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ORIGINAL PAPER

Evaluation of right ventricular dysfunction and prediction of clinical outcomes in acute pulmonary embolism by chest computed tomography: comparisons with echocardiography

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Abstract To evaluate the ability to identify right ventricular (RV) dysfunction, and to predict adverse outcomes of chest computed tomography (CT), we compared CT and echocardiography in acute pulmonary embolism patients. We analyzed 56 patients diagnosed by CT with acute pulmonary embolism, who underwent echocardiography within 48 h of CT scan from January 2004 to December 2008. From the CT scan, the ratio of RV diameter to left ventricular diameter (RVd/LVd), the presence of septal bowing and embolus location were determined. RVd/LVd ($P < 0.001$), septal bowing ($P < 0.001$) and proximal embolism ($P = 0.016$) were associated with echocardiographic RV hypokinesia. The odds ratio for adverse clinical outcomes was 19.2 for the combination of three CT parameters (RVd/LVd > 1, septal bowing, and proximal embolism), and 13.4 for RV hypokinesia (each $P = 0.001$). The positive predictive value (PPV) for adverse clinical outcomes for echocardiographic RV hypokinesia was 55.0%, and the negative predictive value (NPV) was 96.2%. The three-parameter combination predicted adverse clinical outcomes with a PPV of 54.5%, and a NPV of 94.1%. CT parameters including RV dysfunction were significantly associated with poor outcomes. Rapid risk stratification of patients with acute pulmonary

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embolism based on chest CT appears to be comparable with echocardiography, is clinically reliable, and may be useful in guiding management strategy.

Keywords Adverse clinical outcome · Computed tomography · Pulmonary embolism · Right ventricular dysfunction · Right ventricular hypokinesia

Introduction

Acute pulmonary embolism is a potentially fatal disease with an overall 30 days mortality rate of more than 10% [1]. The most common cause of death within 30 days is right ventricular (RV) failure [1, 2]. Rapid risk stratification is crucial for identifying high-risk patients, and for selecting an appropriate treatment strategy. According to the European Society of Cardiology Guidelines on acute pulmonary embolism [3], reperfusion therapy as thrombolysis or surgical embolectomy is indicated for patients with cardiogenic shock or hypotension, and may be considered in selected patients who appear hemodynamically stable but have RV dysfunction and/or myocardial injury. Therefore, echocardiographic assessment of RV dysfunction in patients with acute pulmonary embolism can be important for predicting early mortality, and for guiding decisions regarding reperfusion therapy [4, 5]. However, echocardiography is time-consuming, operator-dependent, and not always available in an emergency situation, and the RV may be difficult to image using a transthoracic approach.

Contrast-enhanced chest computed tomography (CT) has become the first-line imaging test for patients with suspected pulmonary embolism [3, 6, 7]. Sometimes, CT is more rapidly accessible in emergency settings, and is more widely available than echocardiography. CT enables direct visualization of emboli, and provides information about cardiac morphology. Several studies have shown that CT findings, including RV enlargement, pulmonary vascular obstruction score, the ratio of the diameter of the RV to the diameter of the left ventricle (LV) (RVd/LVd), RV to LV area ratio, and interventricular septal bowing are associated with early mortality and clinical outcomes [8–13]. To our knowledge, only a few studies [8, 12, 14] have directly compared CT and echocardiographic findings of RV dysfunction. Therefore, we aimed to test the hypothesis that chest CT is a valuable rapid method of identifying RV dysfunction and predicting poor clinical outcomes, by comparing the two imaging methods in the same patients.

Methods

Patients

This retrospective study reviewed 56 patients with acute pulmonary embolism diagnosed by chest CT from January 2004 to December 2008 at a tertiary medical center. All patients had acute symptoms such as dyspnea, decreased blood oxygen saturation, or hypotension from unknown causes. CT was performed within 14 days of onset of events suggestive of acute pulmonary embolism. Echocardiography was performed within 48 h of the CT scan for direct comparison of the two imaging methods. We excluded patients who were incidentally diagnosed with pulmonary embolism after tests for other indications (e.g., metastatic work-up). Institutional Review Board approval was obtained, and informed consent was not required for this retrospective study.

