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Can systolic pulmonary artery pressure be used as one of the criteria of risk stratification in acute pulmonary embolism?

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SOUHRN

Kontext: Při akutní plicní embolii (PE) hrají významnou roli rychlé stanovení diagnózy a naplánování léčby podle klasifikace rizika úmrtí.
Cíle: Posoudit přínos znalosti hodnot systolického tlaku v plicnici (systolic pulmonary artery pressure, sPAP) při stratifikaci rizika pacientů s akutní PE.
Metody: Naše studie byla retrospektivní, průřezová. Bylo do ní zařazeno celkem 221 pacientů přijatých do naší nemocnice s diagnózou akutní PE, stanovenou CT angiografií plicnice (computed tomography pulmonary angiography, CTPA). Všichni pacienti byli vyšetřeni echokardiograficky (ECHO) a hodnota sPAP byla vypočítána pomocí Bernoulliho rovnice. Vztahy mezi proměnnými, jako jsou stratifikace rizika podle Evropské kardiologické společnosti (European Society of Cardiology, ESC), dysfunkce pravé komory (right ventricular dysfunction, RVD), hodnota troponinu T a sPAP, byly posuzovány pomocí Spearmanových korelačních koeficientů.
Výsledky: Mezi hodnotami sPAP na jedné straně a hodnotami troponinu T, RVD a skupinami s vysokým a středně vysokým rizikem podle ESC na straně druhé byla nalezena těsná pozitivní lineární korelace (Spearmanův korelační koeficient r = 0,615; resp. 0,798; 0,411 a 0,408; $p < 0,001$). Jako optimální mezní hodnota pro sPAP byla stanovena hodnota 41,5 mm Hg s celkovou přesností 0,961 (95% CI 0,937–0,984). Kromě toho se prokázalo, že při mezní hodnotě 41,5 mm Hg dokáže sPAP účinně vyhledávat přítomnost RVD a rizikové skupiny podle ESC ($p < 0,0001$). Naše studie prokázala 85% senzitivitu a 92% specificitu sPAP při vyhledávání RVD nad mezní hodnotou 41,5 mm Hg.
Závěry: I když nelze sPAP považovat za spolehlivý parametr při vyšetření pacientů s akutní PE metodou ECHO, potvrdily výsledky naší studie, že sPAP může představovat užitečný parametr při stratifikaci rizika akutní PE, a hodnotu sPAP by tak bylo vhodné zařadit mezi kritéria RVD ve stratifikaci rizika akutní PE.

Keywords:
Echocardiography
Pulmonary embolism
Right ventricular dysfunction
Risk stratification
Systolic pulmonary artery pressure

ABSTRACT

Background: In acute pulmonary embolism (PE), rapid diagnosis and treatment planning according to mortality risk classification are important.
Objectives: To explore the effectiveness of systolic pulmonary artery pressure (sPAP) values in the risk stratification of the patients with acute PE.
Methods: This study is a retrospective, cross-sectional clinical trial design. A total of 221 patients who were admitted to our hospital and diagnosed as acute PE by thorax computed tomography pulmonary angiography (CTPA) was included in the study. All patients were evaluated by echocardiography (ECHO) and sPAP was calculated by Bernoulli equation. The relationships between variables such as the European Society of Cardiology (ESC) risk stratification, right ventricular dysfunction (RVD), troponin T, and sPAP levels were examined with Spearman's correlation coefficients.
Results: A strong positive linear correlation was found between sPAP, and troponin T, RVD, ESC high-risk, intermediate-high risk groups (Spearman's r = 0.615, 0.798, 0.411, 0.408, $p < 0.001$, respectively). The optimal cut-off value for sPAP was found as 41.5 mmHg with an overall accuracy of 0.961 (95% CI: 0.937–0.984). Besides, at the cut-off value of 41.5 mmHg, sPAP was found effective in determining RVD and ESC risk groups ($p < 0.0001$). Sensitivity and specificity of sPAP was found as 85%, 92%, respectively in detecting RVD above 41.5 mmHg cut-off value.
Conclusions: Although sPAP is not considered as a reliable finding of ECHO in the patients with acute PE, our study results confirmed that it can be valuable in the risk stratification of acute PE. It would be useful to include sPAP to RVD criteria for acute PE risk stratification.

