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Tricuspid Regurgitation Peak Gradient /Tricuspid Annular Plane Systolic Excursion: A Novel Parameter for Risk Stratification in Normotensive Patients with Acute Pulmonary Embolism Using 2D Transthoracic Echocardiography

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Abstract

Background: Pulmonary embolism [PE] results from the occlusion of the pulmonary artery or its branches by a thrombus that has dislodged from a different anatomical location, thereby disrupting normal blood flow in the pulmonary circulation.

Aim of the work: This work was designed to evaluate the prognostic significance of a novel echocardiographic parameter, Tricuspid regurgitation peak gradient /tricuspid annular plane systolic excursion [TRPG/ TAPSE] in predicting in-hospital mortality among acute PE [APE] patients who are hemodynamically stable during their hospital stay.

Patients and Methods: Sixty patients [n=60] with a definitive diagnosis of APE were recruited in this prospective study executed at Al Azhar University Hospital and Mabarat El Asafra Hospital. Patients were divided into two groups: group [1] included patients who didn't achieve clinical endpoint and group [2] included patients who achieved clinical endpoint. The clinical endpoint was defined as the occurrence of at least one of the following: [1] need for cardiopulmonary resuscitation, [2] systolic blood pressure <90 mmHg for at least 15 minutes with signs of end-organ hypoperfusion, [3] need for intravenous catecholamines in vasopressor doses.

Results: Receiver operating characteristic [ROC] analysis revealed that the Area under Curve [AUC] for TAPSE in predicting the clinical endpoint in APE individuals was 0.834. TAPSE's cut-off value was determined as <13 mm, with an AUC of 0.834, 77.4% sensitivity, 85.7% specificity, 84.4% PPV, and 79.1% NPV. The cut-off value of TRPG as > 47 mmHg with AUC of 0.540, 54.7% sensitivity, 71.4% specificity, 65.7% PPV and 61.2% NPV. The TRPG/TAPSE ratio, expressed in mmHg/mm parameter was chosen as the next step for risk stratification. The optimal value of 4.8 mmHg/mm demonstrated 94.3% sensitivity, 100% specificity, 100% PPV and 94.6% NPV.

Conclusion: TRPG/TAPSE, a key echocardiographic parameter, is strongly correlated with clinical deterioration of non-high risk pulmonary embolism and hold potential for risk stratification and forecasting clinical deterioration of hemodynamically stable patients with APE.

Keywords: **Pulmonary Embolism; Acute; Tricuspid Regurgitation Peak Gradient; TAPSE.**

INTRODUCTION

When a thrombus from another site blocks blood flow in the pulmonary artery or its branches, it is known as a pulmonary embolism [PE]. Usually, this occurs when a piece of the thrombus breaks loose and passes through the pulmonary circulation. Rarely, materials other than thrombi, such as fat, air, or tumor cells, may embolize into the pulmonary circulation and cause PE [1].

Classifying PE based on hemodynamic stability is of utmost importance. Hemodynamically unstable PE, previously categorized as 'massive' or 'high-risk' PE, is identified by hypotension - specifically when systolic blood pressure falls below 90 mmHg, decreases by 40 mmHg or more from baseline, or requires vasopressor or inotropic support. It is important to note that the term 'massive' now refers to the hemodynamic impact rather than the physical size of the embolism. These patients face elevated mortality risk owing to obstructive shock, which frequently leads to high grade of failure in the left ventricle [2].

In contrast, hemodynamically stable PE includes a range from minor, minimally symptomatic or asymptomatic cases [classified as low-risk or small PE] to instances of mild hypotension that improve with fluid resuscitation, or cases with RV dysfunction [submassive or intermediate-risk PE] but without hemodynamic instability [3].

Timely identification of PE is paramount because of the considerable mortality and morbidity linked to the condition, which can be reduced with early therapeutic intervention. It is noteworthy that 30 % of untreated PE patients die, while the death rate is reduced to 8% with prompt treatment. Diagnosing PE is challenging, as in APE patients, it manifests as a range of unspecific clinical signs and symptoms [3].

