Prognostic Value of Right Ventricular Dysfunction and Pulmonary Obstruction Index by Computed Tomographic Pulmonary Angiography in Patients with Acute Pulmonary Embolism

Narumol Chaosuwannakit MD*,
Pattarapong Makarawate MD**

** Radiology Department, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand*
*** Cardiology Unit, Internal Medicine Department, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand*

Objective: *To quantify right ventricular dysfunction (RVD) and pulmonary obstruction index assessed by computed tomographic pulmonary angiography (CTPA) to assess the predictive value of these CT parameters for mortality after the initial diagnosis of acute pulmonary embolism (APE).*
Material and Method: *In 81 consecutive patients with proved APE, two readers assessed the extent of RVD, the shape of the interventricular septum, and the extent of obstruction to the pulmonary artery on CTPA images. The readers were blinded for clinical outcome in consensus reading.*
Results: *During follow-up, 20 patients died (25%). CT signs of RVD (RV/LV ratio > 1.0) were seen in 47 patients (58%). The RV/LV ratio, the shape of interventricular septum, and the obstruction index were shown to be significant risk factors for mortality ($p < 0.001$, $p = 0.04$, $p < 0.001$, respectively). The negative predictive value for mortality with an RV/LV ratio ≤ 1.0 and the obstruction index of < 40% were 100%.*
Conclusion: *CTPA quantification of RVD and pulmonary vascular obstruction index are potential useful tools to predict mortality in patients with APE.*

Keywords: *Acute pulmonary embolism, CTPA, Right ventricular dysfunction, Pulmonary obstruction index*

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Acute pulmonary embolism (APE) is a common disease responsible for a natural mortality rate of as much as 30%[1,2]. In some cases, pulmonary embolism may cause so much hemodynamic compromise that death suddenly ensues, sometimes before hospital admission, however, in most severely affected patients, circulatory failure develops progressively, within the first hours after onset of APE[3]. In part, mortality is caused by pressure overload of the right ventricle secondary to acute pulmonary arterial hypertension caused by PE. Initially, this results in right ventricular dysfunction (RVD), which may progress to right ventricular failure and circulatory collapse[4]. This hemodynamic profile results from acute cor pulmonale due to increased pulmonary vascular input impedance that overwhelms the adaptive resources of the right ventricle[4].

The problem facing the medical team is the necessity to detect those patients with life threatening right ventricular (RV) failure even though systemic blood pressure (BP) may be still satisfactorily compensated at presentation. The presence of RVD is a marker for adverse clinical outcome in patients with acute PE[4]. Echocardiography has emerged as first line examination for the detection of RV dysfunction, in addition to the possibility of being serially repeated[5-8].

Thrombi within cardiac cavities or in the pulmonary vascular bed are important adverse prognostic factors. Unfortunately, such thrombi detected by echocardiography are coincidental findings, even when the assessment is done by an experienced operator[7]. APE is therefore suspected when other diseases leading to right heart failure (*i.e.*, pericardial effusion and myocardial infarction) have been reasonably excluded.

Correspondence to:
Chaosuwannakit N, Radiology Department, Faculty of Medicince, Khon Kaen University, Khon Kaen 40000, Thailand.
Phone: 084-646-4648, 081-661-4751
E-mail: docnaruchao@yahoo.com

Since helical computed tomography (CT) is now more routinely used as a first-line technique to diagnose PE, the assessment of RVD and CT would facilitate patient diagnosis[9,10]. Many of the findings of RV overload are also detectable with computed tomographic pulmonary angiography (CTPA)[9-16]. Importantly, this approach is usually available 7-24 in most emergency institutions and it has the potential both to reveal central hemodynamic disturbances and to directly diagnose APE[17]. Clinical studies designed to evaluate whether CTPA can accurately predict short-term prognoses remain relatively scarce and present conflicting conclusions[13,18-20].

The ratio of the right ventricle to the left ventricle short axis diameter (RV/LV) has been proposed as an accurate sign for the presence of RVD, as assessed by CT[9,14,21]. Other criteria have been proposed, including deviation of the interventricular septum and the ratio of the pulmonary artery to the ascending aorta diameters[22]. The arterial thrombus load in the pulmonary arteries has been proposed as a signal parameter for predicting RVD[12,19,21]. The aim of the present study was to retrospectively quantify RVD and the pulmonary artery obstruction index with CT on the basis of the various criteria proposed in the literature and to assess the predictive value of these CT parameters for mortality in consecutive patients who are diagnosed with APE.

Material and Method

Patients

The participants were consecutive in- and out-patients suspected of having PE at clinical presentation but who had not yet undergone diagnostic testing for PE. Patients (a) undergoing thrombolytic therapy because of hemodynamic instability (b) having a contraindication for iodinated contrast material administration and/or (c) having a known case of heart disease were excluded. The present study was approved by the Ethics Committee of the Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand.