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Introduction

Mortality risk stratification of the patients with pulmonary embolism (PE) is the main point in the selection of treatment, especially for the decision of thrombolytic therapy. Both European Society of Cardiology (ESC) 2014 and ESC 2019 guidelines of pulmonary embolism are suggesting risk stratification while managing patients with pulmonary embolism.[1,2] At early stage, it is critical to identify patients with high-risk PE. Hemodynamics of the patients, Pulmonary Embolism Severity Index (PESI), echocardiographic (ECHO) findings of right ventricular dysfunction (RVD) and cardiac markers as troponin are the main factors effecting this classification. Simplified PESI (sPESI) includes the data about age, chronic cardiopulmonary disease, cancer, pulse rate, blood pressure and oxygen saturation, so these clinical findings also take a role while managing the treatment and hospitalization of the patient. Troponin is a marker increasing in myocardial injury. Therefore, if it is high in a patient with PE, it mostly shows the right heart ischemia and coming infarction. The patients who have ECHO findings of RVD, sPESI ≥1 and high troponin levels are considered as high risk in intermediate group.[2] These PE patients may have thrombolytic therapy if necessary.[2–4] Computed tomography pulmonary angiography (CTPA) is also mentioned as a right ventricle evaluating method besides ECHO in the last ESC guideline.[2]

As it is known after acute pulmonary embolism according to the dimension of obstruction; pulmonary artery pressure increases and then right ventricle dilatation develops. Therefore, we planned our study with the suggestion of that pulmonary artery pressure may also be a risk-determining criterion such as other ECHO findings.

Methods

Patient selections and study design

This study has a retrospective, cross-sectional clinical trial design. A total of 221 patients who were admitted to our hospital and diagnosed as acute PE by thorax CTPA were included in the study. Patients with a hematological disease, patients under the age of 18, pregnant women, and patients whose ECHO and cardiac marker data could not be found were excluded from the study. Heart failure (heart failure with reduced ejection fraction [EF], heart failure with mid-range EF and heart failure with preserved EF), moderate to severe heart valve diseases, and congenital or acquired cardiovascular diseases can cause postcapillary pulmonary hypertension (PH). Therefore, patients with a previous history of severe heart disease that may be associated with PH and a previous history of PH have been excluded as this may affect baseline pulmonary artery pressure and study result. The study was approved by the Institutional Local Ethics Committee and it was conducted in accordance with the Declaration of Helsinki. The requirement of informed consent was waived, as this was a retrospective study.

ECHO findings were recorded. ECHO measurements were taken with Philips IE33 (Istanbul, Turkey) device. Four cavities of the heart were examined at the end of the expiration in the apical window while the patients were in the left decubitus position. Systolic pulmonary artery pressure (sPAP) was calculated by adding the tricuspid regurgitation (TR) peak gradient to estimate right atrium pressure (RAP) (simplified Bernoulli equation; $sPAP = 4 \times (TRvel)^2 + RAP$). Systolic PAP was calculated by Bernoulli equation from the peak velocity obtained by dropping continuous wave Doppler on the tricuspid valve insufficiency jet and the measurements were recorded by averaging three consecutive beats. While measuring sPAP in echocardiography performed on patients diagnosed with pulmonary embolism, the estimated right atrial pressure values were used as recommended by current guidelines. Echocardiographic findings of right ventricular end-diastolic diameter ≥30 mm or right ventricular end-diastolic diameter to left ventricle ≥1 were considered as RVD findings. Septum deviation and sPAP were recorded. Flattening of the interventricular septum, paradoxical movement and left deviation (D-septum) were also examined in the parasternal short axis and four apical spaces. Echocardiography was performed on all patients in the same clinic and by cardiologists who applied the same evaluation criteria.

