International Journal of Cardiology Research

ISSN Print: 2663-4104
ISSN Online: 2663-4112
Impact Factor: RJIF 5.29
IJCR 2025; 7(2): 16-22
www.cardiologyjournal.in
Received: 19-04-2025
Accepted: 22-05-2025

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Pulmonary artery pressure-to-stroke volume ratio predicts clinical outcomes in hemodynamically stable acute pulmonary embolism

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DOI: https://www.doi.org/10.33545/26634104.2025.v7.i2a.68

Abstract
Background: Acute pulmonary embolism (PE) carries substantial risk of hemodynamic compromise, yet existing clinical scores incompletely capture right-to-left ventricular interactions. We investigated whether the ratio of pulmonary artery systolic pressure to left ventricular stroke volume (PASP/LVSV), measured within 24 hours of diagnosis, predicts adverse short- and mid-term outcomes in hemodynamically stable PE patients.
Methods: In this prospective, multicenter cohort study, 150 patients with confirmed acute PE underwent standardized transthoracic echocardiography within 24 hours of admission. PASP was estimated from tricuspid regurgitant jet velocity and inferred right atrial pressure, while LVSV was calculated from LV outflow tract area and Doppler velocity time integral. Patients were dichotomized into PASP/LVSV < 1.0 (Group I, n = 72) and ≥ 1.0 (Group II, n = 78). The primary composite endpoint comprised in-hospital mortality, cardiac arrest, or thrombolytic therapy; secondary endpoints included 90-day all-cause mortality and need for ventilatory support.
Results: Group II patients experienced significantly higher rates of the primary composite endpoint (35.9% vs. 12.5%; $p<0.001$) and 90-day mortality or respiratory failure (38.5% vs. 15.3%; $p<0.001$). PASP/LVSV demonstrated superior discrimination for the primary outcome (AUC 0.812; sensitivity 91.0%, specificity (47.2%) compared with Bova (0.645) and PESI (0.605) scores (both $p<0.001$ vs. PASP/LVSV). In multivariate analysis, PASP/LVSV ≥ 1.0 remained an independent predictor of the primary composite (OR 2.15; 95% CI 1.78-5.02; $p<0.001$) and secondary outcomes (OR 1.82; 95% CI 1.18-2.02; p = 0.001).
Conclusion: The PASP/LVSV ratio, reflecting ventriculo-pulmonary coupling, independently predicts adverse in-hospital events and 90-day mortality in stable acute PE. Integrating this echocardiographic marker into early risk stratification may optimize patient management.

Keywords: Pulmonary embolism, echocardiography, ventriculo-pulmonary coupling, pasp/lvsv ratio, risk stratification

Introduction
Acute pulmonary embolism (APE) is a potentially life-threatening condition whereby thrombi, most of which are formed in the deep venous system of the lower extremities, block pulmonary vascular segments. Clinical manifestation is highly variable, ranging between subtle symptomatic dyspnea to acute hemodynamic collapse, and thus rapid and precise risk stratification is needed [1]. Available prognostic tools like Pulmonary Embolism Severity Index (PESI) and Bova score combine clinical, laboratory biomarkers, and simple hemodynamic indicators, however; they do not completely describe the complicated interaction between increased right heart afterload and reduced left ventricular performance [2]. Therefore, the sole use of these instruments may lead to underestimation or overestimation of risk, which causes inadequate escalation or de-escalation of therapy [3].
Recent research has attempted to avoid drawbacks of traditional echocardiographic indices by suggesting measures that directly measure ventriculo-pulmonary coupling. A ratio of pulmonary artery systolic pressure (PASP) to left ventricular stroke volume (LVSV) is one such measure [4]. PASP is noninvasively estimated by continuous-wave Doppler

measurement of the tricuspid regurgitant jet using the simplified Bernoulli equation and including an estimate of right atrial pressure based on the size and collapsibility of the inferior vena cava [5]. The LVSV is calculated by measuring the diameter of the left ventricular outflow tract in parasternal long-axis, calculation of the cross-sectional area, and multiplication by the velocity time integral as determined by Doppler in the apical five-chamber view. The resulting PASP/LVSV ratio therefore represents the correlation between the pressure created by the right ventricle and the stroke output of the left ventricle, which represents the hemodynamic burden of the embolus [6].