The charts and CTPA of all patients who underwent contrast material enhanced CT of the pulmonary arteries (CTPA) to detect or exclude PE between January 1, 2007, and July 31, 2011, were retrospectively reviewed. Among the 732 consecutive patients suspected of having acute PE at clinical presentation, 685 were included. CTPA was performed in 685 and PE was demonstrated in 81 (11.8%) among whom all were treated with anticoagulant therapy. There were 36 men (mean age, 52 ± 16 years; range, 16-84) and 45 women (mean age, 53 ± 13; range, 32-76). There was no statistically significant difference in age by sex.

Imaging studies

CTPA was performed using multi-detector row computed tomography (MDCT) on a Somatom Sensation 4, Siemens, Germany and MDCT Brilliance iCT 128, Philips, Eindhoven, The Netherlands. The caudocranial spiral volumetric acquisition was obtained 2 cm below the top of the diaphragm to a level slightly above the aortic arch during a single breath hold; with a detector collimation of 5 mm (n = 19) or 1 mm (n = 62), 120 kVp, 250 mAs, and a reconstruction interval of 3 mm. Among dyspnic patients, scanning was performed during shallow, gentle respiration. A standardized dose rate and a total dose of non-ionic contrast agent were used. Image acquisition started after a scanning delay of 15-20 seconds, after the start of intravenous injection of the contrast medium. The images were obtained at standard mediastinal settings (window width, 350 HU; window level, 50 HU) and lung settings (window width, 1,500 HU; window level, 500 HU).

The images of patients with a positive diagnosis of APE were selected and reviewed retrospectively at an independent work station in random order by two cardiovascular and thoracic radiologists in consensus (with a respective 7 and 5 years of experience in examining thoracic CT scans). The physicians were unaware of (a) the clinical signs and symptoms (b) the patient's condition at the time of initial presentation and/or (c) the clinical outcome.

CT signs of RVD

The scans were evaluated by measuring the minor axes of the right and left ventricles of the heart in the transverse plane at their widest points between the inner surface of the free wall and the surface of the interventricular septum (Fig. 1). These maximum dimensions were found at various levels. The RV/LV ratio was then calculated. As per the literature, CT scans were considered as showing (a) no RVD if the ratio was ≤ 1.0 (b) modest RVD if the ratio was between 1.0 and 1.5 and (c) severe RVD if the ratio was > 1.5[13,14,21].

Deviation of the interventricular septum was evaluated on a three-point scale: 1 = normal septum (convex toward the right ventricle); 2 = flattened septum; and 3 = septum deviation convex toward the left ventricle[14,21]. The diameters of the main

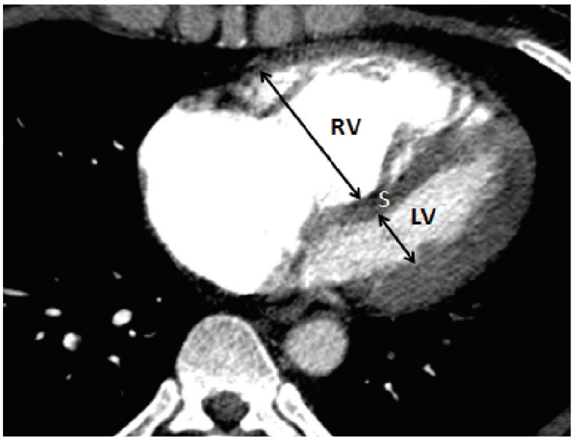

Fig. 1 Axial CTPA shows right ventricle (RV) dilatation and interventricular septum (S) deviation convex toward the left ventricle (displacement grade 3). Black arrows demonstrate the maximum minor axis measurements of the right ventricle (RV) and left ventricle (LV), RV/LV ratio = 1.9

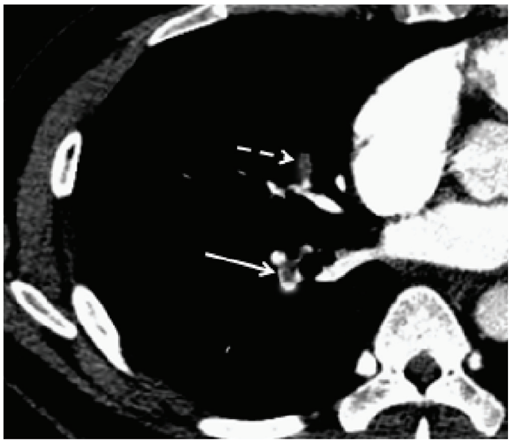

Fig. 2 Axial CTPA shows totally occlusive clot in medial segmental branch of the right middle lobe pulmonary artery (dashed arrow) scored by two investigators as 1 with a weighting factor of 2 and partially occlusive clot in the superior segmental branch of right lower lobe pulmonary artery (solid arrow) scored by two investigators as 1 with a weighting factor of 1