Thorax CTPA findings also recorded since all the patients were diagnosed as pulmonary embolism by CTPA. Demographic findings and troponin T were also recorded. Troponin T levels (Roche Cobas 6000, Tokyo, Japan) were measured by using commercial kit. The normal range of troponin T was 0–14 pg/ml.

Patients were divided into two groups according to the existence of RVD findings by the evaluation with ECHO. Demographic and clinic characteristic findings were compared between the groups.

Statistical analysis

The statistical analysis was performed with the SPSS Statistics software package, version 21.0 (IBM Corporation, Armonk, NY, USA). The distribution of the continuous variables was examined by both Shapiro-Wilk's test and normality plots. Continuous and discrete variables were summarized by median (minimum–maximum). Frequency (%) was given for categorical variables. Differences between groups were assessed with the Mann–Whitney U test for continuous nonparametric variables, and Chi-square test or Fisher exact test for nominal variables. We performed univariate and multivariate logistic regression analyses to identify the risk factors for RVD. In the multivariate logistic regression models, backward logistic regression was used and the criterion for inclusion of the candidate variables in the model was a $p < 0.25$. The relationships between variables such as the ESC risk stratification, troponin T, and sPAP level were examined with Spearman's correlation coefficients. The diagnostic performances of parameters such as sPAP and troponin T were compared by receiver operating characteristic (ROC) curve analysis. The results were summarized in terms of the area under the curve (AUC), with 95% confidence intervals and standard errors. The optimal cut-off points were calculated by using the Youden's index. Values of $p < 0.05$ were considered statistically significant.

Results

Two hundred twenty-one patients with pulmonary thromboembolism were included in the study. Of all the patients, 116 (52.5%) were female, 105 (47.5%) were male, and the mean age was 59.8±17.9 years. PE cases were divided into two groups as those with RVD and those without RVD. One hundred twenty-one (54.8%) PE patients had RDV. There was a difference between the groups in terms of age and gender distribution (p <0.0001, p = 0.004, respectively). The history of cardiovascular disease and chronic neurological disease was significantly higher in the group with RVD (p <0.001). While the median sPAP was 55 (30–100) mmHg in the group with RVD, it was 30 (20–65) mmHg in the group without RVD. Systolic PAP in RVD group was significantly higher than the group without RVD (p <0.0001) (Table 1).

Univariate and multivariate logistic regression models were used to determine the risk factors affecting the RVD. Age, gender, sPAP, and troponin T were found to be the factors affecting RVD in the univariate logistic regression model (p <0.0001, p = 0.005, p <0.0001, p <0.0001, respectively). There was a significant relationship between cardiovascular disease, chronic neurological disease, and RVD (p <0.0001, p = 0.006, respectively). Multivariate logistic regression analysis showed that both sPAP and troponin T values were predictors of RVD. Systolic PAP was found to be more effective than troponin T in predicting RVD (p <0.0001, p = 0.005, respectively) (Table 2).

Spearman's correlation analysis was performed to examine simple correlations between sPAP measured in ECHO and RVD, troponin T levels, ESC risk groups. A strong positive linear correlation was found between sPAP, and troponin T (Fig. 1), RVD, ESC high-risk, intermediate-high risk groups (Spearman's r = 0.615, 0.798, 0.411, 0.408, p < 0.001, respectively). Also negative linear correlation was found between sPAP and ESC intermediate-low, low-risk groups (Spearman's r = –0.249, –0.0586, p <0.001, respectively).

The predictive values for the diagnosis of RVD were calculated by ROC analysis for sPAP and troponin T. The optimal cut-off for sPAP was 41.5 mmHg according to Youden's index. The AUC value for sPAP was 0.961 (95% CI: 0.937–0.984, p <0.0001), which was better than the AUC value of troponin T (Table 3, Fig. 2).

We regrouped our cases based on the cut-off value of 41.5 mmHg for sPAP (Table 4).