Dyspnea, pleuritic chest pain, haemoptysis, cough, presyncope, and syncope are among the most prevalent symptoms of PE. Whereas Dyspnea is usually mild and temporary in small peripheral PE, it can be acute [APE] and severe in central PE. The only noticeable symptom in patients with a history of heart failure or pulmonary disease may be worsening Dyspnea. Also prevalent is chest pain, which is often brought on by pleural irritation from pulmonary infarction and distal emboli. Nevertheless, right ventricular [RV] ischemia in central PE can cause chest pain, which needs to be distinguished from aortic dissection or acute coronary syndrome [4]. Echocardiography serves as a reliable tool for identifying RV dysfunction. In normotensive APE patients, tricuspid annular plane systolic excursion [TAPSE] measurement can be utilized for risk stratification. An echocardiographic feature that can be utilized for risk stratification in APE is the tricuspid regurgitation peak gradient [TRPG], which is an indicator of RV overload [5, 6].

By positioning the cursor on the tricuspid annulus and assessing the longitudinal motion of the annulus at peak systole, TAPSE is measured in M-mode. Color Doppler was utilized to qualitatively evaluate tricuspid valve regurgitation, and a simplified Bernoulli's equation was utilized for calculating the peak gradient utilizing the peak velocity of the regurgitant tricuspid flow [7, 8].

THE AIM OF THE WORK

The study's objective was analysing the predictive value of TRPG/TAPSE as a prognostic echocardiographic marker for mortality during hospitalization in normotensive APE patients.

PATIENTS AND METHODS

Sixty participants [n=60] were recruited in this prospective study during the period between January 2024 to February 2025 with a confirmed diagnosis of APE during their hospital stay who presented to Al Azhar University Hospital.

The patients were subsequently stratified into two groups. Group [1] include patients who did not achieve clinical endpoint and group [2] included patients who achieved clinical endpoint.

Patients who reached the clinical endpoint, defined as the occurrence of at least one of the following: [1] need for cardiopulmonary resuscitation, [2] systolic blood pressure <90 mmHg for at least 15 minutes with signs of end-organ hypoperfusion, or [3] need for intravenous catecholamines in vasopressor doses.

Ethical consideration: Informed consent was obtained from all participants.

Inclusion criteria:

Patients 18 years of age or older, who presented with hemodynamic stability upon admission, defined as a SBP of ≥ 90 mmHg and the absence of peripheral hypoperfusion, and who had a confirmed diagnosis of APE based on CT pulmonary angiography.

Exclusion criteria:

Age below 18 years and patients with a history of chronic thromboembolic hypertension, end stage renal disease [ESRD], chronic obstructive pulmonary disease [COPD], primary and previous history of secondary tricuspid regurgitation, significant valvular heart disease, congenital heart disease, previous cardiac surgery, Idiopathic pulmonary arterial hypertension, right-sided cardiac devices.

All patients were subjected to the following:

Full history talking, general examination, local cardiac examination, vital signs including [temperature, heart rate, respiratory rate**,** oxygen saturation, non-invasive blood pressure [BP] measurement using standard sphygmomanometer and chest examination and laboratory investigations:[CBC, D-Dimer, Troponin, ABG, creatinine].

2D Transthoracic Echocardiography: Following diagnosis and admission, an echocardiographic evaluation was performed within 24 hours of presentation, and all outcomes were recorded digitally. Patient placement was in the left lateral decubitus. In the parasternal long-axis and RV-focused views, the right and left ventricles' dimensions was assessed at the level of the mitral and tricuspid valve tips during late diastole, as demonstrated by the R wave on the continuous ECG trace.

Using the Apical 4-Chamber [A4C] view, we measured RV/LV Ratio during end-diastole [when the ventricles are at their largest size], just before the mitral valve closes. Measure the basal diameter of RV: from the RV free wall to the interventricular septum, the LV: from the LV lateral wall to the interventricular septum.

