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ORIGINAL ARTICLE

Relation between pulmonary embolus volume quantified by multidetector computed tomography and clinical status and outcome for patients with acute pulmonary embolism

Kei Nakada · Takemichi Okada · Hisato Osada
Norinari Honda

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Abstract
Purpose. The aim of this study was to determine whether pulmonary embolus volume (PEV) obtained with multidetector row computed tomography is related to clinical status and outcomes.
Materials and methods. Subjects comprised 48 patients with acute pulmonary embolism (PTE). PEV was measured by tracing the contours manually and compared between sets of two groups divided by clinical status. Correlations of PEV to blood gases and D-dimer levels were investigated. PEV was tested as a predictor of clinical probability of acute PTE using Wells' criteria and as a predictor of survival after PTE by logistic regression analysis.
Results. The PEV was greater in groups with respiratory symptoms ($P < 0.001$), PTE as pretest clinical diagnosis ($P = 0.027$), and heart rate >100 beats/min ($P < 0.001$). It was smaller in subjects with concurrent malignancy ($P = 0.02$). It was correlated with $PaCO_2$ ($P = 0.04$, $\rho = -0.37$) and the D-dimer level ($P = 0.002$, $\rho = 0.46$); it was not a predictor of clinical probability of acute PTE or survival after PTE. The survival rate did not differ between groups with PEV > 10 ml (8/9) or ≤10 ml (32/36).
Conclusion. The PEV in acute PTE may relate to the presence of respiratory symptoms, hypocapnia, and tachycardia. The PEV was smaller in patients with malignancy. It did not contribute to mortality in this study.

Key words Pulmonary embolus volume · Quantitative measurement · Pulmonary thromboembolism · MDCT

K. Nakada (✉) · T. Okada · H. Osada · N. Honda
Department of Radiology, Saitama Medical Center, Saitama Medical University, 1981 Kamoda, Kawagoe 350-8550, Japan
Tel. +81-49-228-3516; Fax +81-49-228-3753
e-mail: keinaka@saitama-med.ac.jp

Introduction

Pulmonary thromboembolism (PTE) is a disease in which the pulmonary arteries become occluded by dislodged thrombi formed acutely or chronically in the venous system, usually from the deep veins of the lower limbs. It has typically been diagnosed using pulmonary angiography. However, the development of multidetector row computed tomography (MDCT) with narrow collimation and the ability to perform multiplanar image reformation via a workstation has led to computed tomography pulmonary angiography (CTPA) becoming the first choice for diagnosing PTE.[1,2]

Comparison of the degree to which pulmonary artery obstruction correlates with patients' outcome has previously been investigated using pulmonary angiography.[3] Few reports have compared outcomes and the degree of obstruction according to MDCT. Qanadli et al.[4] described a semiquantitative CT obstruction index for CT angiography. Wu et al.[5] and Pech et al.[6] compared survival rates and outcomes for acute PTE using the CT obstruction index. However, the results of those two studies are conflicting. The purpose of the present study was thus to test whether embolus volume, a quantitative index, is related to symptoms and patient outcomes. To the best of our knowledge, no previous studies have reported relationships between embolus volume and patient outcome.

Materials and methods

Study population

All study protocols were approved by the institutional review board. The need for written informed consent from the patients was waived owing to the retrospective design of the study. The radiological database from December 2005 to August 2008 identified 76 consecutive patients with acute PTE who were eligible for the study. Diagnosis was based on the presence of a filling defect in the pulmonary arteries on CTPA as identified by board-certified radiologists. Among the 76 examinations, 28 were excluded because of inappropriate section thickness (>3 mm) ($n = 10$), hydrothorax ($n = 7$), extensive consolidation/ground glass opacity ($n = 3$), pneumothorax ($n = 1$), extensive postoperative lung distortion ($n = 1$), multiple metastases ($n = 1$), and past history of PTE ($n = 5$).