Table 1 – Comparison of demographic and clinic characteristic findings of PE patients with and without RVD

	PE patients with RVD	PE patients without RVD	p-value
Number of patients, n (%)	121 (54.8%)	100 (45.2%)	
Age at event (years)	65.63±15.98 69 (20–90)	52.75±17.65 52.50 (21–94)	**<0.0001**
Gender, n (%) Female Male	 74 (61.2%) 47 (38.8%)	 42 (42%) 58 (58%)	**0.005**
Smoking history, n (%) Smokers Non-smokers Exsmokers	 27 (22.3%) 74 (61.2%) 20 (16.5)	 40 (40%) 52 (52%) 8 (8%)	**0.008**
Co-morbidities, n (%) Chronic pulmonary disease Cardiovascular disease Chronic neurological disease Previous PE history	 20 (16.5%) 65 (53.7%) 18 (14.9%) 18 (14.9%)	 8 (8%) 23 (23%) 1 (1%) 8 (8%)	 0.09 **<0.0001** **<0.001** 0.171
DVT, n (%)	72 (59.5%)	45 (45%)	0.032
D-dimer (ng/mL) Median	 5268.5 (343–10000)	 3957 (323–10500)	 0.024
Troponin T (pg/mL) Median	 64 (1–610)	 6 (1–310)	 **<0.0001**
sPAP, mmHg Median	 55 (30–100)	 30 (20–65)	 **<0.0001**
D-septum, n (%)	32 (%26.45)	0	**<0.0001**
Type of embolism, n (%) ESC high risk ESC intermediate–high risk ESC intermediate–low risk ESC low risk	 38 (31.4%) 58 (47.9%) 23 (19%) 2 (1.6%)	 4 (4%) 15 (15%) 33 (33%) 48 (48%)	**<0.0001**

DVT – deep vein thrombosis; ESC – European Society of Cardiology; PE – pulmonary embolism; RVD – right ventricular dysfunction; sPAP – systolic pulmonary artery pressure.

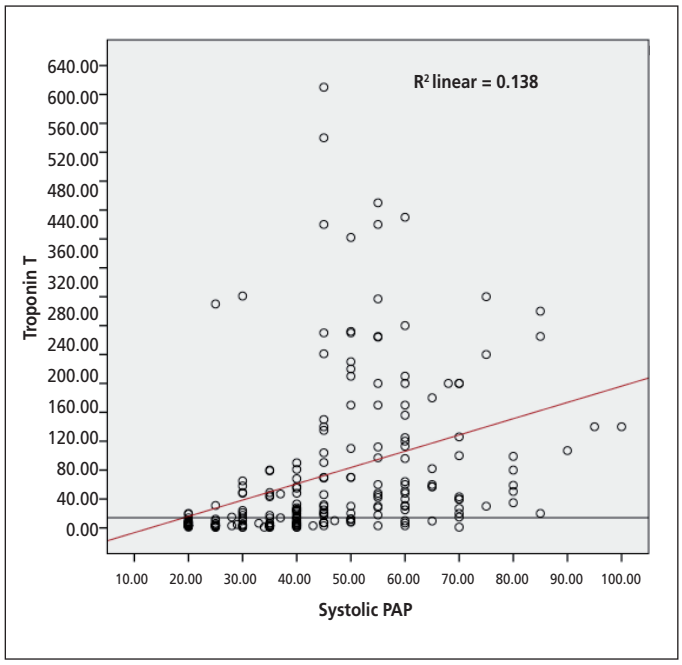

Fig. 1 – Correlation between sPAP and troponin T.