Using continuous-wave Doppler echocardiography, the tricuspid valve peak systolic velocity was measured. The simplified Bernoulli equation was then used to calculate TRPG.

TAPSE, which measures the systolic excursion distance [in mm] of the RV annular segment along its longitudinal axis in a standard apical 4-chamber view, was used to measure RV function using M-mode.

We measured the 60/60 Sign which is a combination of Pulmonary Valve Acceleration Time [PVAT] and Tricuspid Regurgitation [TR] Pressure Gradient.

We obtained the parasternal short-axis view at the level of the aortic valve or RV outflow tract [RVOT]. then we Placed the Doppler sample volume just before the pulmonary valve and measured the time from onset of flow to the peak velocity.

We included McConnell's sign defines as reduced motion of the mid-right ventricular free wall with preserved apical contraction, observed using the apical four-chamber view.

Radiological:

Chest X-Ray was done, and computed tomographic pulmonary angiography [CTPA], which provides direct visualization of emboli within the pulmonary arterial system for confirmation of PE diagnosis. CTPA findings in PE include central or eccentric intraluminal filling defects within the pulmonary arteries, with complete vessel occlusion often presenting as a "cut-off" sign. Pulmonary artery enlargement may be observed, reflecting elevated pulmonary pressures, along with peripheral wedge-shaped opacities suggestive of pulmonary infarction.

Data analysis: The collected data was anonymized and fed to personal computer. The SPSS software computer package was used to carry out all statistical analyses. Continuous data were expressed by their median and interquartile range [IQR], besides the calculation of minimum and maximum values. On the other side, the categorical data were expressed by their frequency and percentages. Independent samples student "t" test, Mann Whitney, Chi square or Fisher exact tests were used to compare between groups according type of data and statistical situation. Receiver operation characteristic [ROC] curve was built to determine the prediction power of TAPSE in the clinical endpoints of APE. P value < 0.05 was considered significant.

RESULTS

The demographic characteristics including gender, age and BMI showed no significant difference between the two groups [p>0.05] [**Table 1].**

There were significant differences between the two groups in terms of D-dimer, high-sensitive troponin, and creatinine levels. However, haemoglobin levels did not differ significantly among the groups [**Table 2].**

There were significant differences between the two groups in heart rate, hypoxia, blood pressure, and sPESI score. Group 2 exhibited higher heart rates, a higher prevalence of hypoxia, and lower systolic and diastolic blood pressures compared to Group 1. Group 2 had a greater proportion of patients with more severe sPESI scores [2 and 3] [**Table 3].**

There were significant differences between the two groups in multiple echocardiographic parameters. Group 2 had worse RV function, indicated by higher TRPG/TAPSE ratios, peak TR velocity, and estimated SPAP. Additionally, Group 2 showed a higher prevalence of McConnell's sign, which is associated with APE. No significant differences were found in TRPG, PAAT, or 60/60 sign, suggesting similar values in these aspects between the groups. Overall, Group 2 presented with more severe PE features [Table 4].

Receiver operating characteristic [ROC] analysis indicated that the Area under the Curve [AUC] for TAPSE in predicting clinical outcomes in APE patients was 0.834. The cut-off value for TAPSE was established at <13 mm, yielding an AUC of 0.834, with 77.4% sensitivity, 85.7% specificity, 84.4% PPV, and 79.1% NPV. For TRPG, the cut-off value was set at > 47 mmHg, with an AUC of 0.540, 54.7% sensitivity, 71.4% specificity, 65.7% PPV, and 61.2% NPV. As a secondary step in echocardiographic risk stratification, the TRPG/TAPSE ratio expressed in mmhg/mm parameter was chosen, with an optimal value of 4.8, providing 94.3% sensitivity, 100% specificity, 100% PPV, and 94.6% NPV [**Table 5]**