Patient stratification based on PASP/LVSV ratios above or below a 1.0 cut-off has demonstrated greater discriminatory value in adverse outcome than either standalone PESI or Bova scores [7]. A ratio of 1.0 or higher defines a subgroup with an inappropriately high extent of pulmonary hypertension compared to left ventricular output and represents poor ventriculo-pulmonary coupling and an increased likelihood of respiratory failure, shock, or death [8]. The integration of the PASP/LVSV into standard echocardiographic assessment would allow earlier detection of the high-risk patients and could guide more specific thrombolysis, catheter-directed treatment, or intensive observation [7, 8]. With the improvement of ultrasound technology and the spread of portable platforms, PASP/LVSV ratio is likely to improve the stratification of APE risk and individual management, shifting from the exclusive clinical scoring to a comprehensive hemodynamic portrait [8]. Hence, we conducted this observational study to further explore the role of PASP/LVSV in predicting the prognosis of PE patients.

Patients and methods

We conducted this study as a prospective observational study, origination from two different centers in Egypt. This study included a total of PE patients admitted to the emergency departments and intensive care units of these centers in a period that spanned between October 2024 to May 2025. In this study we categorized patients into two groups based on the PASP/LVSV ratio to differentiate between high-risk and low-risk groups: Group I (Low risk): PASP/LVSV <1.0. Group II (High risk): PASP/LVSV ≥1.0

Eligibility Criteria

The study employed adult patients with acute pulmonary embolism who were confirmed by computed tomographic pulmonary angiography (CTPA) and categorized the patients as hemodynamically stable (blood pressure = 90 mmHg or more) upon presentation. Patients with a previous history of chronic pulmonary hypertension, chronic pulmonary embolism or right ventricular (RV) infarction or those who had insufficient echocardiographic assessment were excluded.

Study Procedure

All of the included patients in this study underwent the following procedures and assessments:

Clinical and Laboratory Assessment

A detailed medical history was taken including demographic data (age and sex), known cardiovascular risk factors (smoking, diabetes, hypertension), history of previous thromboembolic events and vital signs. Cardiopulmonary examination was done with a focused examination of the heart and lungs to assess chamber enlargement and abnormal sounds. At the same time, the venous blood was taken to conduct investigatory tests, such as high-sensitivity troponin.

Risk stratification

Both the Pulmonary Embolism Severity Index (PESI) and the Bova risk score are reliant on clinical and laboratory variables. PESI integrates eleven variables such as age, cancer history, vital signs, and oxygen saturation to stratify the patients into low, intermediate, and high-risk groups. The Bova score, in turn, categorises normotensive PE patients into three risk groups by combining heart rate, systolic blood pressure, right-ventricular dysfunction, and troponin elevation.

Echocardiography

All echocardiographic assessments was performed within 24h after the diagnosis of PE, utilizing a transthoracic echocardiographic (TTE) machine. LVSL was evaluated by measuring the left ventricular outflow tract (LVOT) in the parasternal long-axis view during the mid-systole period, then we calculated its cross-sectional area (CSA = $\pi \times [\text{diameter}/2]^2$), and multiplied it by the velocity time integral (VTI), that we obtained from pulsed-wave doppler in the apical five-chamber view (SV = CSA × VTI), while PASP was measured from the peak tricuspid regurgitation jet velocity by the simplified Bernoulli equation (PASP = $4 \times \text{velocity}^2$) and then adding an infer-red right atrial pressure based on the inferior vena cava size and collapsibility, then the ratio of PASP/LVSV was estimated to assess its prognostic value.

Outcomes and Follow-up

The follow-up period utilized in our study was 3 months, in which the following outcomes were reported and assessed: 1) Primary Outcomes, which included a composite outcome consisting of (in-hospital mortality, need for systemic thrombolysis, cardiac arrest) and 3-month mortality. 2) Secondary Outcomes, which included the both the need of either non-invasive ventilation or mechanical ventilation.

