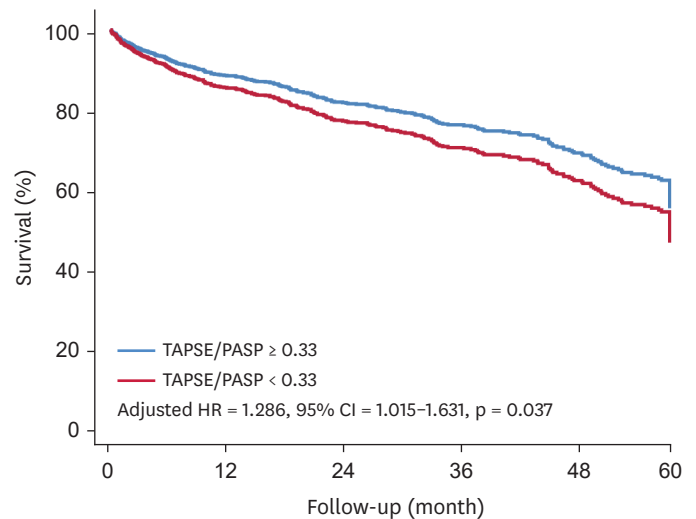
± 13.7 mmHg vs. 41.0 ± 12.9 mmHg, p < 0.001). Thus, TAPSE/ PASP ratio was significantly lower in the death group (0.33 ± 0.16 vs. 0.37 ± 0.17, p < 0.001).

In univariate analysis in the prediction of all-cause death (**Table 2**), TAPSE/PASP ratio (hazard ratio [HR], 0.219; p < 0.001) was a significant variable. Other variables included age, BMI, diastolic blood pressure, NYHA functional class IV, hypertension, DM, IHD, creatinine, total cholesterol, LV end-diastolic dimension, LA diameter, mitral E/E′ ratio, PASP, LAGLS, TAPSE, and use of renin-angiotensin system (RAS)-blockers and beta-blockers.

The results of the multivariate analysis, including the variables that were significant in the univariate analysis, were presented in **Table 3**. TAPSE/PASP ratio was a statistically significant parameter of all-cause mortality (HR, 0.450; p = 0.037) after the adjustment of significant variables in the univariate analysis including age, BMI, NYHA functional class, hypertension, DM, serum creatinine, total cholesterol, LV end-diastolic dimension, LA diameter, and use of RAS-inhibitor and beta-blocker. In the receiver operating curve analysis, the best cut-off level for predicting all-cause death is 0.33 (area under the curve = 0.576, p < 0.001), with a sensitivity of 65% and a specificity of 47%. TAPSE/PASP ratio < 0.33 was associated with increased all-cause mortality after adjustment of other variables (HR, 1.306; p = 0.025; **Figure 1**) after the multivariate analysis.

Figure 1. Survival curve according to tricuspid annular plane systolic excursion/ pulmonary arterial systolic pressure (TAPSE/PASP ratio). Patients with TAPSE/ PASP ratio ≥ 0.33 have better survival.
CI: confidence interval, HR: hazard ratio, PASP: pulmonary artery systolic pressure, TAPSE: tricuspid annular plane systolic excursion.

DISCUSSION

In this study, we found that TAPSE/PASP ratio showed significant correlations with LV systolic function as assessed by LVEF, LV diastolic function as estimated by LA diameter, LAGLS and mitral E/E′ ratio, and RV systolic function as measured by RVFAC. TAPSE/PASP ratio was statistically significant for all-cause death in AHF patients, and TAPSE/PASP ratio < 0.33 was a poor prognostic marker.

TAPSE/PASP ratio is an echocardiographic marker of RV-PA coupling. In the study by Guazzi et al.[12] showed that the echocardiographically assessed TAPSE/PASP ratio was a poor prognostic marker in heart failure (HF) patients. RV dysfunction can be found in patients with HF. Approximately 50% of patients in the study had evidence of RV dysfunction, including patients with advanced HFpEF.[13] A decrease in LA compliance from systolic and diastolic dysfunction in patients with left heart diseases increases the LA pressure, which in turn elevates the pulmonary artery pressure via backward transmission.[4]

In our study, TAPSE/PASP ratio showed significant correlation with echocardiographic markers of LV diastolic function and LA function. Increased LV diastolic pressure and worsening LV diastolic function may worsen LA function. These findings correlate with previous studies.[14][15] TAPSE/PASP ratio had a significant negative correlation with baseline LA v-wave pressure (r = −0.324, p = 0.047) in patients with severe mitral regurgitation undergoing MitraClip procedure.[14] Also, the change of TAPSE/PASP ratio correlated with the change of LA volume index (r = 0.238, p = 0.045).[15] These findings suggest that increased LA stiffness is associated with worse RV-PA coupling as assessed by TAPSE/PASP ratio.

We found that TAPSE/PASP ratio, especially < 0.33, was a significant prognostic factor in patients with AHF. Our results are consistent with previous studies which have recently reported prognostic value of TAPSE/PASP ratio in patients with HF or pulmonary hypertension.[7-9][14] Patients with a TAPSE/PASP ratio < 0.31 had a significantly worse overall survival than those with a higher TAPSE/PASP ratio (p = 0.003) in patients with severe pulmonary hypertension.[9] In patients with severe mitral regurgitation, TAPSE/PASP was an independent prognostic marker for the primary combined endpoint, and TAPSE/PASP ratio ≤ 0.35 was a poor prognostic marker.[14]

This study has several limitations. First, this is a retrospective study from a tertiary care hospital. Second, we assessed the presence of all-cause death using medical records or data from the Korean National Insurance Service. Thus, we did not know the exact cause of death for patients without regular medical follow-up. In the future, a prospective study with a large number of patients and a well-controlled design will confirm the association between TAPSE/PASP ratio and its prognostic significance in AHF patients.

In AHF patients, TAPSE/PASP ratio was significantly associated with RVFAC, LA volume index and LAGLS. Also, decreased TAPSE/PASP ratio < 0.33 was a poor prognostic factor of all-cause mortality. Thus, this ratio can be used in the prediction of poor clinical outcomes in patients with AHF.

ORCID iDs
Youngnam Bok 🆔
https://orcid.org/0000-0002-6341-6164
Ji-Yeon Kim 🆔
https://orcid.org/0009-0009-0173-1968
Jae-Hyeong Park 🆔
https://orcid.org/0000-0001-7035-286X

SUPPLEMENTARY MATERIAL

Supplementary Table 1
Baseline characteristics according to all-cause mortality

Click here to view

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