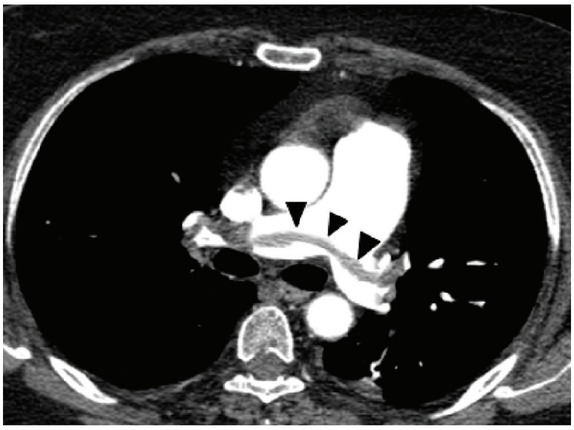

Fig. 3 Axial CTPA shows proximal nonocclusive clots in right and left pulmonary arteries (arrowheads). Both investigators calculated the CT index for obstruction as 50%

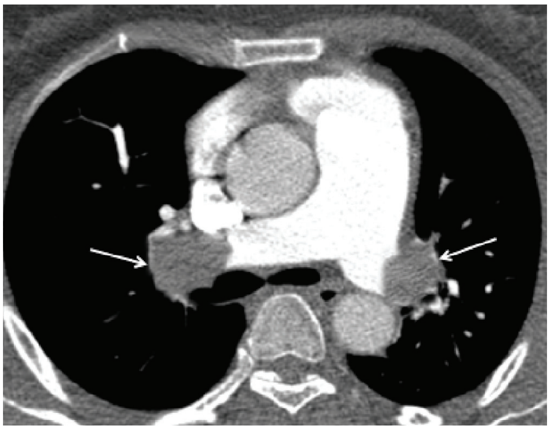

Fig. 4 Axial CTPA shows a proximal clot completely occluding both right and left pulmonary artery (arrows). The CT obstruction index was scored as 100% by both investigators

pulmonary artery and ascending aorta (the diameters of the inner lumina) were measured at a single predefined transverse scanning level; the point at which the right pulmonary artery is in continuity with the main pulmonary artery and sweeps across the midline. The pulmonary artery to the ascending aorta diameter ratio was then calculated[22].

CT obstruction index

The CT obstruction index was quantified using the scoring system devised by Qanadli et al[12]. To define the CT obstruction index, the arterial tree of each lung was regarded as having 10 segmental arteries (3, 2, 2 and 5 in the upper, middle, lingula, and lower lobes, respectively). The presence of an embolus in a segmental artery was given 1 point (Fig. 2), and emboli in the most proximal arterial level got a value equal to the number of segmental arteries arising distally (Fig. 3, 4). To provide additional information about the residual perfusion distal to the embolus, a weighting factor was assigned to each value, depending on the degree of vascular obstruction. This factor was 0 when no thrombus was observed; 1 when a partially occlusive thrombus was observed (Fig. 2, 3); or 2, when there was total occlusion (Fig. 2, 4). The maximal CT obstruction index was 40 per patient. Isolated subsegmental emboli indicated a partially occluded segmental artery and therefore assigned a value of 1.

The percentage of vascular obstruction was calculated by dividing the patient score by the maximal total score and multiplying the result by 100. The CT obstruction index can therefore be expressed as:

$[\Sigma(n \bullet d)/40] \times 100$

where n is the value of the proximal thrombus in the pulmonary arterial tree equal to the number of segmental branches arising distally (minimum, 1; maximum, 20) and d is the degree of obstruction (minimum, 0; maximum, 2).

Therapeutic protocol and follow-up

All of the patients were treated using standard therapeutic protocols for the management of APE[23,24]. Etiological treatment comprised anticoagulation in all patients and thrombolytic therapy in 15 (18.5%). The decision to proceed to thrombolytic therapy was based on the existence of either massive APE and hemodynamic instability or the presence of acute cor pulmonale, evidenced by qualitative echocardiography.

Two patients (2.5%) underwent surgical embolectomy because of a contraindication to thrombolytic therapy or circulatory failure. During the follow-up period, all of the patients received routine clinical care from their physicians.

All patient deaths during follow-up were evaluated by an independent adjudication cardiologist with full access to all of the available clinical and diagnostic data. The adjudication cardiologist determined and recorded whether a patient's death should definitely or most probably be attributed to PE or whether it should be attributed to a cause unrelated to PE.

