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Prognostic Impact of the Tricuspid Annular Plane Systolic Excursion/Pulmonary Arterial Systolic Pressure Ratio in Acute Pulmonary Embolism

ABSTRACT

Background: Currently available risk stratification models for acute pulmonary embolism (PE) include hemodynamic status, cardiac biomarkers, right ventricle (RV) dysfunction on imaging, and clinical scores. Focusing on the length–tension relationship of the ventricle might have a superior predictive capability over RV dysfunction in terms of mortality and classification of patients with acute PE. In this study, our hypothesis suggests that the tricuspid annular plane systolic excursion (TAPSE)/systolic pulmonary artery pressure (sPAP) ratio has superior predictive capability for in-hospital mortality in patients with acute PE compared to TAPSE or sPAP as distinct measures.

Methods: This single-center study comprised retrospectively evaluated 703 patients referred to our tertiary cardiovascular center with acute PE. We divided patients into quartiles based on the TAPSE/sPAP ratio. Different models were developed to quantify the predictive relationship between in-hospital death and echocardiographic measurements. A base model was created with variables including risk status and RV/LV ratio >1. Then, to evaluate the predictive contribution of each measurement; TAPSE/sPAP, TAPSE, and sPAP were sequentially added to the base model. After that, the performance of each model was evaluated.

Results: Predictive and discriminative power was the highest in model containing TAPSE/sPAP. There was still a significant inverse association between TAPSE/sPAP and the risk of in-hospital death even after adjusting for risk status and RV/LV ratio >1. Receiver operating characteristic curve analysis for TAPSE/sPAP revealed the best cut-off value as 0.34.

Conclusion: The outcomes of our study reveal that the ratio of TAPSE/sPAP serves as a more potent predictor of mortality than either of the 2 measurements taken separately. The interpretation and utilization of the TAPSE/sPAP cut-off value in acute PE can assist in identifying patients at risk of deterioration and guide the consideration of more intensive treatment options across all risk groups.

Keywords: Echocardiography, pulmonary embolism, right ventricular function, risk stratification

INTRODUCTION

Pulmonary embolism (PE) ranks as the third most prevalent acute cardiovascular syndrome worldwide, following myocardial infarction and stroke.[1] In the presence of acute pressure overload and right ventricle (RV) dysfunction, PE can lead to mortality. Currently available risk stratification models using clinical scores, biomarkers, computed tomography (CT), and transthoracic echocardiogram (TTE) have been utilized in risk-based treatment strategies in acute PE.[2-7] These methods not only classify patients into risk groups but also help determine who can benefit from advanced therapeutic options. Right ventricle dysfunction in PE can simply be assessed with TTE by tricuspid annular plane systolic excursion (TAPSE), tissue Doppler, and RV/left ventricle (LV) diameter in conjunction with estimated systolic pulmonary arterial pressure (sPAP). Although this assessment holds significant value in the evaluation of patients with acute PE, its positive predictive value for PE-related death is remarkably low.[8] Hence, there appears to be a need for supplementary prognostic markers. The introduction of the TAPSE/sPAP ratio provided a non-invasive and indirect means of measuring RV contractile function

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and RV–pulmonary arterial (PA) coupling. Its significance as a crucial clinical and prognostic parameter has been validated in patients with heart failure, as well as in those diagnosed with pulmonary arterial hypertension (PAH).[9-11] In this study, we propose that the TAPSE/sPAP ratio has superior predictive capability for in-hospital mortality in patients with acute PE compared to TAPSE or sPAP as distinct measures.

METHODS

Design

This single-center study retrospectively evaluated 703 patients (median age of 65 years, interquartile range 50-74.3, 57.2% female) referred to our tertiary cardiovascular center with a confirmed diagnosis of acute PE from August 2012 to April 2023. Patients aged <18 years, those with chronic thromboembolic pulmonary hypertension, onset of symptoms >14 days, or those receiving extracorporeal membrane oxygenation were excluded from the study. The systematic work-up for an initial diagnosis of acute PE and risk stratification included multidetector contrast-enhanced CT angiography, TTE assessments, PE severity indexes, and biomarker evaluation, based on criteria recommended by the ESC/ERS PE guideline.[1] Acute PE was confirmed by the presence of a thrombus located in at least 1 main or lobar pulmonary artery on CT imaging. All-cause in-hospital mortality, defined as death before discharge, was studied as the primary outcome measure. Local Ethics Committee approval was obtained for the study (Decision date: December 12, 2023, Decision number: 2023/19/752), and the study complied with the Declaration of Helsinki.