Adverse clinical outcomes

We defined an adverse clinical outcome as at least one of the following: death associated with embolism within 30 days of diagnosis, cardiopulmonary resuscitation, use of inotropics because of hypotension, mechanical ventilation, thrombolysis or surgical embolectomy. These adverse clinical events were selected according to Management Strategies and Prognosis of Pulmonary Embolism-3 Trial (MAPPET-3) [15].

Computed tomography

CT examinations were performed using an 8-Slice MDCT scanner (LightSpeed Ultra 8; GE Medical Systems). Scanning parameters were as follows; 120 kV, 180 mA, 1:1.375 pitch, and 1.25 mm reconstruction slice thickness. Patients were examined while holding their breath or breathing shallowly, depending on the degree of dyspnea. A total of 120 ml nonionic contrast was administrated intravenously at a rate of 4 mL/s. The scanning was achieved using electrocardiographically (ECG) non-gated manner. The average radiation dose was 9–10 mGy.

Pulmonary embolism was confirmed in all patients by an experienced radiologist. For this study, CT findings were analyzed by a single reviewer who was unaware of the echocardiography results. RVd/LVd, septal bowing toward the LV, embolism location, main pulmonary artery diameter, and inferior vena cava (IVC) diameter were evaluated with axial views. RV and LV diameter were assessed for each single image at the plane of maximal visualization of the ventricular cavities. LV diameter was measured usually at the mitral valve plane, and RV diameter was measured at the tricuspid valve level. LV and RV diameter were defined as the distance from the interventricular septum to the endocardial border perpendicular to the basal third of the long axis of the heart (Fig. 1). RVd/LVd was calculated from RV to LV diameter. Septal bowing was defined as interventricular septal straightening or convex curvature toward the LV, and was subjectively judged as present or absent. Septal bowing was evaluated at the mid-ventricular septum to avoid confounding from basal septal hypertrophy or sigmoid septum. Pulmonary artery diameter was measured as the short axis distance in the main pulmonary trunk at a plane that showed the main trunk and bifurcations. IVC diameter was measured as the minimum diameter at a point just below the junction with the right atrium. Embolus location was classified as proximal or distal. Emboli that involved the main pulmonary trunk or the left/right pulmonary artery were classified as proximal pulmonary embolism. Emboli that did not involve the main pulmonary trunk or the left/right pulmonary artery, and were seen at far distal artery, were classified as distal pulmonary embolism. Classification was by embolus location rather than embolic burden scoring, to ensure rapid risk stratification assessment by general physicians, most of whom were not well trained in the complicated embolic burden scoring system [16].

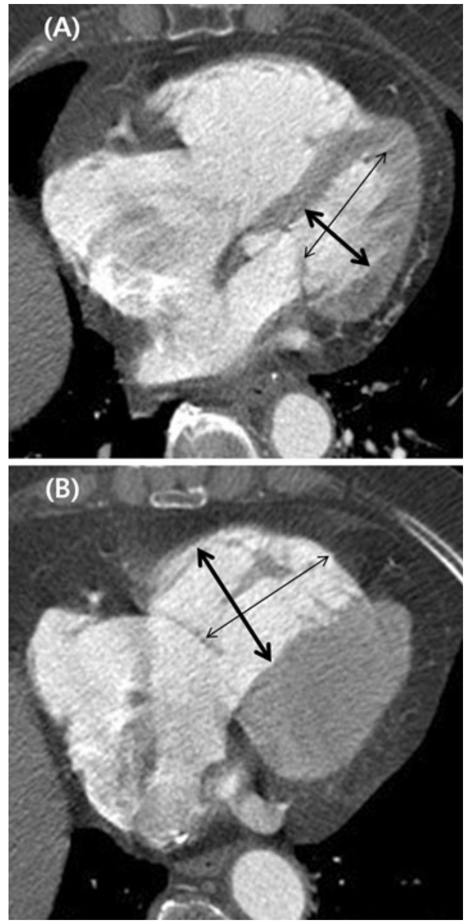

Fig. 1 LV diameter (**a**) and RV diameter (**b**) were defined as the distance from the interventricular septum to the endocardial border perpendicular to the basal third of the long axis of the heart at the plane of maximal visualization of the ventricular cavities