Statistical analysis

Statistical analysis was performed with SPSS version 12.0 (Statistical Package for the Social Sciences, Chicago, IL). The association between death and the RV/LV ratio, the pulmonary artery to ascending aorta ratio, and the CT obstruction index were determined using a Cox regression analysis corrected for age. The regression coefficients and p-values were reported for continuous variables, the hazard ratio and confidence intervals for the CT obstruction index as a categorical variable. The RV/LV ratio, the pulmonary artery-to-ascending aorta ratio, and the CT obstruction index are expressed as a mean ± standard deviation, while the differences in the shape of the interventricular septum are expressed as frequencies. Differences in the shape of the interventricular septum, the pulmonary artery-to-ascending aorta ratio, and the CT obstruction index among the patients – divided into groups according to their RV/LV ratio – were tested with one-way analysis of variance. The least significant difference test was used as a correction for the multiple post hoc comparison testing. Discriminating variables were submitted to receiver operating characteristic (ROC) analysis for predicting mortality and area under curves (AUC) were calculated and tested for significance (H0: AUC > 0.5). A p-value of less than 0.05 was considered statistical significance different.

Results

Clinical follow-up

A three-month follow-up was completed for all patients. During the follow-up period, 61 (75%) patients survived and 20 (25%) died. The mean time to death was 3.5 days (range, 1-9), the day of inclusion was considered day 1. The mean hospital stay was 14.3 days (range, 1-66). According to the independent adjudication cardiologist, death was related to PE in twelve patients, none of whom had any history of cardiac failure or chronic obstructive pulmonary disease.

RV/LV ratio

The mean RV/LV ratio was 1.31 ± 0.52 (Table 1). There was a statistically significant relationship between the RV/LV ratio and PE-related mortality, with a regression coefficient of 1.46 ($p = 0.025$, Table 2). Results of post hoc testing showed that in patients who died of PE, the mean RV/LV ratio was significantly higher than that in patients who survived (1.82 ± 0.46 vs. 1.14 ± 0.42, $p < 0.001$, Fig. 5). The increase in the RV/LV ratio was caused by both an increase in the right ventricular diameter and a decrease in the left ventricular diameter. In the authors' study, 34 (41.9%) patients had RV/LV ratio of ≤ 1.0 (all survived), 17 (20.9%) had RV/LV ratio between 1.0 and 1.5 (six died, 35.3%) and 30 (37.2%) had RV/LV ratio > 1.5 (14 died, 46.7%). There was no significant difference in age or sex distribution between the two groups with elevated RV/LV ratios ($p = 0.69$ and $p = 0.56$, respectively). The positive predictive value for PE-related mortality with an RV/LV ratio > 1.0

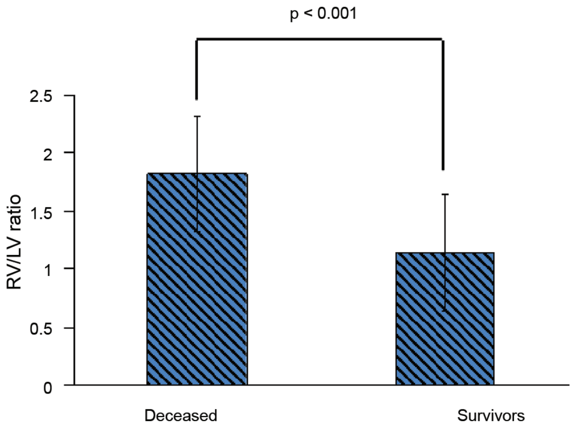

Fig. 5 Graph of mean values of the RV/LV ratio relative to clinical outcome. The mean RV/LV ratio was significantly higher in deceased patients than the survivors ($p < 0.001$)

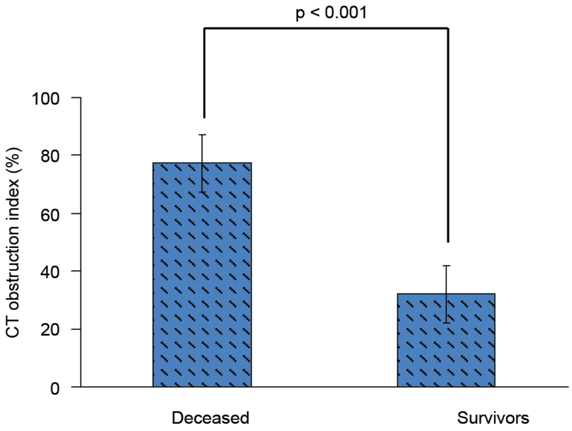

Fig. 6 Graph of mean values of the CT obstruction index relative to clinical outcome. The mean CT obstruction index was significantly higher in deceased patients than the survivors ($p < 0.001$)

was 20.3% (95% CI: 2.7%, 37.1%), and the negative predictive value for an uneventful outcome with an RV/LV ratio of ≤ 1.0 was 100% (95% CI: 95.1%, 100%). In the present study population, an RV/LV ratio of ≤ 1.0 excluded mortality due to PE.

CT-derived vascular obstruction index

The mean value of the vascular obstruction index was 43.2 ± 29.5% (Table 1). Results of the Cox regression analysis showed a statistically significant relationship between the vascular obstruction index and PE-related mortality ($p = 0.018$, Table 2). Results of post hoc testing showed that patients who died of PE during follow-up had, on average, a significantly higher vascular obstruction index than did patients who survived (77.3 ± 13.2 vs. 32.0 ± 24.3, $p < 0.001$ (Fig. 6).