Chest CT Pulmonary Angiography

Computed tomography images were acquired using 64-slice helical CT angiography (Toshiba Aquilion 64™, Toshiba Medical Systems Corp., Tokyo, Japan). A validated CT score for pulmonary arterial occlusion proposed by Qanadli et al[12] [Qanadli score (QS)], RV/LV ratio, RV diameter, right atrial to left atrial diameter ratio (RA/LA ratio), and main, left, and right pulmonary arterial diameters were measured from CT images. Pulmonary infarction was defined as a peripheral wedge-shaped pulmonary consolidation in a hypoperfused segment of the lung.

Echocardiography

Echocardiography was performed in all patients on the first day of admission using a Vivid-7 echocardiography device (GE Vingmed Ultrasound AS, Horten, Norway). Patients were excluded if the image quality was too poor or if echocardiography could not be performed on the first day of admission. Tricuspid annular plane systolic excursion was obtained by positioning an M-mode cursor across the lateral tricuspid annulus and measuring the longitudinal motion of the annulus at peak systole in the standard apical 4-chamber view. The maximal tricuspid regurgitation velocity, obtained using continuous-wave Doppler in accordance with the modified Bernoulli equation, was used in conjunction with the estimated right atrial pressure, measured by the inferior vena cava diameter, to estimate sPAP. Right ventricular lateral annular systolic velocity was obtained using pulsed tissue Doppler, measured on the basal segment of the free wall side of the RV in an apical 4-chamber view. Left ventricular ejection fraction (LVEF) was calculated using the biplane Simpson's method. Septal flattening was qualitatively assessed in the emergency department, and the eccentricity index was used in cases with uncertainty. All measurements and assessments were made in accordance with the American Society of Echocardiography guidelines.[13]

Statistical Analysis

All statistical analyses were performed using R Studio version 4.3.1 (R Project, Vienna, Austria). Normally distributed continuous data were expressed as mean and SD values, whereas non-normally distributed data were expressed as medians and interquartile ranges. Categorical data were described as absolute and percentage values. The normality of the data was determined using histograms and the Shapiro–Wilk test. Patients were divided into quartiles by TAPSE/sPAP ratio. Regarding sample distribution, analysis of variance and Kruskal–Wallis tests were used for the comparison of continuous data groups according to TAPSE/sPAP quartiles, and Pearson χ^2 or Fisher's exact test was used for the comparison of categorical data groups. Group comparisons were assessed with post-hoc tests of Tukey and DSCF, whichever was appropriate. Different models were developed to quantify the predictive relationship between in-hospital death and echocardiographic measurements. To assess model complexity, the event per *df* rule of thumb suggests that the number of events per *df* should be at least 10 to avoid overfitting. We have taken this rule of thumb into consideration when creating models. A base model (model 1) was created with variables including risk status and RV/LV ratio >1. Then, to evaluate the predictive contribution of each measurement, TAPSE/sPAP (model 2), TAPSE (model 3), and sPAP (model 4) were sequentially added to the base model. After that, the performance (R^2 and area under the ROC curve (AUC)) of each model was evaluated. The model with the highest R^2 and AUC values was accepted as the

HIGHLIGHTS

- Although right ventricular dysfunction in pulmonary embolism (PE) can be assessed with a transthoracic echocardiogram by TAPSE, tissue Doppler, RV/LV diameter in conjunction with estimated systolic pulmonary arterial pressure (sPAP), its positive predictive value for PE-related death is remarkably low. Hence, there appears to be a need for supplementary prognostic markers.
- The results of this study indicate that the echocardiographic TAPSE/sPAP ratio is not only an independent predictor of in-hospital mortality in patients with acute PE but also exhibits a greater capability of prediction than TAPSE and sPAP measurements individually.
- The interpretation and utilization of the TAPSE/sPAP cut-off value in acute PE can assist in identifying patients at risk of deterioration and guide the consideration of more intensive treatment options across all risk groups.