ROC analysis demonstrated that TAPSE predicted clinical endpoints in APE patients with an AUC of 0.834 at a cut-off <13 mm [sensitivity 77.4%, specificity 85.7%, PPV 84.4%, NPV 79.1%]. TRPG > 47 mmHg showed lower predictive value [AUC 0.540, sensitivity 54.7%, specificity 71.4%, PPV 65.7%, NPV 61.2%]. The TRPG/TAPSE ratio, with a cut-off >4.8, yielded superior performance [AUC not stated, sensitivity 94.3%, specificity 100%, PPV 100%, NPV 94.6%]. Its AUC was significantly higher than that of TAPSE [p=0.003] and TRPG [p=0.002] [**Figure 1**].

Table [1]: Demographic characteristics among the two studied groups.

		Group [1] [N=53]		Group [2] [N=7]		Test value	P-value
		No.	%	No.	%		
Gender	**Male**	20	37.7%	5	71.4%	X^2= 2.888	0.117^{FET}
	Female	33	62.3%	2	28.6%		
Age [Years]	**Median [IQR]**	50 [40- 65]		65 [48- 66]		Z_{MWU} = 1.017	0.309
	Range	22 - 83		40 - 69			
BMI [Kg/m^2]	**Median [IQR]**	24.6 [23- 27.7]		22 [22- 25]		Z_{MWU} = 1.587	0.112
	Range	21 – 29.7		22 – 30.2			

BMI: body mass index, IQR: Inter-quartile range, P >0.05: Not significant, P <0.05 is significant, SD: standard deviation, X^2: Chi- Square test, ^{Z}MWU: Z value of Mann Whitney U test.

Table [2]: Comparison among the studied groups with regard to laboratory results of PE presentation

	Group [1] [N=53]					Group [2] [N=7]					Test value	P-value
	Median	IQR		Min.	Max.	Median	IQR		Min.	Max.		
D. dimer [mg/l]	1765	917	4227	322	10000	5798	1844	6550	1808	6550	Z_{MWU} = 2.327	**0.020**
High-**sensitive troponin [ng/dl]**	40	14.3	150	3	965	633	423	104	118	1309	Z_{MWU} = 0.578	**<0.001**
Creatinine [mg/dl]	1.1	0.89	1.3	0.4	3.45	1.7	1.48	2.2	0.9	2.35	Z_{MWU} = -2.4	**0.015**
Hemoglobin [g/dl]	11.8	10.1	13.5	7.4	15	12	11	12.3	9	12.7	Z_{MWU} =0.39	0.69

SD: standard deviation, sPESI: Simplified Pulmonary Embolism Severity Index IQR: interquartile range.

Table [3]: Comparison among the studied groups with regard to clinical data of PE presentation

		Group [1] [N=53]					Group [2] [N=7]					Test value	P-value
		Median	IQR		Min.	Max.	Median	IQR		Min.	Max.		
Heart rate [beats/min.]		120	113	127	109	135	135	131	135	123	135	Z_{MWU}=3.396	**<0.001**
Hypoxia		18 [34%]					7 [100%]					X^2=2.888	**0.001**
Systolic blood pressure [mmHg]		130	110	130	90	220	90	90	110	85	110	Z_{MWU}=0.578	**<0.001**
Diastolic blood pressure [mmHg]		65	60	80	60	120	60	55	60	50	65	Z_{MWU}=0.578	**0.003**
sPESI score	**1**	36 [67.9%]					0 [0.0%]					X^2= 17.71	**<0.001**
	2	15 [28.3%]					4 [57.1%]						
	3	2 [3.8%]					3 [42.9%]						

IQR: Inter-quartile range, sPESI: simplified pulmonary embolism severity index p<0.01 is highly significant [HS], X^2: Chi- Square test, ^{Z}MWU: Z value of Mann Whitney U test, SD: standard deviation.