Statistical analysis

The analysis of data in this research was done with IBM SPSS Statistics version 28 (IBM Corp., Chicago, IL). The continuous variables were described by mean and standard deviation, and the normal distribution of each variable was confirmed by Shapiro-Wilk test. In order to assess the differences between groups, unpaired Student t-test was used. The categorical variables were reported as frequencies and percentages; the relationship was tested using the Chi-square test or the Fisher exact test based on the sample size. The p-value of 0.05 or less was considered to be significant and two-tailed. Multivariate logistic regression was then applied to establish the relationship between the dependent variable and various independent covariates.

Results

Table 1 of our study demonstrates that in the pooled cohort, baseline characteristics such as, age, sex, height, weight, body mass index (BMI), prevalence of diabetes mellitus (DM), prevalence of hypertension (HTN) and prevalence of smoking did not statistically differ between the two groups,

which indicates the similarities between the two groups at baseline.

Risk stratification scores were reported in Table 2. Group I was associated with lower PESI and Bova scores, that were both clinically and statistically significant (109.6±21.2 vs 122.5±18.7; $P<0.001$; 4.1±0.78 vs 5.1±0.85, $P<0.001$).

Table 2 demonstrates that the two groups are statistically significantly different regarding the risk prediction scores, as G1 was associated with lower PESI and BOVA scores compared to G2 (109.6 vs 122.5, 4.1 vs 5.1), indicating lower risk of complications and overall better prognosis.

Regarding the echocardiographic parameters utilized in our study, Table 3 reported that across all the included 150 participants, mean LVSV was (51.1±14.5 ml), while mean PASP was (49.4±15.1 mmHg). Further analysis revealed that G1 was associated with a statistically higher LVSV and lower PASP values compared to G2 (59.7±8.1mL vs 42.5±14.2 mL, 39.8±10.5 mmHg vs 58.2±13.1 mmHg), which reveal that patients in G1 had greater stroke volumes and lower pulmonary pressure than patients in G2.

We also observed that G2 patients had significantly worse clinical outcomes as compared to Group I. A closer analysis of the composite endpoints showed that in-hospital mortality (17.9% vs. 6.9%, p = 0.023), the need of thrombolytic therapy (20.5% vs. 9.7%, p = 0.032), and instances of cardiac arrest (14% vs. 5%, p = 0.023) were all more prevalent in Group II than in Group I. The cumulative all-cause mortality at 3 months also showed a greater incidence in Group II (17.9% vs. 6.9%, p = 0.031), thereby confirming the finding of poorer composite outcomes. Table 4

Analysis of our secondary outcomes revealed that patients in Group I were associated with a statistically and clinically higher need for non-invasive ventilation compared to the patients in Group II (14.0% vs 6.94%). Similarly, 23 of the 150 included patients were indicated to receive mechanical ventilation, with the majority of these patients belonging to Group II compared to Group I (21.79% vs 8.33%). Table 5

Table 6 summarizes the diagnostic performance of the individual scoring systems to predict the main outcomes. The higher discriminative ability of the PASP/LVSV ratio can be observed when these univariate findings are compared: namely, it has the highest area under the receiver operating characteristic curve (AUC 0.812). This is better than that of the Bova score (AUC 0.645) and the PESI score (AUC 0.605). The differences are also supported by comparisons of pairs of scores. Direct ROC comparisons (Table 7) show that PASP/LVSV is considerably more accurate than the Bova score (0.167, 95% CI 0.0870.243, $p<0.001$), and the PESI score (0.207, 95% CI 0.1150.299, $p<0.001$), whereas the Bova and PESI scores do not differ significantly (0.040, 95 These findings are confirmed by multivariate logistic regression analysis, which is provided in Table 8: only the PASP/LVSV ratio is independent of the primary outcomes (OR 2.15, 95% CI 1.78 5.02, $p<0.001$). In contrast, the PESI score (OR 1.007, 95% CI 0.9911.023, p = 0.531) and the Bova score (OR 1.082, 95% CI 0.7221.632, p = 0.700) fail to be significant when the rest of the variables in the regression model are controlled.