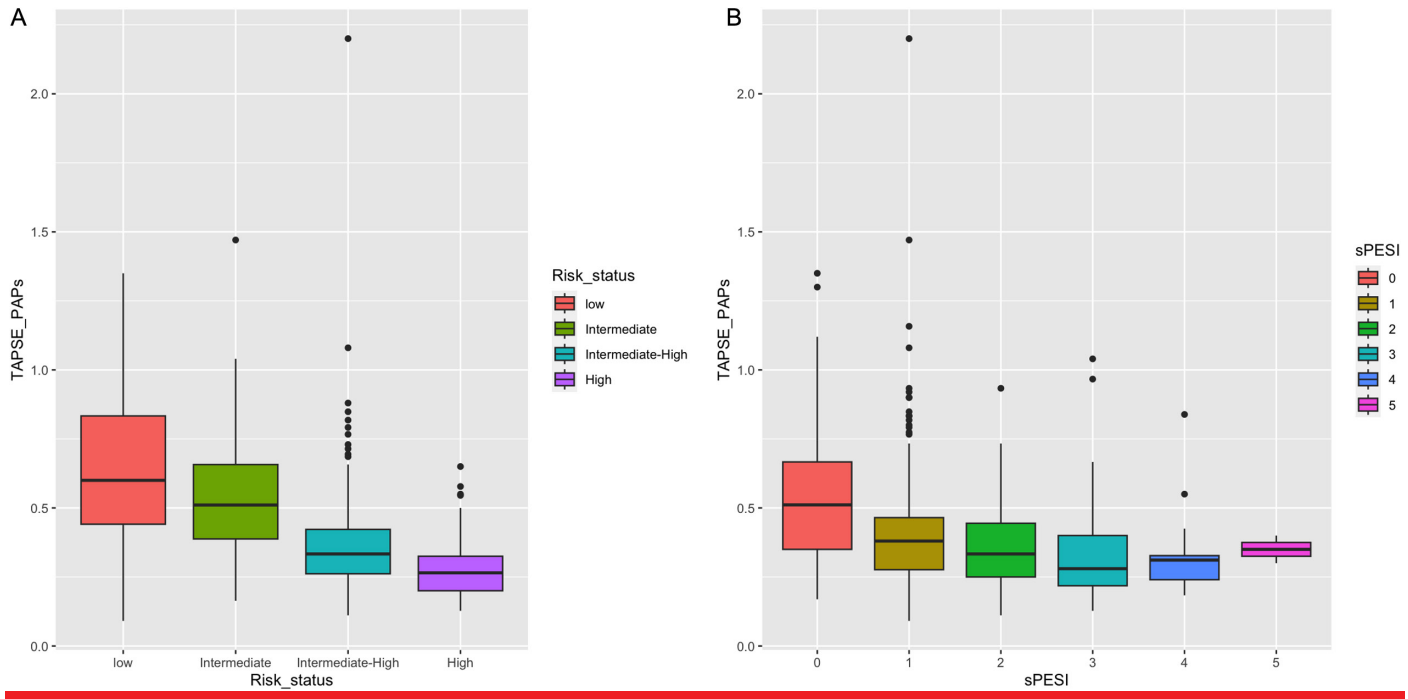

Figure 1. TAPSE/sPAP values according to risk status and sPESI class.

best predictive model. Binary logistic regression was used to quantify the relationship between the response variable (in-hospital death) and the predictors, and the findings were presented with an adjusted odds ratio and 95% CI. Continuous variables were included in the model using restricted cubic spline transformation. Therefore, the odds ratio represents the interquartile odds ratio. To investigate the best cut-off value for TAPSE/sPAP to predict in-hospital death, we also performed receiver operating characteristic (ROC) curve analysis. An AUC value of 0.5 indicates random chance, while an AUC value of 1.0 indicates perfect discriminatory power.