When the previously recommended cut-off value of 40% for the pulmonary vascular obstruction index was used[12], patients with an index of ≥ 40%

Table 1. CT Measurements in patients with acute pulmonary embolism

Parameters	All patients (n = 81)	RV/LV ratio		
		> 1.5 (n = 30)	> 1 and ≤ 1.5 (n = 17)	≤ 1 (n = 34)
Maximum diameter (mm)[a]				
Right ventricle	46.00 ± 9.30	54.50 ± 6.10	47.10 ± 5.20	38.00 ± 5.70
Left ventricle	37.80 ± 9.50	29.30 ± 4.10	37.40 ± 6.30	45.50 ± 7.70
RV/LV ratio[a]	1.31 ± 0.52	1.89 ± 0.35	1.25 ± 0.11	0.83 ± 0.09
Interventricular septum[b,c]				
Normal	34 (42)	0 (0)	0 (0)	34 (100)
Displacement grade 2	25 (31)	8 (27)	17 (100)	0 (0)
Displacement grade 3	22 (27)	22 (73)	0 (0)	0 (0)
PA/Ao ratio[a,d]	1.05 ± 0.27	1.19 ± 0.30	1.19 ± 0.24	0.87 ± 0.09
Obstruction index (%)[a]	43.20 ± 29.50	68.90 ± 17.20	57.60 ± 14.30	12.90 ± 9.63
Obstruction index categoric[d]				
< 40%	38 (47)	1 (3)	3 (18)	34 (100)
≥ 40%	43 (53)	29 (97)	14 (82)	0 (0)

[a] Data are mean ± standard deviation
[b] Data are numbers of patients, and numbers in parentheses are percentages
[c] Grade 2 = flattened, grade 3 = deviated toward the left ventricle
[d] PA/Ao ratio = ratio of inner lumen pulmonary artery diameter to aorta diameter

Table 2. Univariate Cox regression analysis of death due to PE

Parameter	Regression coefficient	Standard error	p-value
RV/LV ratio	1.46	0.63	0.025*
CT Obstruction index	0.035	0.006	0.018*
PA/Ao ratio+	1.21	2.82	0.56

+ PA/Ao ratio = ratio of inner lumen pulmonary artery diameter to aorta diameter
* p value <0.05

had a 13.1-fold increased risk of dying of PE relative to patients with an index of < 40% (hazard ratio, 13.1; 95% CI: 2.3, 90.1). Twenty of 43 patients with an obstruction index of ≥ 40% died of PE vs. none of the 38 patients with an obstruction index of < 40% died. The positive predictive value for PE-related mortality with an obstruction index of ≥ 40% was 25% (95% CI: 6.3%, 30.8%). The negative predictive value for an uneventful outcome with an obstruction index of < 40% was 100% (95% CI: 96.4%, 100%). The positive predictive value for PE-related mortality among patients with RV/LV ratio >1.0 and an obstruction index of ≥ 40% was 28.9% (95% CI: 6.1%, 42.8%), and the negative predictive value for an uneventful outcome among patients with RV/LV ratio of ≤ 1.0 and obstruction index of < 40% was 100% (95% CI: 97.6%, 100%).

Displacement of the interventricular septum

Displacement of the interventricular septum was noted in 47 of 81 patients: flattening of the septum in 25 (30.8%) and inversion convex toward the left ventricle in 22 (27.2%) (Table 1). There were relatively more instances of displacement of the septum noted in patients who died of PE than in the remaining patients, but septum inversion was encountered in both groups. The authors found a significant relationship between the shape of the interventricular septum and PE-related mortality (p = 0.04). There was a relationship between the RV/LV ratio and septum displacement: the shape of the interventricular septum was abnormal in all patients with modest and severe RVD and in only two (5.9%) without RVD (p < 0.001).

Ratio of pulmonary artery to aorta diameters

The mean value of the ratio between the diameters of the pulmonary artery and the ascending aorta was 1.05 ± 0.27. A significant relationship was not found between this ratio and PE-related mortality (p = 0.78) or between this ratio and the RV/LV ratio (p = 0.09).

Discussion

The results of the present study showed that the presence and severity of RVD and the extent of obstruction of the pulmonary arterial tree, as assessed at CT, help to predict mortality of patients presenting with acute pulmonary embolism (APE). It is important to note that both the absence of RVD – as assessed by means of an RV/LV ratio of ≤ 1.0 and an obstruction index of < 40% – were predictive for an uneventful outcome. By contrast, deviation of the interventricular septum and the ratio between the pulmonary artery and ascending aorta diameters seemed to have little prognostic relevance vis-à-vis acute PE.