Echocardiography

RV hypokinesia and tricuspid regurgitation (TR) velocity were assessed by echocardiogram. RV hypokinesia was chosen as an indicator of RV dysfunction according to the criteria of the International Cooperative Pulmonary Embolism Registry (ICOPER) [1], and assessed by qualitative interpretation of systolic RV free wall motion. Degree of RV hypokinesia was not determined. Echocardiographic TR pressure gradients were analyzed to determine whether acute pulmonary embolism-induced pulmonary hypertension was prognostic for adverse clinical events. The pressure gradient between the RV and the right atrium was calculated using TR peak velocity and the simplified Bernoulli equation. We defined pulmonary hypertension as estimated TR pressure gradient $\geq$ 30 mmHg.

Statistical analysis

Continuous variables were expressed as mean $\pm$ standard deviation (SD) or median with interquartile range, and categorical variables as a number and percentage. Correlations of CT findings with RV hypokinesia or pulmonary hypertension on echocardiography were analyzed using the Mann–Whitney test. Comparisons of adverse clinical outcome with CT findings, RV hypokinesia and pulmonary hypertension used a chi-square test and the Mann–Whitney test. Receiver operating characteristic (ROC) analysis was performed to identify the cut-off value for RVd/LVd for RV hypokinesia prediction. The binary logistic regression test was performed to predict the odds ratio (OR) for adverse clinical outcomes. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated using a cross table. Statistical analysis was performed with the statistical package SPSS 17.0 (SPSS Interactive Graphics, Version 17.0, SPSS Inc., Chicago, IL, USA) and P value < 0.05 was considered statistically significant.

Results

Patients and clinical characteristics

The median age of the 56 patients was 63.5 years, and 28 (50%) of the patients were men. Fourteen (25%) patients had adverse clinical events, with 30 days death associated with acute pulmonary embolism present in one patient. Cardiopulmonary resuscitation was performed on two patients. Eleven (20%) patients underwent thrombolysis or surgical embolectomy. A ventilator was used on four patients and inotropics on six. All patients were treated with anticoagulants. Hypotension at initial presentation, defined as a systolic arterial pressure < 90 mmHg, was present in 36% of patients who experienced adverse events, and in 10% who experienced no adverse events ($P = 0.035$). Hypoxia (O_2 saturation $< 90\%$) was present in 43% with adverse events and 26% with no adverse events ($P = 0.199$) (Table 1). Deep vein thrombosis (DVT), recent surgery, and malignancy were not associated with adverse short-term clinical outcomes. The presence of risk factors such as diabetes mellitus, hypertension, and smoking were not associated with adverse clinical outcomes (Table 1).

Correlation of CT findings with echocardiography

When CT findings were compared to the presence or absence of RV hypokinesia on echocardiography, patients with RV hypokinesia showed significantly

Table 1 Baseline characteristics of patients

Characteristics	Total (n = 56)	With adverse events (n = 14)	Without adverse events (n = 42)	P value
Age, years#	63.5 (52–71)	65.0 (52–73)	62.5 (52–71)	0.495
Male sex	28 (50)	10 (71)	18 (43)	0.121
SBP, mmHg#	111.0 (98.0–128.0)	95.0 (76.0–122.0)	115 (100.8–128.3)	0.071
DBP, mmHg#	72.0 (60.0–82.0)	64.0 (53.5–89.0)	72.0 (63.8–80.5)	0.346
HR, bpm#	90 (80–107)	90 (82–112)	90 (80–104)	0.552
SBP < 90 mmHg	9 (16)	5 (36)	4 (10)	0.035
O_2 saturation $< 90\%$	17 (30)	6 (43)	11 (26)	0.199
Comorbidities and risk factors				
DVT	30 (54)	8 (57)	22 (52)	1.000
Recent surgery	13 (23)	1 (7)	12 (71)	0.149
Malignancy	22 (54)	4 (29)	18 (43)	0.529
Diabetes mellitus	8 (19.0)	2 (14)	6 (14)	1.000
Hypertension	24 (14)	6 (43)	18 (43)	1.000
Current smoking	5 (9)	2 (14)	3 (7)	0.590
COPD	0 (0)			