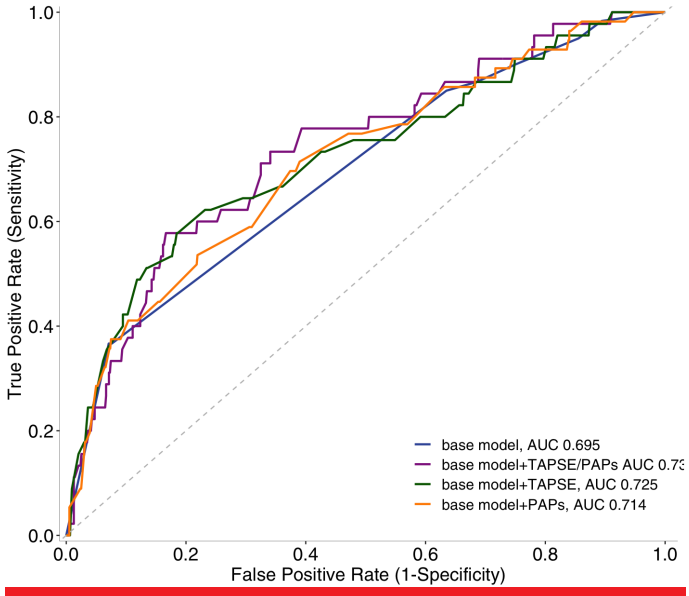

Figure 2. The AUC values obtained from ROC analysis for various models.

To find the best cut-off value for the echocardiographic measurements, we used the Youden index, which maximizes the difference between the true positive rate and the false positive rate. A *P* value of <.05 was considered the limit for statistical significance. During the preparation of this article, the authors did not use artificial intelligence-assisted technologies.

RESULTS

A total of 703 patients were included in the study (median age: 65, interquartile range: 50-74.3 years, 57.2% female). We divided patients into quartiles by TAPSE/sPAP ratio. The median TAPSE/sPAP values are 0.22, 0.32, 0.42, and 0.63 for Q1, Q2, Q3, and Q4, respectively. Baseline clinical characteristics are shown in Table 1 according to TAPSE/sPAP quartiles. Patients in Q1 had significantly lower blood pressure, oxygen saturation, and TAPSE and higher risk status, PESI and PESI Class, RV/LV ratio, and sPAP compared with Q2, Q3, and Q4. Two hundred eighty-one patients (39.9%) underwent catheter-directed treatments (CDT), with Q1 showing a higher preference for CDT compared to other quartiles. Ultrasound-assisted thrombolysis (USAT) was the primary CDT method, utilized in 224 patients (32%), while rheolytic thrombectomy followed as the second most employed CDT, with 57 patients (8.1%). In Q1, RV diameter, RV/LV ratio, and QS were higher, while RV function was decreased compared to other quartiles (Table 2). In addition, TAPSE/sPAP values are shown in Figure 1 according to risk status and sPESI class. Tricuspid annular plane systolic excursion/sPAP values decreased as risk status and sPESI increased (*P* values <.001 for both). In-hospital mortality was observed in a total of 60 patients (8.5%). In the deceased, TAPSE/sPAP [0.26 (0.20-0.33) vs. 0.38 (0.28-0.51), OR = 0.27, 95% CI: 0.15-0.52, *P* value <.001] and TAPSE [1.50 (1.20-1.80) vs. 1.90 (1.60-2.20), OR = 0.36, 95% CI:

Table 1. Baseline Characteristics According to TAPSE/sPAP Quartiles

Variables	Q1, n = 178	Q2, n = 174	Q3, n = 182	Q4, n = 169	*P*
Age (years)	67 (53, 76)	68 (52, 76)	62 (50, 71)	57 (44, 70)	<.001[b,c]
Male sex	69 (39%)	66 (38%)	76 (42%)	94 (56%)	.003
Syncope	74 (41.5%)	55 (31.6%)	50 (27.5%)	26 (15.4%)	<.001
Symptom duration, days	3 (1-7)	4 (2-7)	3 (2-6.25)	3 (2-5)	.526
HT	60 (38%)	72 (44%)	70 (41%)	51 (32%)	.2
CAD	19 (12%)	27 (17%)	12 (7.0%)	15 (9.5%)	.038
DM	27 (17%)	24 (15%)	35 (20%)	23 (15%)	.4
Smoking	11 (7.1%)	15 (9.7%)	17 (11%)	23 (16%)	.069
Acute DVT	98 (59%)	81 (49%)	104 (58%)	89 (55%)	.3
Previous VTE	13 (7.3%)	14 (8.1%)	21 (12%)	19 (11%)	.4
Malignancy	31 (17%)	24 (14%)	26 (14%)	21 (12%)	.6
SBP, mm Hg	112 (95-131)	120 (106-137)	126 (113-140)	130 (115-141)	<.001[a,b,c]
DBP, mm Hg	70 (60-85)	74 (65-84)	80 (70-87)	79 (66-87)	.002[b,c]
HR, beats/min	110 (102-123)	110 (96-120)	109 (92-119)	94 (82-111)	<.001[b,c]
SO_2, %	88 (84-90)	89 (85-92)	90 (86-93)	94 (91-96)	<.001[a,b,c]
Troponin, ng/mL	0.1 (0.05-0.3)	0.115 (0.05-0.3)	0.07 (0.03-0.2)	0.023 (0.007-0.1)	<.001[b,c]
NT-proBNP, pg/mL	1117 (111-2227)	756 (247-3112)	551 (136-1713)	195 (44.5-980)	.02[a,b,c]
D-Dimer, mg/L	9.97 (4.9-20)	8.26 (3.98-14.8)	7.6 (3.66-15)	4.21 (2.9-8.83)	<.001[c]
PESI	117 (98-142)	101 (81-123)	94 (74-118)	75 (60-97)	<.001[a,b,c]
PESI class					<.001
1	6 (3.8%)	13 (7.5%)	30 (16.5%)	62 (36.7%)	
2	15 (8.4%)	37 (21.2%)	45 (24.7%)	50 (29.6%)	
3	40 (22.5%)	45 (25.9%)	46 (25.3%)	27 (16%)	
4	43 (24.1%)	37 (21.3%)	30 (16.5%)	18 (10.6%)	
5	74 (41.5%)	42 (24.1%)	31 (17%)	12 (7.1%)	
sPESI	2 (1-3)	1 (1-2)	1 (1-2)	0 (0-1)	<.001[a,b,c]
Risk status					<.001
Low	8 (4.5%)	11 (6.3%)	19 (10%)	60 (36%)	
Low-inter	10 (5.6%)	15 (8.6%)	31 (17%)	57 (34%)	
Inter-high	121 (68%)	129 (74%)	124 (68%)	48 (28%)	
High	39 (22%)	19 (11%)	8 (4.4%)	4 (2.4%)	
t-PA	66 (37%)	54 (31%)	48 (26%)	25 (15%)	<.001
USAT (EKOS)	75 (42%)	64 (37%)	63 (35%)	22 (13%)	<.001
Rheolytic thrombectomy (AngioJet)	21 (11.8%)	17 (9.8%)	14 (7.7%)	5 (2.9%)	.013

CAD, coronary artery disease; DBP, diastolic blood pressure; DM, diabetes mellitus; DVT, deep vein thrombosis; EKOS, EkoSonic endovascular system; HR, heart rate; HT, hypertension; PESI, pulmonary embolism severity index; SBP, systolic blood pressure; SO_2, oxygen saturation; t-PA, tissue plasminogen activator; USAT, ultrasound accelerated thrombolysis; VTE, venous thromboembolism.
[a]Q1 vs. Q2.
[b]Q1 vs. Q3.
[c]Q1 vs. Q4.

0.23-0.56, *P* value <.001] were found to be significantly lower. Besides, sPAP [55 (50-65) vs. 50 (40-60), OR = 1.77, 95% CI: 1.23-2.54, *P* value = .002] was significantly higher.

In multiple regression analyses, the R^2 values were 0.094, 0.119, 0.113, and 0.096 for models 1, 2, 3, and 4, respectively. Area under the ROC curve values were 0.695, 0.738, 0.725, and 0.714 for models 1, 2, 3, and 4, respectively (Figure 2). When models were compared pairwise using the DeLong test, the AUC value of model 2 was significantly higher than the AUC values of the other models (*P* = .018). Therefore, the predictive and discriminative power was the highest for the model containing TAPSE/sPAP. The univariable and multiple regression analysis results are shown in Figure 3. More importantly, there was still a significant inverse association between TAPSE/sPAP and the risk of in-hospital death even after adjusting for risk status and RV/LV ratio >1 (change from 0.27 to 0.50, OR = 0.36, 95% CI: 0.20-0.64, *P* = .038) (Figure 4). According to ROC curve analysis, the best cut-off value based on the Youden index for TAPSE/sPAP was 0.34 (sensitivity 60%, specificity 80%, PPV 98%, and NPV 12%).