In multivariate logistic regression analysis for the secondary outcomes, both the PESI and the BOVA scores failed to significantly predict the incidence of the secondary outcomes OR 1.011; p = 0.325, OR 1.040; p = 0.880). On the other hand, the PASP/LVSV ratio was significantly and independently associated with the need for respiratory support (OR 1.820; p = 0.001). Table 9

Discussion: In this observational cohort study, we included a total of 150 patients diagnosed with PE and stratified based on the PASP/LVSV ratio into low-risk group (G1) and high-risk group (G2).

While the initial troponin level was positively higher in Group II (74.4%) compared to Group I (44.4%), other baseline characteristics were similar between the two groups, including comparable rates of smokers (≈30%), diabetics (≈40%), and hypertensives (≈37%).

Higher concentrations of troponin in patients with PE are linked to a higher risk of poor clinical outcomes, such as hemodynamic collapse, PE recurrence, and overall death during the 30-day follow-up period [9]. In normotensive PE patients, high troponin is a strong predictor of overall mortality (odds ratio 4.26), but it is not very useful in separating high- and low-risk patients. The use of high-sensitivity troponin assays allows identifying more patients with only modest increases, although conventionally measured troponin (cTnI) concentrations are more closely associated with complex clinical courses [10].

In concordance with our findings, evidence from literature indicates that while both hypertension and smoking are risk factors for developing venous thromboembolisms (VTEs), which included PC, their role in predicting the prognosis of patients with PE is either poor like in the cases of smoking or unclear like in the cases of hypertension [11, 12].

In our study, we observed that patients in Group I were associated with better risk prediction scores than Group II, evident by the significantly lower PESI score in Group I (109.6±21.2) than in Group II (122.5±18.7; $p<0.001$), as well as BOVA score 4.1±0.78 in Group I versus 5.1±0.85 in Group II ($p<0.001$).

This is consistent with Kamran *et al.*, that reported that in patients with PASP/LVSV ≥1.0 mm Hg/mL, both the mean PESI and BOVA scores were lower than in patients with PASP/LVSV <1.0 (119.5 vs 108, 3.9 vs 3.2) [13].

Regarding the echocardiographic parameters reported in our analysis, we observed that Group I exhibited higher LVSV (59.7±8.1 mL) compared with Group II (42.5±14.2 mL; $p<0.001$), while conversely, PASP was lower in Group I (39.8±10.5 mmHg) than in Group II (58.2±13.1 mmHg; $p<0.001$).

In concordance with our findings, Karman *et al.*, which was a registry-based cohort study evaluating the data of 215 patients after stratifying them into two groups based on the PASP/LVSV ratio. In this study PASP levels were significantly higher in the high-risk group compared to the low-risk group (57.5±15.6 vs 39.8±9.7) [13].

Additionally, in other relevant studies investigating the predictive value of TAPSE/sPAP in PE patients, PASP values were also significantly higher in the high-risk group compared with the low-risk groups (65 vs 30 mmHg) [14].

Regarding our primary outcomes, Group II was consistently associated with worse outcomes, evident by the higher rates of in-hospital mortality, cardiac arrest, need for thrombolytic therapy as well as the higher 90-day mortality risk.

This is consistent with Karman *et al.*, which reported a higher 30-day mortality rate in the high-risk group (7.0% vs 0%) and 90-day mortality rate (12.5% vs 9.7%). Furthermore, the incidences of cardiac arrest was also

higher in the high-risk group (6.2% vs 2.9%), as well as the need for thrombolytic therapy (45.5% vs 29.1%) [13].

Our findings demonstrated that the PASP/LVSV ratio showed superior discrimination ability (AUC 0.812; sensitivity 91.0%; specificity 47.2%; $p<0.001$) over Bova (AUC 0.645) and PESI (AUC 0.605). Further analysis with ROC curve comparisons supported the superiority of PASP/LVSV over both PESI and Bova predictive scores.

In alignment with our findings, Karman *et al.*, reported that a ≥1.0 mm Hg/mL was an optimal cut-off value for PASP/LVSV ratio, which in the study outperformed both PESI (OR 1.43, p=0.06) and Bova (OR 1.28, p=0.01) scores in the prediction of clinical outcomes [13].

Moreover, we also observed that patients in Group II were associated with a higher need for respiratory support, evident by the statistically higher rates of non-invasive ventilation (6.9% vs. 20.5%; p = 0.026), as well as the need for mechanical ventilation (8.3% vs. 21.8%; p = 0.048).