Importantly, the clinically relevant parameters of RVD can be obtained even from CT examinations that are not gated to the electrocardiogram. These data are consistent with the pathophysiological scheme for APE induced circulatory failure. It should be remembered that the normal pulmonary vascular bed is a low resistive, but highly expandable system, mechanically coupled with the right ventricle, which operates physiologically as a low pressure, volume relaxator[25,26]. The cardiovascular effects of an APE must therefore be regarded as both the result of the degree of pulmonary vascular obstruction and the degree to which it requires the right ventricle to function as a high-pressure pump[26,27]. Failure therefore occurs if the thin right ventricle walls are unsuccessful in compensating for the sudden increase in parietal tension. It is possibly the uncoupling of right ventricular resources from the pulmonary vascular load, rather than obstruction per se, that induces ventricular dilatation and dysfunction, which successively leads to decreased stroke volume, tricuspid regurgitation, reduced venous return, and finally circulatory collapse[4].

The lack of knowledge of the normal indices at which the right ventricle is actually operating therefore precludes using only the obstruction index or pulmonary arterial pressure to predict potential circulatory failure and subsequent death. This CTPA study demonstrates that proportional increases in the RV/LV ratio in severely affected patients is consistent with the occurrence of RV failure. These observations are in agreement with other studies in which similar CTPA variables were recognized as providing valuable prognostic information[9,13,20,27]. For example, Quiroz et al recently reported that an RV/LV ratio greater than a critical value of 0.9 (obtained by using the reconstructed four-chamber view) was associated with a sensitivity of 83.3% and a specificity of 48.7% for predicting adverse events, including not only 30-day mortality but also the need for resuscitation[13].

These data from axial views show that most patients who die rapidly from APE have an RV/LV ratio of more than 1.5 on presentation. This threshold value might be useful for predicting in-hospital survival. It is possible that abnormalities in the motion of the wall of the right ventricle will be a more powerful predictor of outcomes related to APE than the RV/LV ratio. Notwithstanding, neither echocardiography nor CT image acquisition with ECG gating are routinely available in many tertiary institutions for immediate APE diagnosis.

In terms of practical utility for most emergency departments, ungated CT assessment of the RV/LV ratio has the advantage of being simply and rapidly ascertained with only visual inspection of axial images[9]. Various diagnostic techniques or laboratory tests have been proposed to stratify patients with PE at clinical presentation into groups with higher or lower risk for fatal PE, with the ultimate aim of identifying those patients who might benefit from more aggressive fibrinolytic therapy. Results of previous studies have shown that echocardiography might be a useful method to predict RVD and clinical outcome[4-7]. As an alternative, blood tests, including measurement of plasma brain natriuretic peptides[28,29] or cardiac troponins T and I[30], have been proposed as prognostic indicators for benign vs. complicated courses. Ten Wolde et al[28] showed that the adjusted odds ratio of a brain natriuretic peptide level > 21.7 pmol/L for death related to PE was 14.1 (95% CI: 1.5, 131.1).

One of the main limitations of the present study was its retrospective design. Furthermore, the number of deaths related to PE that were observed during the follow-up period was somewhat limited and, as a consequence, the confidence limits around the point estimates are wide. Nevertheless, these findings are in agreement with those of Wu et al[19], who suggested that the quantification of a clot at CT pulmonary angiography is an important predictor of patient death in the setting of PE. On the other hand, authors of another study with an even smaller number of PE-related deaths did not find a significant relationship between RVD or the extent of vascular obstruction of the pulmonary artery circulation and clinical outcome[18]. Thus, a larger prospective study should be conducted to evaluate both the role of CTPA and echocardiography and cardiac biomarkers (*i.e.*, troponins or brain natriuretic peptides) for assessment of prognosis. In addition, echocardiographic evaluations did not provide quantitative data regarding either cardiac chamber dimensions or assessment of cardiac wall motion, preventing any further analysis or comparisons with CT data of cardiac strain. Finally, ECG ungated CT scanning was probably not well-suited for conducting cardiac chamber measurements because of the absence of a reference point for diastole in the cardiac cycle. Therefore, it is assumed that these axial images satisfactorily bisected both ventricles along their maximal diastolic dimensions.

In conclusion, these results suggest that the RVD, RV/LV ratio and the pulmonary vascular obstruction index assessed by MDCT are potential useful tools for predicting mortality in acute PE at clinical presentation. In particular, both the absence of RVD as assessed by means of an RV/LV ratio of ≤ 1.0 and an obstruction index of < 40% were predictive for a better outcome.

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Potential conflicts of interests

None.

References

1. Alpert JS, Smith R, Carlson J, Ockene IS, Dexter L, Dalen JE. Mortality in patients treated for pulmonary embolism. JAMA 1976; 236: 1477-80.
2. Carson JL, Kelley MA, Duff A, Weg JG, Fulkerson WJ, Palevsky HI, et al. The clinical course of pulmonary embolism. N Engl J Med 1992; 326: 1240-5.
3. Dalen JE, Alpert JS. Natural history of pulmonary embolism. Prog Cardiovasc Dis 1975; 17: 259-70.
4. Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. Chest 2002; 121: 877-905.
5. Goldhaber SZ. Echocardiography in the management of pulmonary embolism. Ann Intern Med 2002; 136: 691-700.

