

International Journal of Cardiology Sciences

ISSN Print: 2664-9020
ISSN Online: 2664-9039
Impact Factor: RJIF 5.63
IJCS 2025; 7(2): 15-20
www.cardiologyjournals.net
Received: 07-05-2025
Accepted: 09-06-2025

Ahmed Mohsen Elsawah
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Mosaad Lamey Ghanem
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Ahmed Ali Ali
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Hatem Khairy
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Amr Mohamed Imam
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Mohamed Makram
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Ramy Omar
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Mohamed Sabry Elhadainy
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Corresponding Author:
Mohamed Sabry Elhadainy
Department of Cardiology,
National Heart Institute,
Cairo, Egypt

Echocardiographic right ventricular metrics and clinical outcomes in acute PE: Insights from a prospective cohort

Ahmed Mohsen Elsawah, Mosaad Lamey Ghanem, Ahmed Ali Ali, Hatem Khairy, Amr Mohamed Imam, Mohamed Makram, Ramy Omar, and Mohamed Sabry Elhadainy

DOI: <https://www.doi.org/10.33545/26649020.2025.v7.i2a.114>

Abstract

Background: Acute pulmonary embolism (PE) imposes abrupt hemodynamic stress on the right ventricle; however, the prognostic value of specific echocardiographic markers remains unclear. We aimed to determine whether RV parameters assessed within 24 hours of diagnosis predict poor clinical outcomes.

Methods: In this multicenter prospective cohort, 150 hemodynamically stable PE patients underwent transthoracic echocardiography within 24 hours. Parameters including TAPSE, RVFAC, RVOT VTI, RVOT AT, and RV tissue Doppler S' velocity were recorded. Patients were stratified by PASP/LVSV ratio and followed for a composite endpoint of in-hospital mortality, cardiac arrest, or thrombolysis, plus 90-day mortality.

Results: High-risk patients (PASP/LVSV ≥ 1.0 ; n=78) exhibited significantly lower TAPSE (1.52 ± 0.26 vs 1.72 ± 0.28 cm), RVOT VTI (9.7 ± 1.3 vs 12.3 ± 1.7 cm), RVOT AT (59 ± 12 vs 79 ± 15 ms), RVFAC (31 ± 11 vs $45 \pm 8.5\%$), and S' velocity (9.0 ± 1.5 vs 11.4 ± 2.4 cm/s) (all $P < 0.001$). Composite outcomes occurred more frequently in this group ($P < 0.05$). Multivariate analysis identified RVOT AT (OR 1.022 per ms; $p = 0.043$) and S' velocity (OR 1.054 per cm/s; $p = 0.038$) as independent predictors.

Conclusion: Echocardiographic RVOT acceleration time and tissue Doppler systolic velocity independently predict adverse short-term outcomes in acute PE. Incorporating these metrics into early risk assessment may improve identification of high-risk patients and guide timely intervention.

Keywords: Pulmonary embolism; echocardiography; right ventricular dysfunction; RVOT acceleration time; tissue Doppler imaging

Introduction

Traditionally considered in the periphery of the cardiopulmonary paradigm, the role of the right ventricle (RV) in mediating the pathophysiology has regained academic attention, especially in the setting of acute pulmonary embolism (PE) [1]. In contrast to left-sided indices that have long been the focus of clinical interest, recent data suggest that RV strain as a clinically apparent by dilation, regional hypokinesia or increased central pressures is an antecedent of poor clinical outcomes [2]. In this sense, echocardiography provides a conveniently accessible, non-invasive method to question the dynamics of RV: indices such as tricuspid annular plane systolic excursion (TAPSE), right ventricular fractional area change (RVFAC) and tissue Doppler-derived systolic velocities (S x) have been proposed as surrogate markers of hemodynamic compromise [3]. However, there have been methodological disparities in measuring procedures and diagnostic cut-offs that have hindered its extensive inclusion into standardized risk-stratification models [1-3].