DISCUSSION

The present results show that the echocardiographic TAPSE/sPAP ratio is not only an independent predictor of in-hospital mortality in patients with acute PE but also exhibits a greater

Table 2. Echocardiographic and Tomographic Variables of the Study Population

Variables	Q1, n = 178	Q2, n = 174	Q3, n = 182	Q4, n = 169	*P*
RV/LV >1 (CT)	164 (93%)	153 (90%)	143 (80%)	46 (30%)	<.001
sPAP, mm Hg	65 (58-70)	55 (50-60)	46 (40-50)	35 (30-40)	<.001[a,b,c]
TAPSE, cm	1.4 (1.2-1.6)	1.8 (1.6-2.0)	2.0 (1.8-2.2)	2.3 (2.1-2.5)	<.001[a,b,c]
TAPSE/sPAP	0.22 (0.19-0.25)	0.32 (0.30-0.34)	0.42 (0.40-0.45)	0.63 (0.55-0.83)	<.001[a,b,c]
RV TDI, cm/s	9 (8-11)	10 (9-12)	12 (10.1-14)	14 (13-15.8)	<.001[a,b,c]
Flattening of the septum	(34.1%)	(30.8%)	(27.1%)	(20.4%)	.062
LVEF	60 (60-65)	60 (60-65)	60 (60-65)	60 (60-65)	.482
RV diameter (CT), mm	45.9 (5.64)	44.5 (6.85)	43.7 (6.37)	38.5 (7.15)	<.001[b,c]
RV/LV ratio (CT)	1.25 (1.13-1.40)	1.21 (1.09-1.38)	1.19 (1.06-1.33)	0.95 (0.82-1.11)	<.001[b,c]
Qanadli score	25 (6.42)	22.8 (6.18)	20.4 (6.27)	14.9 (6.57)	<.001[a,b,c]
RA/LA ratio (CT)	1.41 (0.32)	1.32 (0.26)	1.29 (0.28)	1.06 (0.22)	<.001[b,c]
MPA, mm	31.3 (4.74)	30.6 (4.07)	29.6 (3.79)	27.8 (3.77)	<.001[b,c]
MPA/Ao	0.9 (0.81-1.03)	0.9 (0.8-0.99)	0.88 (0.81-0.96)	0.87 (0.77-0.97)	.881
RPA, mm	24.6 (3.47)	23.6 (3.35)	22.9 (3.04)	21.9 (3.28)	.001[b,c]
LPA, mm	23.6 (3.16)	23.1 (2.83)	22.5 (2.6)	21 (2.87)	.001[b,c]
Pulmonary infarction	31 (17.4%)	22 (12.6%)	19 (10.4%)	20 (11.8%)	.046

Ao, ascending aorta; CT, computed tomography; LA, left atrium; LPA, left pulmonary artery; LVEF, left ventricular ejection fraction; LV, left ventricle; MPA, main pulmonary artery; RA, right atrium; RPA, right pulmonary artery; RV, right ventricle; RV TDI, right ventricular tissue Doppler imaging; sPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion.
[a]Q1 vs. Q2.
[b]Q1 vs. Q3.
[c]Q1 vs. Q4.