6. Grifoni S, Olivotto I, Cecchini P, Pieralli F, Camaiti A, Santoro G, et al. Short-term clinical outcome of patients with acute pulmonary embolism, normal blood pressure, and echocardiographic right ventricular dysfunction. Circulation 2000; 101: 2817-22.
7. Kasper W, Konstantinides S, Geibel A, Tiede N, Krause T, Just H. Prognostic significance of right ventricular afterload stress detected by echocardiography in patients with clinically suspected pulmonary embolism. Heart 1997; 77: 346-9.
8. Vieillard-Baron A, Page B, Augarde R, Prin S, Qanadli S, Beauchet A, et al. Acute cor pulmonale in massive pulmonary embolism: incidence, echocardiographic pattern, clinical implications and recovery rate. Intensive Care Med 2001; 27: 1481-6.
9. Contractor S, Maldjian PD, Sharma VK, Gor DM. Role of helical CT in detecting right ventricular dysfunction secondary to acute pulmonary embolism. J Comput Assist Tomogr 2002; 26: 587-91.
10. Groves AM, Win T, Charman SC, Wisbey C, Pepke-Zaba J, Coulden RA. Semi-quantitative assessment of tricuspid regurgitation on contrast-enhanced multidetector CT. Clin Radiol 2004; 59: 715-9.
11. Mastora I, Remy-Jardin M, Masson P, Galland E, Delannoy V, Bauchart JJ, et al. Severity of acute pulmonary embolism: evaluation of a new spiral CT angiographic score in correlation with echocardiographic data. Eur Radiol 2003; 13: 29-35.
12. Qanadli SD, El Hajjam M, Vieillard-Baron A, Joseph T, Mesurolle B, Oliva VL, et al. New CT index to quantify arterial obstruction in pulmonary embolism: comparison with angiographic index and echocardiography. AJR Am J Roentgenol 2001; 176: 1415-20.
13. Quiroz R, Kucher N, Schoepf UJ, Kipfmueller F, Solomon SD, Costello P, et al. Right ventricular enlargement on chest computed tomography: prognostic role in acute pulmonary embolism. Circulation 2004; 109: 2401-4.
14. Reid JH, Murchison JT. Acute right ventricular dilatation: a new helical CT sign of massive pulmonary embolism. Clin Radiol 1998; 53: 694-8.
15. Reid JH. Semi-quantitative assessment of tricuspid regurgitation on contrast-enhanced multidetector CT. Clin Radiol 2004; 59: 713-4.
16. Bankier AA, Janata K, Fleischmann D, Kreuzer S, Mallek R, Frossard M, et al. Severity assessment of acute pulmonary embolism with spiral CT: evaluation of two modified angiographic scores and comparison with clinical data. J Thorac Imaging 1997; 12: 150-8.
17. Schoepf UJ, Goldhaber SZ, Costello P. Spiral computed tomography for acute pulmonary embolism. Circulation 2004; 109: 2160-7.
18. Araoz PA, Gotway MB, Trowbridge RL, Bailey RA, Auerbach AD, Reddy GP, et al. Helical CT pulmonary angiography predictors of in-hospital morbidity and mortality in patients with acute pulmonary embolism. J Thorac Imaging 2003; 18: 207-16.
19. Wu AS, Pezzullo JA, Cronan JJ, Hou DD, Mayo-Smith WW. CT pulmonary angiography: quantification of pulmonary embolus as a predictor of patient outcome--initial experience. Radiology 2004; 230: 831-5.
20. Schoepf UJ, Kucher N, Kipfmueller F, Quiroz R, Costello P, Goldhaber SZ. Right ventricular enlargement on chest computed tomography: a predictor of early death in acute pulmonary embolism. Circulation 2004; 110: 3276-80.
21. Collomb D, Paramelle PJ, Calaque O, Bosson JL, Vanzetto G, Barnoud D, et al. Severity assessment of acute pulmonary embolism: evaluation using helical CT. Eur Radiol 2003; 13: 1508-14.
22. Ng CS, Wells AU, Padley SP. A CT sign of chronic pulmonary arterial hypertension: the ratio of main pulmonary artery to aortic diameter. J Thorac Imaging 1999; 14: 270-8.
23. Task Force on Pulmonary Embolism, European Society of Cardiology. Guidelines on diagnosis and management of acute pulmonary embolism. Eur Heart J 2000; 21: 1301-36.
24. British Thoracic Society, Standards of Care Committee. Suspected acute pulmonary embolism: a practical approach. Thorax 1997; 52 (Suppl 4): S1-24.
25. Sharma GV, McIntyre KM, Sharma S, Sasahara AA. Clinical and hemodynamic correlates in pulmonary embolism. Clin Chest Med 1984; 5: 421-37.
26. Fourie PR, Coetzee AR, Bolliger CT. Pulmonary artery compliance: its role in right ventricular-arterial coupling. Cardiovasc Res 1992; 26: 839-44.
27. Ghuysen A, Lambermont B, Dogne JM, Kolh P, Tchana-Sato V, Morimont P, et al. Effect of BM-573 [N-terbutyl-N’-[2-(4’-methylphenylamino)-5-nitro-benzenesulfonyl]urea], a dual thromboxane synthase inhibitor and thromboxane receptor