The clinical manifestation of acute PE has now come to be accepted as pathophysiological mechanisms. The radiological hallmark of pulmonary embolism, vascular obstruction followed by elevated pulmonary vascular resistance, leads to a sudden increase in right ventricular afterload [4]. Since the right ventricle is structurally designed to handle large volumes, but not to produce high pressures over long periods, this sudden rise in afterload

exceeds the structural capacity of the right ventricle, leading to acute dilation and severe loss of contractility^[5]. The subsequent decrease in stroke volume triggers tachycardia, which is an adaptation aimed at sustaining systemic perfusion, instantly. In case such regulatory compensations fail later on, poor end-organ perfusion occurs^[6].

The aim of the current study was to clarify which RV parameters, considered separately, are capable of predicting poor outcomes in the case of acute PE. These results could optimize bedside risk estimation and guide therapeutic choices, because they could help identify high-risk patients early, accelerate the initiation of interventions (e.g., thrombolysis or catheter-directed therapy), and increase survival in the end^[6, 7]. As a result, the careful design of methodology and thorough examination, the study was to shed light in the prognostic subtlety of RV dysfunction and to identify new avenues of improved care of PE victims.

Methodology

This was a multi-center prospective observational cohort study conducted during the period from October 2024 to May 2025, following the approval of the institutional ethics committee. The study included 150 patients diagnosed with PE and admitted to the study locations. 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.

Prior to participation, informed written consent was obtained from all subjects, then each participant received a unique identification code to maintain confidentiality.

Eligibility Criteria

Our inclusion criteria included patients who had a confirmed diagnosis of acute PE, based on computed tomographic pulmonary angiography (CTPA), and were defined as hemodynamically stable (BP ≥ 90 mmHg) upon admission. We excluded patients with previous history of chronic pulmonary hypertension, chronic pulmonary embolism, right ventricular (RV) infarction, or inadequate echocardiographic evaluation.

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 obtained, including demographic data (age, sex), risk factors (smoking, diabetes, hypertension), prior thromboembolic events, vital signs. Patients also received a focused cardiopulmonary examination, assessing chamber enlargement and abnormal heart and lung sounds, while blood samples were collected for various investigatory tests, such as high-sensitivity troponin.

Risk stratification

Clinical and laboratory parameters were utilized in the calculation of Pulmonary Embolism Severity Index (PESI) and Bova risk scores. PESI is a combination of eleven variables (age, cancer history, vital signs, oxygen saturation) to classify patients into low, intermediate, and high-risk groups. Bova score on the other hand categorizes normotensive PE patients into three risk levels by

integrating the heart rate, systolic blood pressure, right-ventricular dysfunction, and troponin elevation.

Echocardiography

Within 24 hours of PE diagnosis, we utilized transthoracic echocardiography (TTE) to evaluate the clinical condition of the admitted patients. Multiple relevant echocardiographic parameters were recorded for each patient: left ventricular outflow tract velocity-time integral (LVOT VTI), left ventricular ejection fraction (LVEF), tricuspid annular plane systolic excursion (TAPSE), right ventricular outflow tract velocity-time integral (RVOT VTI), right ventricular outflow tract acceleration time (RVOT AT), right ventricular fractional area change (RVFAC), and right ventricular tissue Doppler imaging systolic velocity (RV TDI S'). Other echocardiographic variables such as, pulmonary artery systolic pressure (PASP), Left ventricular stroke volume (LVSV) and PASP/LVSL were recorded and will be evaluated in another report.

Outcomes and Follow-up

Patients were followed for up to 3 months, which included the in-hospital and post-discharge periods. In this study, we utilized a primary composite outcome, which was comprised of (in hospital mortality, systemic thrombolysis, or cardiac arrest). The secondary outcome was the Cumulative mortality for the 3-month follow-up period.