Examinations from a total of 48 patients (16 men, 32 women; mean age 65 years; outpatients/inpatients 24/24) were thus entered into the study. History included cancer in 16 patients, collagen disease in 2, fracture in 1, benign tumor of the female pelvis in 2, inflammatory bowel disease in 1, and puerperium (≤6 weeks after delivery) in 8. Seven patients had a history of cardiopulmonary disease (postoperative state after thoracic aortic aneurysm, $n = 1$; arrhythmia, $n = 2$; coronary heart disease, $n = 1$; old myocardial infarction with coronary-aorta bypass graft, $n = 1$; hypertension, $n = 1$; and past history of local lung surgery, $n = 1$). Heart function was normal with the left ventricular ejection fraction >60% on echocardiography.

CT imaging

All CTPA-CT venography (CTV) studies were performed using either an 8- or 16-MDCT scanner (Light Speed Ultra, GE Yokokawa, Tokyo, Japan; or Emotion 16, Siemens, Erlangen, Germany). For both 8- and 16-MDCT scanners, the automatic bolus trigger was used, with a region of interest placed over the main pulmonary artery, triggering the scanner at an enhancement level (i.e., increase in CT value of 100 HU). CTV was obtained 150 s after injection of contrast material. For all studies, 100 ml of nonionic iodinated contrast medium (Iopamiron 370, Nippon Bayer Pharmaceuticals, Tokyo, Japan; or Omnipaque 300, Daiichi-Sankyo, Tokyo, Japan) was administered intravenously at an injection rate of 3 ml/s using a power injector. Omnipaque 300 was usually employed, but Iopamiron 370 was used for patients weighing over 65 kg.

Scanning parameters used for the 8- and 16-detector-row scanners were, respectively: detector-configuration 8.0 × 1.25 mm and 16 × 0.75 mm; section thickness 1.25 mm and 1.5 mm; pitch, 1.0 and 0.7; rotation time 0.5 and 0.8 s, with 120 mA/120 kVp, and 512 × 512 matrix. Reconstruction section thickness and intervals for scanners were 1.25 mm/0.625 mm (for 8-row MDCT) and 1.5 mm/1.0 mm (for 16-row MDCT) for diagnosis and multiplanar image reformation (MPR).

Images of CTV for the pelvis and lower extremities were reconstructed with 10-mm section thickness and 10-mm intervals. All images were reviewed and interpreted on a PACS workstation (Virtual Place Lexus; AZE, Tokyo, Japan) for image analysis and on a DICOM viewer (i-PACS viewer; Konica-Minolta, Tokyo, Japan) for diagnosis of PTE and deep vein thrombosis (DVT).

Measurement of embolus volume

The data set of 1.25 or 1.5 mm thick sections with an interval of 0.625 or 1.0 mm was transferred to the workstation. Volume measurement was performed using a window width of mean CT value of the main pulmonary artery and ascending aorta on the workstation. The window level was set 100–150 HU.[7] After identifying a filling defect as far as visually recognizable down to subsegmental arteries, contours were defined manually on several transverse sections by one of the authors (K.N.). The workstation automatically interpolated contours between these selected sections and calculated the pulmonary embolus volume (PEV) based on the number of the voxels within the contours. If multiple emboli were found, the sum of each volume was used for the PEV. The defined contours were confirmed from multiple angles using the multiplanar reformation capability of the workstation.

We evaluated inter- and intraobserver reproducibility of the PEV measurement. For this analysis, we selected 20 patients based on random numbers between 1 and 48 generated by a software program (Ranpo, http://www.vector.co.jp/soft/win95/util/se123548.html). The PEV was measured on the 20 patients for defining intraobserver reproducibility by one of the authors (K.N.) 9 months after completion of the serial measurements on the 48 patients. Another author (T.O.) measured the PEV independently for defining interobserver reproducibility on a different set of 20 patients selected as described above.