antagonist, in a porcine model of acute pulmonary embolism. J Pharmacol Exp Ther 2004; 310: 964-72.
29. ten Wolde M, Tulevski II, Mulder JW, Sohne M, Boomsma F, Mulder BJ, et al. Brain natriuretic peptide as a predictor of adverse outcome in patients with pulmonary embolism. Circulation 2003; 107: 2082-4.
30. Kucher N, Printzen G, Goldhaber SZ. Prognostic role of brain natriuretic peptide in acute pulmonary embolism. Circulation 2003; 107: 2545-7.
31. Konstantinides S, Geibel A, Olschewski M, Kasper W, Hruska N, Jackle S, et al. Importance of cardiac troponins I and T in risk stratification of patients with acute pulmonary embolism. Circulation 2002; 106: 1263-8.

การทำนายการดำเนินของโรคในผู้ป่วยโรคลิ่มเลือดอุดกั้นที่ปอดอย่างเฉียบพลันโดยการประเมินหัวใจห้องล่างขวาและดัชนีการอุดกั้นของหลอดเลือดแดงที่ปอดโดยการตรวจด้วยเครื่องเอกซเรย์คอมพิวเตอร์

นฤมล เชาว์สุวรรณกิจ, ภัทรพงษ์ มกรเวส

วัตถุประสงค์: *เพื่อทำนายอัตราการเสียชีวิตในผู้ป่วยโรคลิ่มเลือดอุดกั้นที่ปอดอย่างเฉียบพลันโดยการประเมินหัวใจห้องล่างขวาและดัชนีการอุดกั้นของหลอดเลือดแดงที่ปอดโดยการตรวจด้วยเครื่องเอกซเรย์คอมพิวเตอร์*
วัสดุและวิธีการ: *มีผู้ป่วยที่ได้รับการวินิจฉัยว่าเป็นโรคลิ่มเลือดอุดกั้นที่ปอดอย่างเฉียบพลัน โดยการตรวจด้วยเครื่องเอกซเรย์คอมพิวเตอร์ 81 ราย ที่ได้เข้าการศึกษา และได้รับการประเมินหัวใจห้องล่างขวา รูปร่างของผนังหัวใจห้องล่าง และดัชนีการอุดกั้นของหลอดเลือดแดงที่ปอดโดยผู้ประเมิน 2 ราย ที่ไม่ทราบการดำเนินของโรค*
ผลการศึกษา: *ในระยะเวลา 3 เดือน ที่ติดตามผู้ป่วย มีผู้ป่วย 20 ราย เสียชีวิต (ร้อยละ 25) พบผู้ป่วย 47 ราย ที่มีภาวะหัวใจห้องล่างขวาล้มเหลวโดยการประเมินจากเครื่องเอกซเรย์คอมพิวเตอร์ การศึกษาพบว่าอัตราส่วนของความกว้างของหัวใจห้องล่างขวาต่อหัวใจห้องล่างซ้าย รูปร่างของผนังหัวใจห้องล่าง และดัชนีการอุดกั้นของหลอดเลือดแดงที่ปอดสามารถทำนายอัตราการเสียชีวิตของผู้ป่วยอย่างมีนัยสำคัญ ($p < 0.001$, $p = 0.04$, $p < 0.001$ ตามลำดับ) โดยคุณค่าการทำนายผลลบของอัตราส่วนของความกว้างของหัวใจห้องล่างขวาต่อหัวใจห้องล่างซ้ายที่มีค่าน้อยกว่าหรือเท่ากับ 1.0 และดัชนีการอุดกั้นของหลอดเลือดแดงที่ปอดที่น้อยกว่าร้อยละ 40 เป็นร้อยละ 100 ทั้งคู่*
สรุป: *การประเมินหัวใจห้องล่างขวาและดัชนีการอุดกั้นของหลอดเลือดแดงที่ปอดโดยการตรวจด้วยเครื่องเอกซเรย์คอมพิวเตอร์เป็นวิธีที่สามารถใช้ประโยชน์ในการทำนายอัตราการเสียชีวิตของผู้ป่วยโรคลิ่มเลือดอุดกั้นที่ปอดอย่างเฉียบพลันได้*