Statistical analysis

Data analysis in the present study was performed using the IBM SPSS Statistics version 28 (IBM Corp., Chicago, IL). Continuous variables were presented as mean SD and the normalcy of the distributions was established using Shapiro-Wilk test. The unpaired Student t-test was used to determine the differences between groups. The categorical variables were described as frequencies and percentages and the relationship between these variables was investigated using the Chi-square test or Fisher exact test as per the need. The p-value of two-tailed 0.05 and below was considered to be significant. Multivariate logistic regression was used to assess the correlation between dependent and multiple independent variables.

Results

In Table 1, we demonstrated that the baseline characteristics of our included 150 patients did not differ significantly between the two groups (Group I= 72 patients, Group II= 78 patients). The overall cohort had a mean age of 58.2±10.9 years, weight of 75.2±7.8kg, height of 1.67±0.05m and body mass index (BMI) of 27.3±3.2Kg/m². Males comprised 53.3% of the patients, while females were 46.7% of the cohort. Diabetes mellitus was the most common present risk factor in this cohort (40%), followed by hypertension (37.3%) and smoking (30%).

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, $P<0.001$)

A quantitative overview of the echocardiographic results achieved in the series of 150 participants is provided in Table 3 that is stratified into Group I (n = 72) and Group II (n = 78). There was no significant difference in the left ventricular ejection fraction (LVEF) between the two groups: 59.3 + 4.3% in Group I and 59.7 + 4.7% in Group II

($p = 0.501$). On the other hand, measures of left ventricular (LV) outflow tract function and right ventricular (RV) measures were all significantly reduced in Group II. Group I averaged 17.5 ± 2.8 cm and Group II averaged 14.5 ± 2.1 cm ($p < 0.001$) LVOT VTI. In a similar manner, tricuspid annular plane systolic excursion (TAPSE) was 1.72 ± 0.28 cm in Group I and 1.52 ± 0.26 cm in Group II ($p < 0.001$). Velocity-time integral of right ventricular outflow tract (RVOT VTI) and acceleration time (RVOT AT) were also similarly impaired: 9.7 ± 1.3 cm versus 12.3 ± 1.7 cm ($p < 0.001$) and 59.0 ± 12.0 ms versus 79.0 ± 15.0 ms ($p < 0.001$), respectively. There were parallel reductions in RV fractional area change (RVFAC) and tissue Doppler S' velocity representing reduced RV systolic performance: $31.0 \pm 11.0\%$ versus $45.0 \pm 8.5\%$ ($p < 0.001$) and 9.0 ± 1.5 cm/s versus 11.4 ± 2.4 cm/s ($p < 0.001$). All of this collectively shows significant worsening of RV function in Group II even with maintained LV systolic performance.

Regarding the clinical outcomes of our study, we observed that in Group II patients experienced significantly worse composite outcomes compared to Group I. Further analysis of the individual outcomes of the composite outcomes revealed that in-hospital mortality was higher in Group II (17.9% vs. 6.9%, $p = 0.023$), as well as the need for thrombolytic therapy (20.5% vs. 9.7%, $p = 0.032$) and cardiac arrest (14% vs. 5%, $p = 0.023$). Additionally, 90-days cumulative all-cause mortality was elevated in Group II compared to Group I (17.9% vs. 6.9%, $p = 0.031$). Table 4 Patients who experienced the composite outcome ($n=50$) demonstrated significantly worse right ventricular function than those without events ($n=100$), with lower TAPSE (1.48 ± 0.32 cm vs. 1.65 ± 0.29 cm; $p = 0.039$), RVOT VTI (10.2 ± 1.9 cm vs. 11.3 ± 2.2 cm; $p = 0.001$), LVOT VTI (15.1 ± 2.6 cm vs. 16.7 ± 2.8 cm; $p = 0.019$), RV tissue Doppler velocity (9.6 ± 2.0 cm/s vs. 10.4 ± 2.2 cm/s; $p = 0.037$), and RVFAC ($31.0 \pm 12.0\%$ vs. $42.0 \pm 10.5\%$; $p = 0.021$), whereas differences in RVOT AT (65.0 ± 14.8 ms vs. 71.5 ± 17.5 ms; $p = 0.069$) and LVEF ($58.1 \pm 4.7\%$ vs. $60.2 \pm 4.3\%$; $p = 0.254$) were not statistically significant. Table 5