Table [4]: Comparison between the studied groups regarding Echocardiographic data

	Group [1] [N=53]					Group [2] [N=7]					Test value	P-value
	Median	IQR		Min.	Max.	Median	IQR		Min.	Max.		
TRPG/TAPSE	2.70	1.60	4.30	0.60	6.00	6.00	4.80	6.00	4.50	6.40	Z_{MWU} = 3.795	**<0.001**
TRPG [mmHg]	50.0	37.0	55.0	13.0	77.0	50.0	36.0	58.0	25.0	60.0	Z_{MWU} = 0.473	0.636
TAPSE [mm]	16.0	14.0	20.0	11.0	26.0	12.0	11.0	12.0	11.0	15.0	Z_{MWU} = 3.145	**0.002**
Peak TR velocity [m/s]	3.4	3	3.7	1.8	4	4	3.8	4.3	3.7	4.4	Z_{MWU} = 3.676	**<0.001**
Estimated SPAP [mmHg]	57	40	67	21	86	84	68	89	42	92	Z_{MWU} = 3.117	**0.002**
PAAT [ms]	58	55	93	42	122	55	49	83	49	98	Z_{MWU} = 1.492	0.136
McConnell's sign	5 [9.4%]					7 [100%]					X^2= 31.7	**<0.001**
IVC [mm]	22	20	26	4	31	30	20	31	20	31	Z_{MWU} = 2.079	**0.038**
60/60 sign	25 [47.2%]					2 [28.6%]					X^2= 0.864	0.442
RV/LV	1.1	0.8	1.1	0.7	1.2	1.2	1.2	1.2	1.1	1.2	Z_{MWU} = 3.144	**0.002**

IVC: inferior vena cava, IQR: interquartile range, LVEF: left ventricular ejection fraction, PAAT: Pulmonary artery acceleration time, P-value: prognostic value, PE: pulmonary embolism, RV: right ventricle, LV: left ventricle, SD: standard deviation, SPAP: systolic pulmonary artery pressure, TRPG: tricuspid regurgitant peak velocity, TAPSE: tricuspid annular peak systolic excursion.

Table 5: Validity of TRPG, TAPSE and TRPG/ TAPSE ratio in prediction of mortality in APE patients

Parameter	Cutoff value	AUC	Sensitivity	Specificity	PPV	NPV	P value	Asymptotic 95% Confidence Interval	
								Lower Bound	Upper Bound
TRPG/ TAPSE	4.8	0.992	94.3%	100%	100%	94.6%	**<0.001**	0.943	0.997
TAPSE	13	0.834	77.4%	85.7%	84.4%	79.1%	**<0.001**	0.753	0.877
TRPG	47	0.540	54.7%	71.4%	65.7%	61.2%	0.789	0.553	0.707

PPV: Positive Predictive Value, NPV: Negative Predictive Value, AUC: Area Under Curve. SD: standard deviation, IQR: interquartile range, TRPG: Tricuspid regurgitant peak velocity, TAPSE: Tricuspid annular peak systolic excursion.