Data analysis

Interobserver and intraobserver reproducibility was analyzed by linear regression analysis and Bland-Altman

plot. Embolus volumes between sets of two patient groups divided by the following indices were compared: respiratory symptoms; lower limb symptoms (swelling, pain, and reddening either uni- or bilaterally); existence of the DVT on CTV; concurrent disease; concurrent malignancy; right heart overload as detected on echocardiography; and each scoring item from Wells' criteria.[8] Two-by-two contingency tables were enumerated for (1) the survival rate grouped by embolus volume and (2) the rate of symptomatic PTE grouped by the existence of concurrent malignancy. Logistic regression analysis was performed to determine whether the PEV was a predictor of clinical probability assessed by Wells' criteria[8] or was a predictor of survival after PTE. Linear regression analysis was applied between the PEV and PaO_2, between the $PaCO_2$ and D-dimer, and between the PEV and Wells' score.

Statistical tests

A paired *t*-test was applied to comparison of means for inter- and intraobserver reproducibility. The Mann-Whitney U-test was applied to group comparisons. A two-by-two contingency table was tested using Fisher's exact probability method. Spearman's rank correlation coefficients were used to assess linear regression analysis. Values of $P < 0.05$ were deemed significant. All statistical analyses were performed using a software program (version 5.0; StatView; SAS Institute, Cary, NC, USA) running on a personal computer.

Results

Of the 48 examinations, 18 patients (37.5%) exhibited respiratory symptoms (exertional dyspnea, $n = 7$; chest pain, $n = 4$; dyspnea at rest, $n = 5$; chest oppression, $n = 2$). One patient entered a state of shock after embolectomy of PTE. Eighteen patients (37.5%) reported lower limb symptoms. Among the 14 asymptomatic patients (29%), hypoxemia was seen in 7 and an abnormal D-dimer level in 3. Measurements of arterial blood gases were conducted in 32 of the 48 patients (mean PaO_2 73 mmHg, range 28.4–99.0 mmHg; mean $PaCO_2$ 33.5 mmHg, range 22.0–42.9 mmHg). Measurement of D-dimer (LPIA-ACE D-dimer; Mitsubishi Chemical Medience, Tokyo, Japan) was conducted for 45 patients (mean 14.1 μg/ml; range 1.57–29.45 μg/ml). Echocardiography was conducted in 24 patients, and 6 patients showed right heart overload.

Interobserver and intraobserver reproducibility was good as shown by linear regression analysis and Bland-Altman plots (Fig. 1). Means of the PEV were not statistically different in intraobserver comparisons (8.9 and 8.3 ml, respectively; $P = 0.425$, paired *t*-test), and interobserver comparisons (8.5 and 8.1, respectively; $P = 0.547$, paired *t*-test).

The PEV was significantly greater in patients displaying respiratory symptoms than in patients without such symptoms ($P < 0.001$) (Fig. 2a). Lower limb symptoms, symptomatic deep venous thrombosis, and concurrent disease were not significantly associated with PEV ($P = 0.09$, $P = 0.64$, and $P = 0.05$, respectively). However, embolus volume was smaller in patients with malignancy than in patients without malignancy ($P = 0.02$) (Fig. 2b). Furthermore, the rate of asymptomatic PTE was higher in patients with malignancy (Table 1). Other results of group comparisons are shown in Table 2. Among scoring items for Wells' criteria, the subjective probability of

Table 1. Relation between symptoms and concurrent malignancy

Condition	Concurrent malignancy	No concurrent disease	Total
Symptoms	7	17	24
No symptoms	9	1	10
Total	16	18	34