In multivariate logistic regression—including PESI and Bova scores, LVOT VTI, RVOT VTI, RVOT AT, RV tissue Doppler velocity, RVFAC, and LVEF—only RVOT AT (OR 1.022 per ms increase; 95% CI: 1.001-1.044; $p = 0.043$) and RV tissue Doppler velocity (OR 1.054 per cm/s increase; 95% CI: 1.003-1.108; $p = 0.038$) remained independent predictors of the primary composite outcome. Table 6

Discussion

This study evaluated 150 patients diagnosed with PE, which was confirmed by CTPA, who were classified into two groups based on the risk of clinical deterioration, evident by the PASP/LVSV ratio. Group II was associated with higher risk the composite primary endpoint (in-hospital mortality, need for thrombolytic therapy and cardiac arrest) as well as 90-day mortality risk compared to Group I. Moreover, patients who had the primary outcomes were consistently associated with worse RV functions evident by the worse parameters of TAPSE, RVOT VTI, LVOT VTI, and RV tissue doppler velocity. Finally, our multivariate analysis revealed that of all parameters, only RVOT AT and RV tissue doppler velocity were independent predictors of the primary composite endpoint.

The current study confirms the previous reporting of the statistically significant impairment of various parameters of RV functions in patients with high PASP/LVSV ratios [8, 9]. In particular, Group II subjects showed lower TAPSE, RVOT VTI, RVOT AT, RVFAC, and RV tissue Doppler velocity, which are in agreement with other studies that have classified these values as predictors of RV dysfunction. TAPSE, specifically, has undergone a lot of validation as a prognostic factor in acute PE; a value below 16-18 mm is linked to higher mortality risk. The present results, 1.48 ± 0.32 cm in the adverse-outcome group and 1.65 ± 0.29 cm in the favorable-outcome group, support this cut-off point and the clinical usefulness of TAPSE [10].

It is important that multivariate analysis revealed right ventricular outflow tract acceleration time (RVOT AT) and right ventricular tissue Doppler velocity as independent predictors of the primary composite outcome. RVOT AT is a close approximation of mean pulmonary artery pressure and less than 60 ms suggests severe pulmonary hypertension [11]. The association of RVOT AT less than 60 ms and pulmonary artery systolic pressure less than 60 mmHg (the 60/60 sign) has been specifically linked with acute cor pulmonale caused by pulmonary embolism, with high specificity (94%) [12]. Similarly, RV tissue Doppler velocity transmits data on myocardial contractility; reduced velocities indicate poor performance. The fact that these parameters have independent prognostic value means that they measure different aspects of right ventricular dysfunction and, together, can be used to give complementary variables to risk stratification [13].

The study's mortality rates align with published literature, with the high-risk group (Group II) experiencing 17.9% in-hospital mortality and 17.9% 90-day mortality. These figures are consistent with intermediate-risk PE mortality rates reported in large registries, which typically range from 5-25% [14]. The need for thrombolytic therapy in 20.5% of high-risk patients also reflects appropriate identification of patients requiring advanced interventions [15].

This study explains the mechanistic basis that regulates the association between RV functional dynamics and clinical outcomes in acute pulmonary embolism. Although left ventricular ejection fraction was similar in the study cohorts, there was a significant difference in the right ventricular parameters, especially dilation, decreased systolic performance, and increased strain, which highlights the selective effect of acute PE on the right-sided cardiac homeostasis. These results support the paradigm of acute cor pulmonale in which RV dysfunction is noted in the presence of intact LV systolic competence [16].