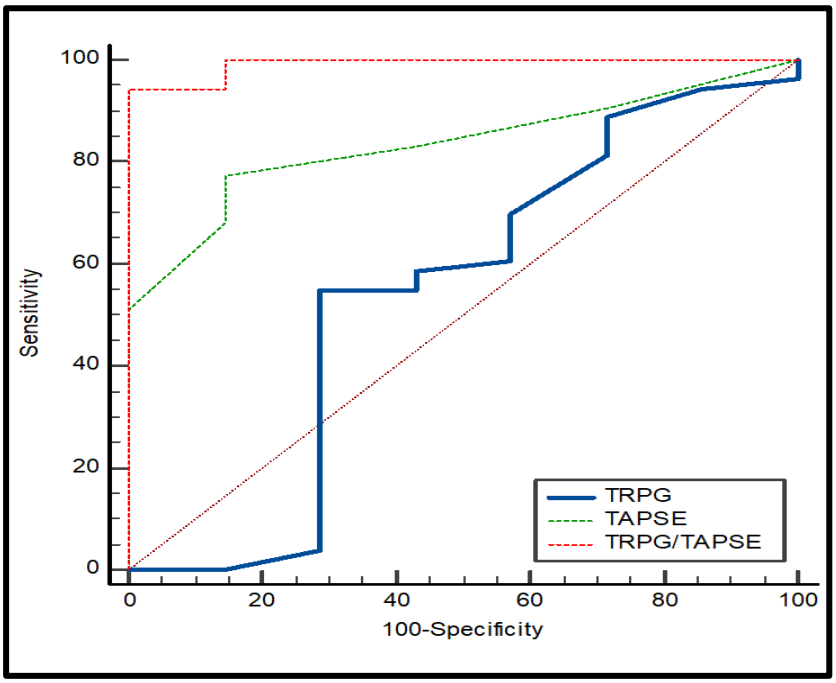

Figure [1]: ROC curve of TRPG, TAPSE and TRPG/ TAPSE ratio in prediction of clinical endpoint in APE patients

DISCUSSION

The study showed that clinical endpoints were achieved in seven cases [11.7%]. Regarding demographic characteristics including gender, age and BMI revealed insignificant difference between the two groups: group [1] include patients who did not achieve clinical endpoint [N=53], and group [2] included patients who achieved clinical endpoint [N=7] with p value > 0.05. Our investigations are in accordance with those of

El-Morshedy *et al.* [9] who estimated the prognostic significance and accuracy of TRPG, TAPSE, and pulmonary artery acceleration time for risk stratification in moderate-risk PE. The research included [n=60] normotensive patients [24 men and 36 women] with moderate-risk PE, using a simplified PE severity index [sPESI] exceeding 1.

Concerning comparison between the studied groups regarding clinical and laboratory PE presentation, the present study showed that patients who did not achieve clinical endpoint had significantly lower [D. dimer], heart rate and highly sensitive troponin than patients with achieved clinical endpoint. Hypoxia was notably elevated in patients who reached the clinical endpoint [p < 0.001]. In addition, Patients who did not achieve clinical endpoint had significantly higher systolic and diastolic BP than patients with achieved clinical endpoint.

Concerning comparison between the studied groups regarding Echocardiographic data, the present study demonstrated that patients in group [2] had significantly lower TAPSE, higher TRPG/TAPSE, higher Peak TR velocity, higher estimated SPAP, greater IVC, and McConnell's sign more often. They also had a higher RV/LV ratio compared with those in group [1]. In agreement with the current study, **Ciurzyński *et al.*** [10] explored the TRPG/TAPSE ratio's prognostic significance in estimating APE-related mortality or the requirement for rescue thrombolysis in normotensive APE patients. Their study encompassed four hundred non-high-risk APE cases, comprising 298 patients with intermediate-risk APE [58 classified as intermediate-low risk and 240 as intermediate-high risk] and 102 patients with low-risk APE.

The results demonstrated that patients with TRPG/TAPSE greater than 4.5 mmHg/mm had significantly higher TRPG and TRPG/TAPSE values, along with significantly lower TAPSE and AcT, compared to those with TRPG/TAPSE equal 4.5 mmHg/mm or less.

Similarly, **Pruszczyk *et al.*** [12] found that a TAPSE measurement of less than 16 mm was a useful predictor of clinical events in patients with submissive PE, while an RV/LV ratio greater than 1 showed a lower predictive value. The TAPSE value above 20 was associated with a remarkably high likelihood of not experiencing adverse events, while the RV/LV ratio greater than one was also associated with a high likelihood of not having complications.

Consistent with our study, **El-Morshedy *et al.*** [9] demonstrated that RVD, the RVD/LVD ratio, TR velocity, and TRPG/TAPSE were significantly greater in group [2] patients, whereas TAPSE and acceleration time were substantially lower in the same group.