Data are expressed as the median value (25–75 percentiles)

Data are expressed as the number (percentage) of patients unless otherwise specified

The P value compares patients who experienced and did not experience adverse events. *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *DVT* deep vein thrombosis, *COPD* chronic obstructive pulmonary disease

Table 2 Comparison of CT findings and echocardiographic RV hypokinesia

	Presence of RV hypokinesia (n = 20)	No RV hypokinesia (n = 36)	*P* value
RV diameter (cm)[#]	4.86 ± 0.62	4.05 ± 0.73	<0.001
LV diameter (cm)[#]	3.23 ± 0.67	4.18 ± 0.60	<0.001
RVd/LVd[#]	1.58 ± 0.47	0.99 ± 0.30	<0.001
Septal bowing[a]	19 (95%)	8 (22%)	<0.001
Proximal PE[a]	18 (90%)	21 (58%)	0.016
PA diameter (cm)[#]	2.99 ± 0.72	3.08 ± 0.47	0.846
IVC diameter (cm)[#]	2.23 ± 0.40	2.19 ± 0.33	0.771
Both PAs involved[a]	19 (95%)	30 (83%)	0.402

[#] Mean ± SD

[a] Number (percentage) of patients

CT computed tomography, *RV* right ventricle, *LV* left ventricle, *RVd/LVd* ratio of RV diameter to LV diameter, *PE* pulmonary embolism, *PA* pulmonary artery, *IVC* inferior vena cava

Table 3 Comparison of CT findings and echocardiographic pulmonary hypertension

	TR pressure gradient ≥ 30 mmHg (n = 40)	TR pressure gradient < 30 mmHg (n = 12)	*P* value
RV diameter (cm)[#]	4.57 ± 0.75	4.20 ± 0.74	0.092
LV diameter (cm)[#]	3.67 ± 0.75	4.01 ± 0.84	0.137
RVd/LVd[#]	1.33 ± 0.46	1.13 ± 0.46	0.108
Septal bowing[a]	25 (62.5)	4 (33.3)	0.102
Proximal PE[a]	29 (72.5)	8 (66.7)	0.726
PA diameter (cm)[#]	3.11 ± 0.63	2.99 ± 0.33	0.464
IVC diameter (cm)[#]	2.27 ± 0.36	2.11 ± 0.35	0.146

[#] Mean ± SD

[a] Number (percentage) of patients

CT computed tomography, *TR* tricuspid regurgitation, *RV* right ventricle, *LV* left ventricle, *RVd/LVd* ratio of RV diameter to LV diameter, *PE* pulmonary embolism, *PA* pulmonary artery, *IVC* inferior vena cava

higher RVd/LVd (1.58 ± 0.47 vs. 0.99 ± 0.30, $P < 0.001$), larger RV diameter, and smaller LV diameter (Table 2). Septal bowing ($P < 0.001$) and proximal pulmonary embolism ($P = 0.016$) were also significantly associated with RV hypokinesia. However, pulmonary artery diameter, IVC diameter, and involvement of both pulmonary arteries were not associated with RV hypokinesia (Table 2).