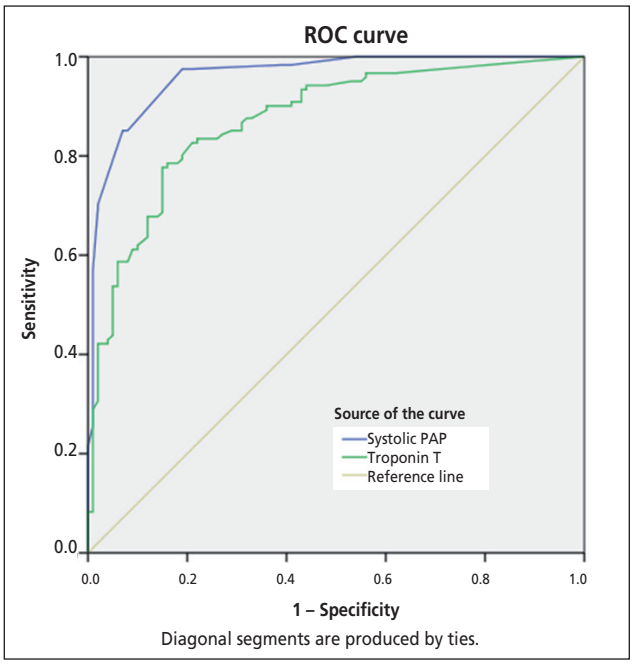

Fig. 2 – ROC curve analysis of sPAP and troponin T for the diagnosis RVD.

Table 2 – Results of the univariate and multivariate logistic regression analyses of the potential predictors of RVD

Variable	ß	SE	OR	95% CI	*p*-value
Univariate analysis					
Age	0.044	0.009	0.957	0.941–0.973	**<0.0001**
Gender	0.777	0.275	0.460	0.268–0.789	**0.005**
sPAP (mmHg)	0.263	0.039	0.769	0.712–0.830	**<0.0001**
Troponin T (pg/mL)	0.031	0.006	0.970	0.959–0.981	**<0.0001**
Chronic pulmonary disease	0.823	0.442	0.439	0.184–1.045	0.063
Cardiovascular disease	1.357	0.300	0.257	0.143–0.463	**<0.0001**
Chronic neurological disease	2.851	1.037	0.058	0.008–0.441	**0.006**
Previous PE history	0.698	0.448	0.498	0.207–1.198	0.128
Multivariate analysis					
Cardiovascular disease	0.976	0.522	0.377	0.136–1.047	0.061
sPAP (mmHg)	0.247	0.042	0.781	0.720–0.848	**<0.0001**
Troponin T (pg/mL)	0.013	0.005	0.987	0.978–0.996	**0.005**

PE – pulmonary embolism; sPAP – systolic pulmonary artery pressure.

Table 3 – The predictive values for the diagnosis of RVD of sPAP and troponin T

	AUC (%95)	Cut-off	*p*-value	Sensitivity (%)	Specificity (%)
sPAP (mm Hg)	0.961 (0.937–0.984)	41.5	**<0.0001**	85.1	92
Troponin T (pg/mL)	0.872 (0.826–0.919)	20.1	**<0.0001**	80.2	81

AUC – area under the curve; sPAP – systolic pulmonary artery pressure.

Table 4 – The comparison of ECHO findings, troponin T, and ESC mortality risk groups according to the cut-off value of sPAP: 41.5 mmHg

	ECHO RVD n = 121	ECHO D-septum n = 32	Troponin T (pg/mL) n = 221	ESC high risk n = 42	ESC intermediate–high risk n = 73	ESC intermediate–low risk n = 56	ESC low risk n = 50
	n (%)	n (%)	Median (min–max)	n (%)	n (%)	n (%)	n (%)
sPAP ≥41.5 mmHg (n = 111)	103 (85.1%)	28 (87.5%)	70 (1–610)	39 (92.9%)	57 (78.1%)	15 (26.8%)	0
sPAP <41.5 mmHg (n = 110)	18 (14.9%)	4 (12.5%)	6.4 (1-321)	3 (7.1%)	16 (21.9%)	41 (73.2%)	50 (100%)
***p*-value**	**<0.0001**	**<0.0001**	**<0.001**	**<0.0001**	**<0.0001**	**<0.0001**	**<0.0001**

ECHO – echocardiography; ESC – European Society of Cardiology; RVD – right ventricular dysfunction; sPAP – systolic pulmonary artery pressure.