Interestingly, our multivariate analysis revealed that only PASP/LVSV had the ability to accurately predict the secondary outcomes of the study (OR 1.82; p = 0.001); because PESI and Bova scores were non-significant.

In literature, evidence support our findings, as lower PASP/LVSV ratio was associated with higher rates of need of respiratory support compared with higher PASP/LVSV ratio [15].

PASP/LVSV echocardiographic ratio forms a paradigm shift in the risk assessment of PE, as they have a higher predictive accuracy than that of conventional clinical scoring systems, including the PESI and Bova scores [16]. These easily available bedside measures enable clinicians to recognize high-risk patients with high 30-day mortality rates (up to 7%) and apply aggressive interventional treatment. The ratio is useful in predicting the need of advanced reperfusion treatments (45.5% versus 29.1% in high-risk versus low-risk groups) and thrombolytic therapy (26.3% versus 0%), thus becoming an invaluable tool in the emergency department triage and early decision to escalate treatment [17].

Assessment of these echocardiographic ratios significantly re-arranges resource use in the management of acute pulmonary embolism by identifying patients who need constant monitoring and respiratory support. The results indicate that high-risk patients have significantly high chances of mechanical ventilation and non-invasive ventilation (46.4% vs 22.3%), which necessitates prior consultation with intensive-care unit staff and respiratory therapists [18]. The ability to reassess in real-time allows constant surveillance of clinical status, optimal bed use, and proactive intervention to prevent a decline [19]. These statistics support the necessity to incorporate echocardiographic ratio evaluation into the routine management procedures, especially in the facilities that have pulmonary embolism response teams, with the aim to improve patient outcomes and increase the efficiency of healthcare resources [20].

Table 1: Baseline characteristics of the included patients

Variables		Total (n=150)	G I (n=72)	G II (n=78)	P value
Age (years)	Mean ±SD	58.2±10.9	57.1±11.5	59.1±10.2	0.245
	Range	36 - 74	36 - 74	38 - 74	
Sex	Male	80 (53.3%)	36 (50.0%)	44 (56.4%)	0.366
	Female	70 (46.7%)	36 (50.0%)	34 (43.6%)	
Weight (Kg)	Mean ±SD	75.2±7.8	74.9±7.5	75.5±8.0	0.765
	Range	62 - 88	62 - 88	63 - 88	
Height (m)	Mean ±SD	1.67±0.05	1.67±0.04	1.67±0.05	0.724
	Range	1.58 - 1.75	1.58 - 1.73	1.59 - 1.75	
BMI (Kg/m^2)	Mean ±SD	27.3±3.2	27.0±3.1	27.5±3.2	0.521
	Range	21.0 - 35.0	21.0 - 35.0	21.5 - 34.5	
Troponin (ng/mL)	Positive	90 (60.0%)	32 (44.4%)	58 (74.4%)	<0.001*
	Negative	60 (40.0%)	40 (55.6%)	20 (25.6%)	
Smoking	Frequency (Percentage)	45 (30.0%)	22 (30.6%)	23 (29.5%)	0.701
DM	Frequency (Percentage)	60 (40.0%)	28 (38.9%)	32 (41.0%)	0.766
HTN	Frequency (Percentage)	56 (37.3%)	28 (38.9%)	28 (35.9%)	0.427

BMI; body mass index, DM; diabetes mellitus, HTN; hypertension, *; statistical significancy p value ≤ 0.05

Table 2: Risk scores of the included patients

Variables		Total (n=150)	G I (n=72)	G II (n=78)	P value
PESI score	Mean ±SD	116.8±20.4	109.6±21.2	122.5±18.7	<0.001*
	Range	74 - 150	74 - 145	88 - 150	
Bova score	Mean ±SD	4.7±0.88	4.1±0.78	5.1±0.85	<0.001*
	Range	3 - 6	3 - 5	4 - 6	

PESI; pulmonary embolism severity index, *; statistical significancy p value ≤ 0.05.