Fisher exact probability: $P = 0.002$

Table 2. Relation between clinical status and pulmonary embolus volume

Item	Median pulmonary embolus volume (ml)		
	Item present	Item absent	*P*
Respiratory symptom (48)	9.5 (19)	0.7 (29)	0.0003*
Lower limb symptom (48)	0.7 (18)	4.0 (30)	0.09
Symptomatic DVT (34)	0.7 (17)	1.5 (17)	0.64
Concurrent disease (48)	0.7 (30)	3.8 (18)	0.052
Concurrent malignancy (34)	0.5 (16)	3.8 (18)	0.02*
Right heart load on UCG (24)	16.1 (6)	2.4 (18)	0.07

The numbers in parentheses represent the number of patients
DVT, deep vein thrombosis; UCG, ultrasonic echocardiography

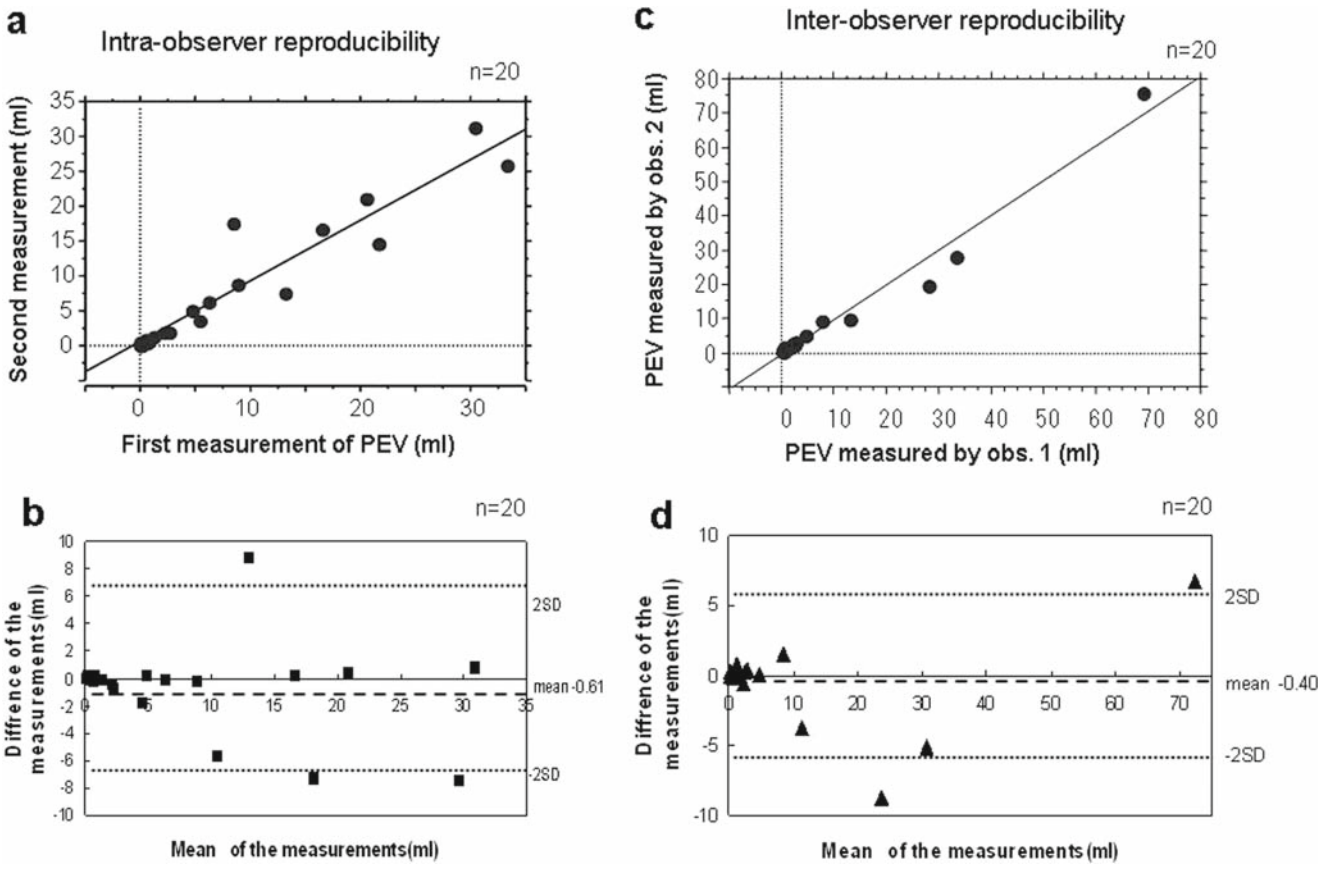

Fig. 1. Intraobserver (**a**, **b**) and interobserver (**c**, **d**) reproducibility of pulmonary embolus volume (*PEV*) measurements. There was good, though not perfect, reproducibility in the measurements demonstrated by linear regression analysis (**a**, **c**) and Bland-Altman (**b**, **d**) plots. The mean difference of measured PEV in the same patients was nearly 0, showing no bias between the measurements. Standard deviations of the difference of the measurement were 3.3 ml (**b**) and 2.8 ml (**d**)