TR pressure gradient measurements were available in only 49 patients (88%) because of poor imaging quality for some patients. TR pressure gradient was not associated with RV hypokinesia (48.1 ± 15.9 mmHg with RV hypokinesia vs. 40.5 ± 15.4 mmHg without, $P = 0.097$), and was not associated with adverse clinical outcomes (49.0 ± 17.6 mmHg with adverse clinical events vs. 41.4 ± 14.9 mmHg without, $P = 0.135$). No CT findings correlated with pulmonary hypertension, defined as TR pressure gradient ≥ 30 mmHg (Table 3). Therefore, this was not included in the analysis of prognostic factors.

Predictive value of CT and echocardiography findings

We performed ROC curve analysis to determine the cut-off value for RVd/LVd. At an RVd/LVd cut-off value of 1.0, the sensitivity for predicting RV hypokinesia was 95%, and the specificity was 67%. The area under the ROC curve was 0.88 for RV hypokinesia prediction, and 0.78 for adverse clinical outcome prediction (Fig. 2).

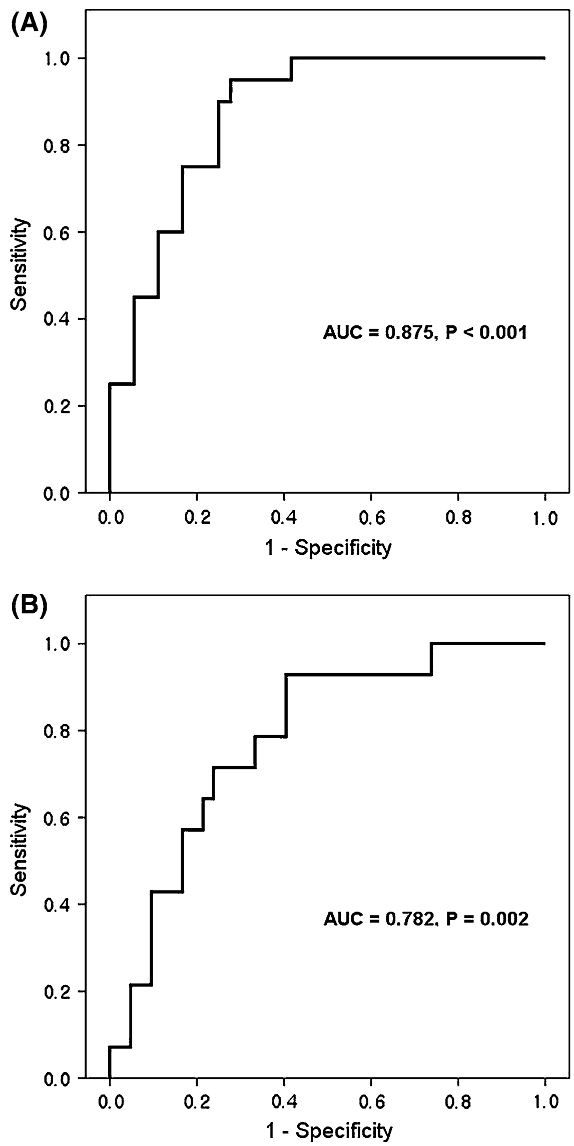

Fig. 2 Receiver operating characteristic (*ROC*) curve of RVd/LVd for RV hypokinesia (**a**) and ROC curve of RVd/LVd for adverse clinical outcomes (**b**) RVd/LVd, ratio of right ventricle diameter to left ventricle diameter; *AUC*, area under the ROC curve

Based on the data presented in Table 2 and Fig. 1, we selected RVd/LVd > 1, septal bowing, and proximal pulmonary embolism for assessing prognostic value. An RVd/LVd > 1 was present in 93% of patients who experienced adverse events, and 40% of patients who did not experience adverse events ($P = 0.001$). Septal bowing occurred in 86% of patients with adverse events, and 36% with no adverse events ($P = 0.002$), and proximal pulmonary embolism occurred in 93 and 62%, respectively ($P = 0.043$). All three parameters were seen in 86% of patients with adverse events, and 29% of patients with no adverse clinical events ($P < 0.001$) (Table 4).