Discussion

The importance of mortality risk assessment in pulmonary embolism has been emphasized especially in the last two ESC guidelines. In the last guideline, it is recommended to make risk assessment with ECHO right ventricular dilatation findings, troponin values and PESI. Patients with massive (high-risk) PE require immediate intervention including thrombolytic and thrombectomy, with or without mechanical hemodynamic support. Normotensive patients are further risk stratified using clinical scores such as the PESI[5] and its simplified version (sPESI),[6] biomarkers, and imaging modalities that detect right ventricle (RV) strain. The presence of RV strain by biomarkers indicates a high-risk, submassive PE and portends worse prognosis, which may need more aggressive treatment.[7]

RV dysfunction findings evaluated by ECHO in ESC guideline are described as: A. enlarged right ventricle in parasternal long-axis view, B. dilated RV with basal RV/LV ratio >1.0, and McConnell sign in four-chamber view, C. flattened intraventricular septum in parasternal short-axis view, D. distended inferior vena cava with diminished inspiratory collapsibility in subcostal view, E. 60/60 sign: coexistence of acceleration time of pulmonary ejection <60 ms and midsystolic "notch" with mildly elevated (<60 mmHg) peak systolic gradient at the tricuspid valve, F. decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<16 mm), G. decreased peak systolic (S')velocity of tricuspid annulus(<9.5 cm/s) and H. Mobile right-heart thrombi.[2]

PAP measurement values are not included in the PE mortality risk assessment criteria of the ESC. The reason why PAP measurement is not used in determining the mortality risk of acute PE may be the thought that the measurements are not reliable. Lafitte et al. found a strong correlation between Doppler sPAP values and measurements in right heart catheterization.[8] Some other studies reported limitations in accuracy of echocardiography in hemodynamic assessment of pulmonary hypertension.[9,10] It should also be kept in mind that sPAP can vary with age and body mass index. Beside all these limitations, higher sPAP was connected with an increased mortality.[10]

There is a strong linear relationship between sPAP and mean pulmonary artery pressure (mPAP) which is determined by pulmonary angiography. The 25 mmHg threshold used to define mean pulmonary hypertension (PH) corresponds to a sPAP of 38 mm Hg. Further invasive studies are needed to improve the precision of the sPAP-derived mPAP estimate.[11]

The RV is a part of a low-pressure system with a low-resistance afterload of the pulmonary artery, and is compliant, thin-walled and able to accommodate a large amount of blood, but without a lot of pressure generated. The left ventricle (LV), on the other hand, is thick-walled and non-compliant, generating a large amount of pressure against a high-resistance afterload of the aorta.[12] Acute right-sided heart failure due to increased pulmonary vascular resistance (PVR) and increased PAP is the prime cause of death in PE. The rapid rise in afterload causes dilatation of the right ventricle, afterwards systemic hypotension develops. Decrease of coronary perfusion, leads to myocardial ischemia and sometimes even infarction. The septal shift resulting from RV dilatation further reduces left ventricular preload and right side heart failure occurs.[13] The high mortality of major acute PE is generally accepted to be associated with sudden right ventricular dysfunction. RV dilation and hypokinesis in the presence of normal or hypotensive arterial pressure may also lead to myocardial ischemia and then RV infarct.[14] The dimensions and the amount of the pulmonary arterial thrombus is also an important factor affecting the development of pulmonary hypertension. The changes in pulmonary arterial systolic pressure are closely related to the area of embolism after acute PE. As autopsy results demonstrate, mPAP begins to increase when the pulmonary vascular obstruction reaches >25–30%, and reaches to 40 mmHg if the obstruction is 40–50%. The mortality increases if pulmonary vascular obstruction reaches >60%, and sudden death may occur because of RV infarct if the obstruction reaches >80%.[15]

Mobile right-heart thrombi are associated with high early mortality, especially in patients with RV dysfunction[16] and are detected by transthoracic or transoesophageal ECHO, or by CTPA, in <4% of patients with PE. Their

prevalence may reach 18% among PE patients in the intensive care setting.[17]