According to validity of TRPG, TAPSE and TRPG/ TAPSE ratio in predicting the clinical endpoint in APE patients, the present study indicated that ROC analysis illustrated that the AUC for TAPSE in predicting the clinical endpoint in APE patients was 0.834. We established the TAPSE cut-off value at < 13 mm, which yielded an AUC of 0.834, with 77.4% sensitivity, 85.7% specificity, 84.4% PPV, and 79.1% NPV. The cut-off value of TRPG as >47 with AUC of 0.540, 54.7% sensitivity, 71.4% specificity, 65.7% PPV and 61.2% NPV. We selected the TRPG/TAPSE parameter as the second echocardiographic step for risk stratification. With 94.3% sensitivity, 100% specificity, 100% PPV, and 94.6% NPV, the TAPSE cut-off value is 4.8.

As observed in the current study, **Ciurzyński *et al.*** [10] reported that ROC analysis revealed an AUC of 0.94 [95% CI 0.8–1.0] for TAPSE in predicting clinical endpoints [CE]. They established the TAPSE cut-off value at >20 mm, which provided a 100% negative predictive value [NPV] for CE. As a result, all patients [193 patients, 48.25%] with TAPSE >20 mm were categorized as low-risk and had a positive

prognosis. In 207 patients with TAPSE ≤ 20 mm, 8 cases of CE [3.9%] were identified.

To further assess risk, we used the TRPG/TAPSE ratio as an additional echocardiographic measure. The analysis showed that the TRPG/TAPSE ratio had a notably higher diagnostic value compared to either TAPSE or TRPG alone. The difference between the TRPG/TAPSE ratio and TRPG alone was significant, as was the difference between the TRPG/TAPSE ratio and TAPSE alone. In this study population, TRPG/TAPSE greater than 4.5 mmHg/mm had a high negative predictive value, indicating that most patients with this ratio were unlikely to experience adverse events. Additionally, complications occurred more frequently in patients with a TRPG/TAPSE ratio above 4.5 mmHg/mm compared to those with a lower ratio. Through ROC analysis, TAPSE of 14 mm was identified as the optimal point with the highest sensitivity and specificity. In univariable Cox analysis, TRPG/TAPSE and TAPSE were the only significant predictors of clinical outcomes, showing a strong association with the likelihood of adverse events.

In accordance with **Kurnicka *et al.*** [11] sought to directly compare the prognostic value of the RV/LV ratio, TAPSE, and TRPG/TAPSE for complicated clinical courses comprising 490 patients [229F], 64±18 years old: rescue thrombolysis, hemodynamic collapse, or in-hospital APE-related mortality.

TAPSE Sensitivity was 52%, Specificity was 85%, PPV was 18%, NPV was 96%. RV/LV Sensitivity was 74%, Specificity was 63% PPV was 12% NPV was 95% and TRPG/TAPSE Sensitivity was 10% Specificity was 94% PPV was 10% NPV was 94%.

In contrast to our study, **El-Morshedy *et al.*** [9] found that TAPSE at a cutoff point <1.7 cm had 100% sensitivity and 100% specificity for predicting intermediate-high-risk APE with 100% accuracy and the area under the curve was 1. TRPG/TAPSE ratio had 79% sensitivity and 94% specificity for predicting intermediate- high-risk APE with 86.9% accuracy and the area under the curve was 0.89. This difference returned to the classification of patients into intermediate low-risk and intermediate high-risk pulmonary embolism.

This study was limited by a small sample size and single-centre design, which may affect the generalizability of the findings. Additionally, the absence of long-term follow-up precludes assessment of delayed outcomes.

Conclusions: We concluded from our study that TRPG/TAPSE, an echocardiographic parameter, is strongly correlated with clinical deterioration of non-high risk pulmonary embolism and can be used for echocardiographic risk stratification and prediction of mortality in normotensive patients with acute pulmonary embolism.

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