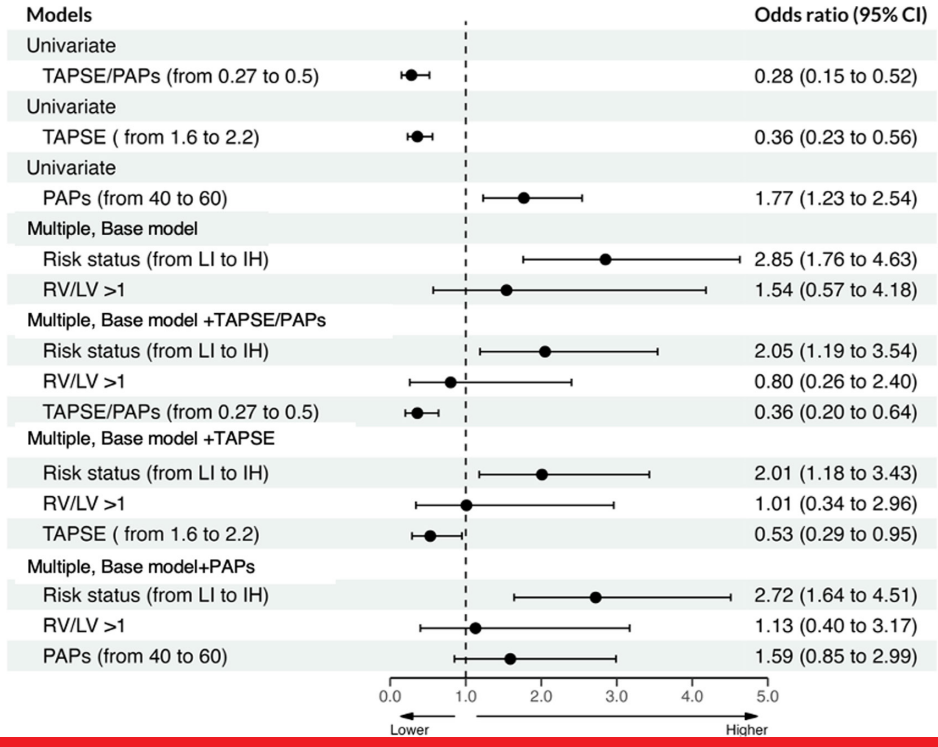

Figure 3. The univariate and multiple regression analyses result in the prediction of in-hospital death.

capability of prediction than TAPSE and sPAP measurements individually. Our findings suggest that as RV–PA coupling deteriorates in patients with acute PE, blood pressure and oxygen saturation decrease, while risk status, PESI, and RV/LV ratio increase.

Assessing the risk of patients with acute PE is crucial for determining the optimal therapeutic management strategy and identifying the notable risk of early mortality.[1] Currently, available risk stratification models for acute PE include hemodynamic status, cardiac biomarkers, RV dysfunction on

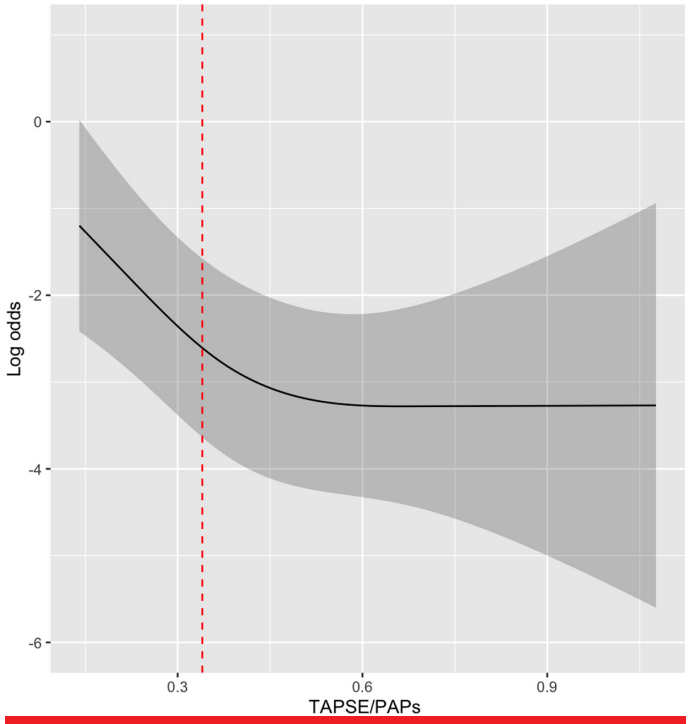

Figure 4. Partial effect plot of TAPSE/sPAP in the prediction of in-hospital death. The vertical dashed red line indicates the best cut-off value of TAPSE/sPAP based on the Youden index (0.34).

TTE or CT angiography, and clinical scores such as PESI and SPESI.[1] The presence of echocardiographic RV dysfunction is associated with short-term mortality even in patients without hemodynamic compromise.[8] Focusing on the length–tension relationship of the ventricle may provide superior predictive capability compared to RV dysfunction for classifying patients with acute PE.