Table 3: Echocardiographic parameters of the included patients

Variables		Total (n=150)	G I (n=72)	G II (n=78)	P value
LVSV (ml)	Mean ±SD	51.1±14.5	59.7±8.1	42.5±14.2	<0.001*
	Range	22 - 84	43 - 84	23 - 71	
PASP (mmHg)	Mean ±SD	49.4±15.1	39.8±10.5	58.2±13.1	<0.001*
	Range	22 - 82	22 - 61	28 - 81	

LVSV; Left ventricular stroke volume, PASP; pulmonary arterial systolic pressure, *; statistical significancy p value ≤ 0.05.

Table 4: Primary Outcomes of the study

Variables	Total (n=150)	G I (n=72)	G II (n=78)	P value
In-hospital mortality	17 (11.3%)	5 (6.9%)	13 (16.7%)	0.019*
Cardiac arrest	20 (13.3%)	5 (6.9%)	14 (17.9%)	0.023*
Thrombolytic therapy	23 (15.3%)	7 (9.7%)	16 (20.5%)	0.032*
Cumulative 90-day all-cause mortality	19 (12.7%)	5 (6.9%)	14 (17.9%)	0.031*

*; statistical significancy p value ≤ 0.05, data is presented as frequency (percentage).

Table 5: Secondary Outcomes of the study

Variables	Total (n=150)	G I (n=72)	G II (n=78)	P value
Non-invasive ventilation	21 (14.0%)	5 (6.94%)	16 (20.51%)	0.026*
Mechanical ventilation	23 (15.3%)	6 (8.33%)	17 (21.79%)	0.048*

*; statistical significancy p value ≤ 0.05, data is presented as frequency (percentage).

Table 6: Diagnostic accuracy for prediction of the primary outcomes

Variables	Sensitivity	Specificity	PPV	NPV	AUC	P value
PESI score	78.2	36.5	42.3	70.1	0.605	0.004*
Bova score	67.1	58.8	48.7	74.5	0.645	<0.001*
PASP/LVSV	91.0	47.2	51.3	89.0	0.812	<0.001*

PESI; pulmonary embolism severity index, PPV; positive predictive value, NPV; negative predictive value, AUC; area under the curve, *: statistically significant as p value <0.05.

Table 7: ROC curve comparison between Bova score, PESI score and PASP/LVSV for prediction of the primary outcomes

PASP/LVSV ~ Bova score	
Difference between areas	0.167
95% Confidence Interval	0.087 to 0.243
Significance level	*P*<0.001*
Bova score ~ PESI score	
Difference between areas	0.040
95% Confidence Interval	-0.062 to 0.142
Significance level	P = 0.3990
PASP/LVSV ~ PESI score	
Difference between areas	0.207
95% Confidence Interval	0.115 to 0.299
Significance level	*P*<0.001*

*: statistically significant as p value <0.05.

Table 8: Multivariate logistic regression analysis of predictive scoring systems for the primary outcomes

Variables	OR	95% CI	P value
PESI score	1.007	0.991 - 1.023	0.531
Bova score	1.082	0.722 - 1.632	0.700
PASP/LVSV	2.15	1.78 to 5.02	<0.001*

PESI; pulmonary embolism severity index, LVSV; Left ventricular stroke volume, PASP; pulmonary arterial systolic pressure *; statistical significancy p value ≤ 0.05.

Table 9: Multivariate logistic regression analysis of predictive scoring systems for the secondary outcomes

Variables	OR	95% CI	P value
PESI score	1.011	0.993 to 1.029	0.325
Bova score	1.04	0.68 to 1.61	0.880
PASP/LVSV	1.82	1.18 to 2.02	0.001*

PESI; pulmonary embolism severity index, LVSV; Left ventricular stroke volume, PASP; pulmonary arterial systolic pressure *; statistical significancy p value ≤ 0.05.