LVOT VTI analysis was used to show a statistically significant decrease in the high-risk group, although LVEF was preserved. This finding favors the clinical entity of ventricular interdependence, whereby the dilation of the right ventricle (RV) causes impairment of left ventricular (LV) filling by pushing the interventricular septum to the LV cavity. This hemodynamic limitation is deeply reflected by the ratio of pulmonary systolic pressure to LV stroke volume, which measures the equilibrium between afterload and forward flow [17].

The time-course association RV dysfunction and clinical outcomes indicate the significance of early evaluation. The protocol of the current study requiring echocardiography within 24 hours of the diagnosis of PE is in agreement with the existing recommendations on the early risk stratification.

The brief duration is essential, as RV dysfunction may develop fast in acute PE with the risk of hemodynamic collapse within hours of arrival [18].

Clinical Implications

The current clinical practice guidelines suggest that thrombolytic therapy should be used in patients with acute PE and hemodynamic instability; its application in PE with intermediate risk is less evident. The current research suggests that patients with RV dysfunction could benefit more with more invasive treatment methods, including thrombolytic therapy or catheter-based procedures. This hypothesis is supported by the increased rate of thrombolytic therapy applied to Group II patients (20.5%) than to Group I patients (9.7%) [19].

The prognostic value of single echocardiographic variables deserves special attention in the current cardiology practice. The acceleration time of right ventricular outflow tract (RVOT AT) and the velocity of right ventricular tissue Doppler have been demonstrated repeatedly to serve as independent predictors of adverse outcomes and thus extend the clinical applicability of conventional measures of RV performance. Furthermore, the parameters can be easily added to modern echocardiographic protocols, providing clinicians with extra prognostic data without being too expensive additions to the standard evaluation [20].

The current research confirms the usefulness of early echocardiographic study in patients with acute PE. The early

detection of high-risk individuals within 24 h of diagnosis allows the timely initiation of specific monitoring and treatment. This is in line with the modern focus on multidisciplinary Pulmonary Embolism Response Teams (PERT), the decision-making of which is based on the quick risk stratification [15].

Strengths, Limitations and Recommendations

The current study was conducted in a prospective, multicentric manner and included the standardized echocardiographic assessment within 24 h of the PE diagnosis, which enhanced the internal validity of the research and its clinical applicability. However, several limitations reduce generalizability and level of inference: a relatively small sample size, possible inter-observer variability, the lack of usage of advanced imaging technologies, the lack of invasive hemodynamic confirmation, and the limited follow-up period limit the scope of conclusions. Future studies must therefore attempt to confirm the prognostic utility of the PASP/LVSV ratio in multicentre populations, incorporate novel echocardiographic technologies, explore comparisons with gold-standard measures and perform randomized trials to determine whether echocardiographic-based risk stratification alters therapeutic outcomes and therefore optimize clinical management of patients with acute pulmonary embolism.

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 significance 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 significance 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
LVOT VTI (cm)	Mean±SD	16.0±3.0	17.5±2.8	14.5±2.1	<0.001*
	Range	10 - 23	13 - 23	10 - 17	
LVEF (%)	Mean±SD	59.5±4.5	59.3±4.3	59.7±4.7	0.501
	Range	50 - 68	52 - 68	50 - 67	
TAPSE (cm)	Mean±SD	1.62±0.30	1.72±0.28	1.52±0.26	<0.001*
	Range	1.0 - 2.3	1.1 - 2.3	1.0 - 2.0	
RVOT VTI (cm)	Mean±SD	11.0±2.2	12.3±1.7	9.7±1.3	<0.001*
	Range	7.0 - 16	9.0 - 16	7.0 - 12	
RVOT AT (ms)	Mean±SD	68.0±16.5	79.0±15.0	59.0±12.0	<0.001*
	Range	38 - 102	54 - 102	38 - 80	
RVFAC (%)	Mean±SD	38.5±12.0	45.0±8.5	31.0±11.0	<0.001*
	Range	14 - 65	31 - 65	14 - 52	
RV tissue Doppler velocity (cm/s)	Mean±SD	10.0±2.3	11.4±2.4	9.0±1.5	<0.001*
	Range	7 - 16	7 - 16	7 - 11	