We compared the ability of CT parameters and echocardiographic RV hypokinesia to predict adverse clinical outcomes (Table 5). The sensitivity 92.9, NPV 96.2% and OR 19.1 of a RVd/LVd > 1 were greater than those of RV hypokinesia (sensitivity 78.6, NPV 91.7% and OR 13.4). However, specificity and PPV of RVd/LVd > 1 were lower than for RV hypokinesia (specificity, 59.5% vs. 78.6% and PPV, 43.3% vs. 55.0%, respectively). Combination of the CT parameters had a specificity and PPV for adverse clinical outcomes that were higher than any individual parameter (Table 5).

Discussion

We found that the CT parameters of high RVd/LVd, septal bowing, and proximal pulmonary embolism were significantly associated with RV hypokinesia by echocardiography. None of CT findings correlated with pulmonary hypertension, which *per se* did not predict adverse clinical outcomes. Importantly, the predictive value of these CT findings for adverse clinical outcomes was comparable to echocardiographic RV hypokinesia.

Acute pulmonary embolism leads to an increase in RV vascular resistance and RV afterload. An abrupt increase in RV afterload may cause RV dilatation and hypokinesis, TR, and ultimately, RV failure. RV failure results in systemic arterial hypotension and cardiac arrest [17]. Thus, assessment of RV dysfunction is essential for risk stratification of patients with pulmonary embolism. Whether hemodynamically stable patients with RV dysfunction should be treated with a reperfusion therapy is still under debate. Nonetheless, reports suggest that RV dysfunction is an independent risk factor for hemodynamically stable pulmonary embolism [1, 4, 5]. A randomized trial of intermediate-risk pulmonary embolism patients by Konstantinides et al. [15] showed significant reduction of death or clinical deterioration requiring escalation of treatment in a thrombolytic group compared with a heparin group. In our study, the OR of

Table 4 Comparison of CT parameters and adverse clinical events

	With adverse events (n = 14)	Without adverse events (n = 42)	*P* value
RVd/LVd > 1	13 (93)	17 (40)	0.001
Septal bowing	12 (86)	15 (36)	0.002
Proximal PE	13 (93)	26 (62)	0.043
All three CT parameters#	12 (86)	12 (29)	<0.001

All data are expressed as the number (percentage) of patients

CT computed tomography, *RVd/LVd* ratio of right ventricle diameter to left ventricle diameter, *PE* pulmonary embolism

Patients with all three of the following: an RVd/LVd > 1, presence of septal bowing, and proximal pulmonary embolism

Table 5 Comparison of CT parameters and echocardiographic RV hypokinesia for adverse clinical outcomes (n = 56)

	RV hypokinesia	CT parameters			
		RVd/LVd > 1	Septal bowing	Proximal PE	All three CT parameters#
Sensitivity (%)	78.6	92.9	85.7	92.9	85.7
Specificity (%)	78.6	59.5	64.3	61.9	76.2
PPV (%)	55.0	43.3	44.4	33.3	54.5
NPV (%)	91.7	96.2	93.1	84.2	94.1
Odds ratio	13.4	19.1	10.8	8.0	19.2
95% CI	3.1–58.7	2.3–160.1	2.1–54.8	1.0–67.1	3.7–100.7
P value	<0.001	0.001	0.001	0.043	<0.001

CT computed tomography, *RV* right ventricle, *RVd/LVd* ratio of RV diameter to left ventricle diameter, *PE* pulmonary embolism, *PPV* positive predictive value, *NPV* negative predictive value, *CI* confidence interval

Patients with all three of the following: an RVd/LVd > 1, presence of septal bowing, and proximal pulmonary embolism

echocardiographic RV hypokinesia for a poor clinical outcome was 13.4, indicating that RV hypokinesia is an important risk factor.

However, echocardiography is not always available in emergency settings and has limitations including poor RV image quality, and lack of a universal definition of RV dysfunction. CT has become the diagnostic procedure of choice for pulmonary embolism. Assessing RV function at the same time as diagnosis of pulmonary embolism would enable rapid risk stratification, and facilitate the selection of an appropriate pulmonary embolism treatment. Several studies have evaluated RV dysfunction using CT and determined a predictive value for outcome [8–14]. However, few reports have conducted a direct comparison of CT findings with echocardiographic RV dysfunction.