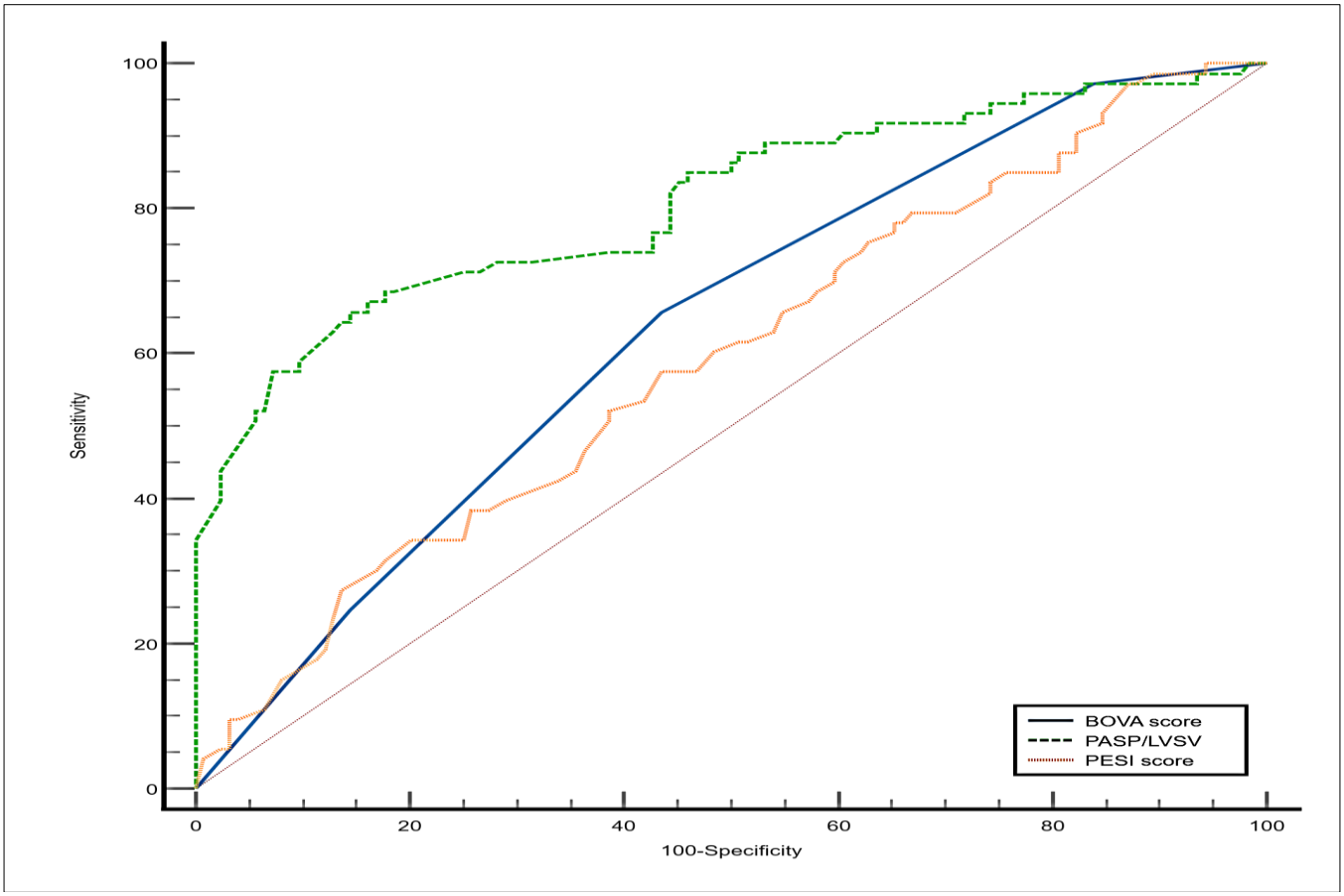

Fig 1: ROC curve comparison between BOVA score, PESI score and PASP/LVSV for prediction of the primary outcomes

Conclusion

In hemodynamically stable acute PE, a PASP/LVSV ratio ≥ 1.0 independently predicts adverse in-hospital and 90-day outcomes, outperforming PESI and Bova scores; integrating this metric may substantially enhance clinical risk stratification.

Sources of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

Authors contributed equally to the study**.**

Conflicts of interest

No conflicts of interest

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How to Cite This Article
Elsawah AM, Ghanem ML, Ali AA, Khairy H, Imam AM, Makram M, Omar R, Elhadainy MS. Pulmonary artery pressure-to-stroke volume ratio predicts clinical outcomes in hemodynamically stable acute pulmonary embolism. International Journal of Cardiology Research 2025;7(2):16-22