LVOT; left ventricular outflow tract, VTI; velocity time integral, LVEF; left ventricular ejection fraction, RVOT; right ventricular outflow tract, AT; acceleration time, RVFAC; right ventricular fractional area change, *, statistical significance p value ≤ 0.05.

Table 4: Clinical 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 significance p value ≤ 0.05, data is presented as frequency (percentage).

Table 5: Comparison between patients with and without primary outcomes as regard to echocardiographic parameters

Variables	No Composite outcome (n= 100)	Composite outcome (n= 50)	P-Value
TAPSE (cm)	1.65 (0.29)	1.48 (0.32)	0.039*
RVOT VTI (cm)	11.3 (2.2)	10.2 (1.9)	0.001*
LVOT VTI (cm)	16.7 (2.8)	15.1 (2.6)	0.019*
RVOT AT (ms)	71.5 (17.5)	65.0 (14.8)	0.069
RV tissue Doppler velocity (cm/s)	10.4 (2.2)	9.6 (2.0)	0.037*
RVFAC (%)	42.0 (10.5)	31.0 (12.0)	0.021*
LVEF (%)	60.2 (4.3)	58.1 (4.7)	0.254

Data is presented as mean (SD), *, statistical significance p value ≤ 0.05.

Table 6: 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
LVOT VTI	1.082	0.945 - 1.239	0.310
RVOT VTI	1.152	0.921 - 1.441	0.205
RVOT AT	1.022	1.001 - 1.044	0.043*
RV tissue Doppler velocity	1.054	1.003 - 1.108	0.038*
RVFAC	0.979	0.944 - 1.014	0.218
LVEF	0.992	0.964 - 1.021	0.608

PESI; pulmonary embolism severity index, LVOT; left ventricular outflow tract, VTI; velocity time integral, RVOT; right ventricular outflow tract, PASP; pulmonary arterial systolic pressure, *, statistical significance p value ≤ 0.05.

Conclusion

Uncontrolled HIV viremia and low CD4⁺ counts significantly increase cerebrovascular risk. Early ART initiation and management of traditional risk factors are essential to reduce stroke burden in HIV patients.

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

References

- Li SY, Zhang Y, Shen TT, Lu TT, Li X. Measuring of strain parameters reflects changes of right ventricular function before and after thrombolytic therapy in patients with acute pulmonary embolism. *Int J Cardiovasc Imaging*. 2022 Oct;38(10):2199-2208.
- Alerhand S, Sundaram T, Gottlieb M. What are the echocardiographic findings of acute right ventricular strain that suggest pulmonary embolism? *Anaesth Crit Care Pain Med*. 2021 Apr;40(2):100852.
- Alerhand S, Adrian RJ. What echocardiographic findings differentiate acute pulmonary embolism and chronic pulmonary hypertension? *Am J Emerg Med*. 2023 Oct;72:72-84.