In this study, heart morphology and size (RVd/LVd, septal bowing), vessel size (pulmonary artery and IVC diameter), and emboli locations were evaluated using CT. CT findings were compared with RV hypokinesia on echocardiography for adverse clinical outcomes. Although echocardiographic criteria for RV dysfunction differ among studies [5, 18, 19], we evaluated RV hypokinesia as an index of RV dysfunction in accordance with the ICOPER [1].

Large RV diameter, small LV diameter, high RVd/LVd, septal bowing, and proximal pulmonary embolism were significantly associated with RV hypokinesia on echocardiography and predicted an adverse clinical outcome. CT pulmonary artery diameter and IVC diameter were not associated with RV hypokinesia. TR pressure gradient on echocardiography was not associated with adverse clinical outcome. A combination of RVd/LVd, septal bowing, and proximal pulmonary embolism parameters gave a higher specificity and PPV for adverse clinical outcomes than any of the individual parameters, and RVd/LVd > 1 had the highest sensitivity and NPV. We also compared the predictive values of pairs of CT parameters with the three-parameter combination, and found the combination of all three CT parameters had the highest predictive value. Our results showed that the predictive values of CT parameters were not inferior to those of RV hypokinesia on echocardiography.

Several reports showed that RV dilatation on CT is associated with poor clinical events or death [9–14], however, the cut-off values used for RVd/LVd vary among reports. Schoepf et al. [9] used an RVd/LVd > 0.9 for prediction of 30 days mortality rate. Collomb et al. [10] suggested an RVd/LVd > 1.5, and Mansencal et al. [12] suggested an RV to LV area ratio > 1 for mortality prediction. Van der Meer et al. [11] reported that an RVd/LVd > 1 had an NPV of 100% for 3 months mortality rate. We chose RVd/LVd > 1 because ROC curve analysis showed high sensitivity and specificity.

This study has several limitations. First, we measured ventricular diameter using an axial view, which can overestimate RV diameter compared to the four-chamber view used by Schoepf et al. [9]. A reconstructed CT four-chamber view may be more accurate, but offline two-dimensional reconstruction of a standardized four-chamber cardiac view is not always available, and is time-consuming and operator-dependent. Therefore, a reconstructed four-chamber view is impractical in an emergency situation. Secondly, interventricular septal bowing depends on the cardiac cycle and could be unreliable in a non-ECG gated CT. Whereas RV volume overload produces LV deformity with septal bowing at end diastole, RV pressure overload by pulmonary embolism results in septal bowing throughout the cardiac cycle, [20, 21]. However, the degree of septal bowing might be variable according the pressure differences between the RV and LV chambers in the cardiac cycle. The third limitation was selection bias caused by small number of patients. Especially, there were no patients with chronic obstructive pulmonary disease or myocardial infarction, which could affect RV wall motion abnormalities induced by pulmonary embolism. Finally, to make our data more useful, CT parameters and cut-off values in this study need to be tested in further prospective studies.

In conclusion, our results showed that RVd/LVd, septal bowing, and proximal pulmonary embolism were associated with RV hypokinesia and adverse clinical outcomes. Because of its high sensitivity and NPV, an RVd/LVd > 1 is valuable for identifying high-risk patients. In addition to RVd/LVd, assessment of septal bowing and the location of a pulmonary embolism should be considered factors for risk stratification, because specificity and PPV were enhanced when these CT parameters were combined with RVd/LVd. The accuracy of CT measurements was comparable to echocardiographic RV hypokinesia for identification of patients with a high-risk of adverse clinical outcomes. Additional prospective studies are required to validate the predictive value of these CT parameters for identifying at-risk patients, and to assess the utility of CT findings as criteria for reperfusion therapy in patients with acute pulmonary embolism.

Conflicts of interest None.

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