In an acute setting, the RV responds to increased afterload with increased contractility. When this homeometric response becomes impaired, the RV resorts to Starling's law, also known as heterometric or dimensional adaptation. This adaptation mechanism increases pulmonary artery pressure and maintains cardiac output.[14]

Various measurements, including TAPSE/sPAP, fractional area change/invasively measured mean PA pressure, RV area change/end-systolic area, and TAPSE/PA acceleration time, have been suggested as surrogate markers for RV–PA coupling. Guazzi et al[15] demonstrated the robust validation and correlation of the TAPSE/sPAP ratio with RV systolic elastance (Ees) and arterial elastance (Ea), establishing its suitability for clinical practice in acute settings. Furthermore, of these measurements, only TAPSE/sPAP emerged as an independent predictor of Ees/Ea in patients with pulmonary hypertension.[16] Although invasive pressure–volume analysis is considered the gold standard for assessing RV–PA coupling, it is not applicable in evaluating patients with acute PE. Therefore, TAPSE/sPAP, a non-invasive and inexpensive correlate of pressure–volume analysis, might have clinical relevance in evaluating RV dysfunction in patients with acute PE.

Recently, Falsetti et al[17] reported that in intermediate-risk pulmonary embolism, the TAPSE/sPAP ratio independently predicts mortality to a greater extent than CT angiography and troponin. Similarly, Lyhne et al[18] published a retrospective analysis of a pulmonary embolism registry from a single center, including 627 patients. They have indicated that the TAPSE/sPAP ratio can predict short-term adverse outcomes in acute PE. Our retrospective single-center analysis confirms these findings, with a larger and more heterogeneous sample size, including all risk groups and patients treated with CDT and low-dose fibrinolytic drugs.

Based on the ROC curve analysis of our model, the optimal cut-off value of TAPSE/sPAP, determined by the Youden index, was 0.34. This cut-off is comparable to the previously suggested value of 0.4 by Lyhne et al[18] and 0.31 for patients with pulmonary hypertension.[16] Moreover, our analysis still shows a significant inverse association between TAPSE/sPAP and the risk of in-hospital death even after adjusting for risk status in our analysis (change from 0.27 to 0.50, OR = 0.36, 95% CI: 0.20-0.64, $P = .038$). In our single-center PE series, ultrasound-assisted thrombolysis and rheolytic thrombectomy cohorts represent the largest single-center series ever published.[19-23] Considering the relatively large number of patients treated with CDT in this study, the TAPSE/sPAP cut-off value can be interpreted and used to identify patients at risk of deterioration, and guide us to consider more aggressive treatment options across all risk groups.

Study Limitations

In this study, we retrospectively analyzed our single-center data containing a large population of patients. This heterogeneous population includes all risk groups and treatment modalities; therefore, generalizability is not limited. However, a prospective study is needed to define a more accurate TAPSE/sPAP cut-off value to guide aggressive treatments such as CDT in intermediate and intermediate-high-risk groups. Major limitations of echocardiographic evaluation are observer dependency and assessment of RV function. We performed TTE on the first day of presentation; using TAPSE to evaluate RV function may reduce observer dependency and increase reliability and simplicity in acutely presented patients. It is important to highlight that the calculated sPAP is derived from the TR jet using the modified Bernoulli's formula, which may underestimate sPAP in patients with severe TR, constituting significant limitations of the study. Additionally, TAPSE might be overestimated in this particular group of patients. Although using TAPSE/sPAP in this patient group might lead to misleading conclusions, only a small proportion of patients with acute PE present with severe TR. Another limitation is that TAPSE might not reflect the global function of the RV as it only represents longitudinal systolic motion of the RV.

CONCLUSION

The outcomes of our study reveal that the ratio of TAPSE/sPAP serves as a more potent predictor of mortality than either of the 2 measurements taken separately. The interpretation and utilization of the TAPSE/sPAP cut-off value in acute PE can assist in identifying patients at risk of

deterioration and guide the consideration of more intensive treatment options across all risk groups.

Ethics Committee Approval: Ethics Committee approval was obtained at Kartal Koşuyolu Training and Research Hospital (Decision date: December 12, 2023, Decision number: 2023/19/752) and the study complied with the Declaration of Helsinki.

Informed Consent: Informed consent is not applicable due to the design of the study.

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