4. Wang D, Fan G, Zhang X, Xi L, Chen Y, Li A, *et al.* Prevalence of long-term right ventricular dysfunction after acute pulmonary embolism: a systematic review and meta-analysis. *EClinicalMedicine*. 2023 Aug;62:102153.
5. Lyhne MD, Hansen JV, Andersen S, Schultz JG, Sørensen SG, Kirk ME, *et al.* Right ventricular to pulmonary artery coupling in chronic thromboembolic pulmonary hypertension. *Int J Cardiol*. 2025 Jan 1;418:132639.
6. Tzourtzos I, Lakkas L, Katsouras CS. Right ventricular longitudinal strain-related indices in acute pulmonary embolism. *Medicina (Kaunas)*. 2024 Sep 27;60(10):1586.
7. O'Corragain O, Alashram R, Millio G, Vanchiere C, Hwang JH, Kumaran M, *et al.* Pulmonary artery diameter correlates with echocardiographic parameters of right ventricular dysfunction in patients with acute pulmonary embolism. *Lung India*. 2023;40(4):306-311.
8. Paczyńska M, Sobieraj P, Burzyński Ł, Kostrubiec M, Wiśniewska M, Bienias P, *et al.* Tricuspid annulus plane systolic excursion (TAPSE) has superior predictive value compared to right ventricular to left ventricular ratio in normotensive patients with acute pulmonary embolism. *Arch Med Sci*. 2016 Oct 1;12(5):1008-1014.
9. Dahhan T, Siddiqui I, Tapson VF, Velazquez EJ, Sun S, Davenport CA, *et al.* Clinical and echocardiographic predictors of mortality in acute pulmonary embolism. *Cardiovasc Ultrasound*. 2016 Oct 28;14(1):44.
10. Schmid E, Hilberath JN, Blumenstock G, Shekar PS, Kling S, Sherman SK, *et al.* Tricuspid annular plane systolic excursion (TAPSE) predicts poor outcome in patients undergoing acute pulmonary embolectomy. *Heart Lung Vessel*. 2015;7(2):151-158.
11. Li Q, Zhang M. Echocardiography assessment of right ventricular-pulmonary artery coupling: Validation of surrogates and clinical utilities. *Int J Cardiol*. 2024 Jan 1;394:131358.
12. Chen X, Zhang P, Lou J, Zhao R, Zhang S, Xie M, *et al.* Application of an echocardiographic index to characterize right ventricular-pulmonary arterial coupling in heart failure. *ESC Heart Fail*. 2024 Jun;11(3):1290-304.
13. Lacerda Teixeira B, Albuquerque F, Santos R, Ferreira A, Carvalheiro R, Reis J, *et al.* Right ventricular myocardial work: proof-of-concept for the assessment of pressure-strain loops of patients with pre-capillary pulmonary hypertension. *Cardiovasc Ultrasound*. 2025 Jan 3;22(1):16.
14. Leidi A, Bex S, Righini M, Berner A, Groscurin O, Marti C. Risk stratification in patients with acute pulmonary embolism: current evidence and perspectives. *J Clin Med*. 2022 Apr 30;11(9):2533.
15. Ajah ON. Pulmonary embolism and right ventricular dysfunction: mechanism and management. *Cureus*. 2024;16(9):e70561.
16. Sorathia S, Almanzar A, Bhandiwad A, Nandar PP. Managing right ventricular failure in the setting of pulmonary embolism. *Cleve Clin J Med*. 2025 May 1;92(5):301-309.
17. Petit M, Vieillard-Baron A. Ventricular interdependence in critically ill patients: from physiology to bedside. *Front Physiol*. 2023;14:1232340.
18. Oh JK, Park JH. Role of echocardiography in acute pulmonary embolism. *Korean J Intern Med*. 2023 Jul;38(4):456-470.
19. Ucar EY. Update on thrombolytic therapy in acute pulmonary thromboembolism. *Eurasian J Med*. 2019 Jun;51(2):186-190.
20. Bartel B. Systemic thrombolysis for acute pulmonary embolism. *Hosp Pract (1995)*. 2015;43(1):22-27.

How to Cite This Article

Elsawah AM, Ghanem ML, Ali AA, Khairy H, Imam AM, Makram M, Omar R, Elhadainy MS. Echocardiographic right ventricular metrics and clinical outcomes in acute PE: Insights from a prospective cohort. *International Journal of Cardiology Sciences*. 2025;7(2):15-20

Creative Commons (CC) License

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.