Cardiac-specific isoforms of troponin have been reported to be sensitive biochemical markers for minor myocardial damage. Troponin I and troponin T are essential components of striated muscle and released as a result of myocardial necrosis. Many studies have demonstrated that measurements of cardiac troponins are useful for the objective risk assessment in patients presenting with unstable angina pectoris and myocardial infarction.[18–20] In one study, 39% of the patients clinically diagnosed as having acute PE presented with elevated troponin I serum concentrations. It was demonstrated that positive troponin I tests were significantly associated with the occurrence of RV dysfunction.[14] Becattini et al. performed a meta-analysis of 20 retrospective or prospective researches in patients with acute PE to study the prognostic value of troponin levels for short-term death and adverse outcome events (composite of death, shock, need for thrombolysis, endotracheal intubation, inotrope/vasopressor infusion, cardiopulmonary resuscitation, or recurrent pulmonary embolism). From this meta-analysis it was concluded that elevated troponin levels are associated with short-term death and adverse outcome events in acute PE patients.[21] Troponin T test was used in our study and strong positive linear correlation was found between sPAP, and troponin T (Spearmen's r = 0.615, p <0.001). Therefore, sPAP was strongly associated with myocardial necrosis. Sensitivity and specificity of troponin T in detecting RVD above 20.1 cut-off value was found as 80.2% and 81%, respectively. Systolic PAP showed better correlation than troponin with RVD.

Mishra et al. reported in a review that elevated sPAP was significant in predicting mortality in severe COVID-19 patients with chronic obstructive pulmonary disease, congestive heart failure, myocardial injury, and pulmonary embolism. Presence of pulmonary embolism, and thrombotic microangiopathy can also contribute to the development of PH.[22]

Keller et al. investigated sPAP as a single parameter for risk stratification and especially for prediction of myocardial necrosis and RVD in 133 patients having acute PE. In their analysis, older PE patients with higher sPAP, had higher cardiac troponin I values and RVD was found more often in ECHO. Systolic PAP was strongly associated with myocardial necrosis and RVD. An increase of sPAP within 1 mmHg leads to an increase of the risk for myocardial necrosis of 2.1% and of the risk of presence of RVD of 10.1%. The effectiveness of sPAP to predict myocardial necrosis with cut-off value of 41.00 mmHg was moderate (AUC 0.628), while that of sPAP predicting RVD with cut-off value of 31.00 mmHg was excellent (AUC 0.829).[23] In a prospective study, 520 acute PE patients analyzed by Zhu et al. and they showed that 14-day mortality in normotensive patient with RVD was higher. Best cut-off value of sPAP for 14-day mortality was 60 mmHg. Systolic PAP was found to be an independent predictor of 3-month clinical outcomes.[24]

There are a few restrictive factors in our study. Firstly, this is a retrospective design clinical trial. Secondly, there were four patients who did not have RVD on ECHO, but were considered high-risk due to hemodynamic instability and prearrest clinical findings. Again, although there was no evidence of RVD on ECHO, 15 cases were considered to be at intermediate high mortality risk due to class 4 dyspnea definition and progression to cardiogenic shock due to blood pressure arterial and pulse values. In these cases, it was thought that RVD could not be detected since ECHO findings would vary according to the clinical experience of the physician.

In our study, strong positive linear correlation was found between sPAP and troponin T. It was also found that, between sPAP and RDV, ESC high-risk, intermediate-high risk groups (Spearman's r = 0.615, 0.798, 0.411, 0.408 p < 0.001, respectively). The optimal cut-off value for sPAP was 41.5 mmHg and the AUC value for sPAP was 0.961 (95% CI: 0.937–0.984), which is excellent. Systolic PAP had a sensitivity of 85% and a specificity of 92% in detecting RVD above 41.5 mmHg cut-off value. Besides, at the cut-off value of 41.5 mmHg, sPAP was found effective in determining RVD and ESC risk groups (p <0.0001).

Conclusion

The effectiveness of sPAP to predict myocardial necrosis in acute PE and the strong association between sPAP and RVD suggests that it would be useful to include sPAP in RVD criteria for acute PE risk stratification. ECHO is a reliable tool that is used in every step of diagnosis, risk stratification and, management of acute PE. Systolic PAP is a practical easy applicable measure of ECHO and should be used frequently in the decision of the treatment, prescience of prognosis in acute PE.

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