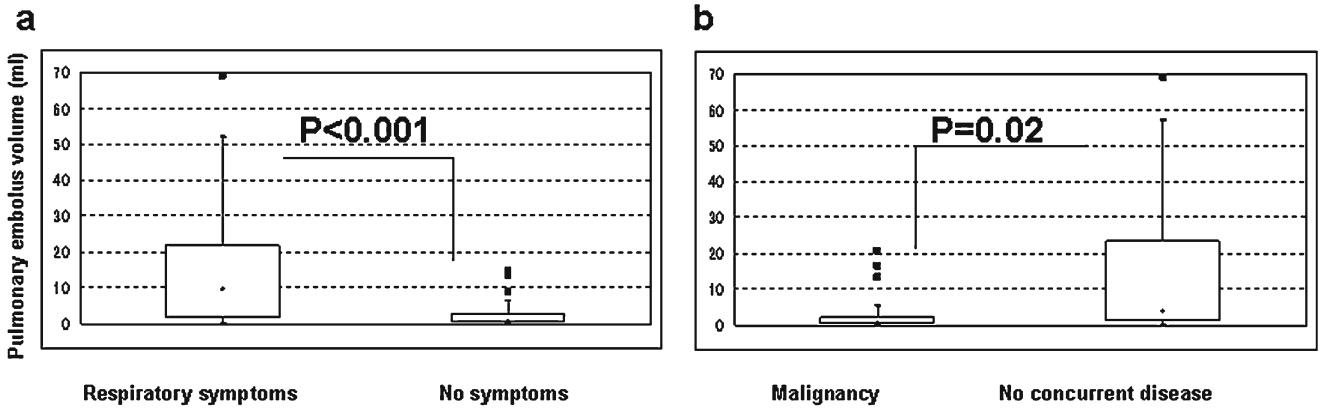

Fig. 2. Box plot showing PEV in patients with and without respiratory symptoms (**a**) and in patients with malignancy and without concurrent disease (**b**)

PTE according to physicians, heart rate >100 beats/min, and concurrent malignancy were significantly associated with embolus volume (Table 3).

Follow-up CTPA was available for 29 patients, with a study interval of 3–120 days. Emboli disappeared in 20 patients with a median disappearance time of 16.5 days. Embolus volume was significantly smaller in patients with a disappearance time of <16.5 days than in patients with disappearance time ≥16.5 days or incomplete resolution ($P = 0.02$) (Fig. 3).

Table 3. Relation between Wells' criteria and pulmonary embolus volume

Scoring item	Median pulmonary embolus volume (ml)		*P*
	Item present	Item absent	
Subjective probability of PTE	8.1 (16)	0.8 (32)	0.027*
Suspected DVT	0.7 (16)	2.6 (32)	0.128
Tachycardia (>100 beats/min)	14.1 (10)	0.7 (31)	0.0005*
Surgery or immobilization in previous 4 weeks	2.6 (21)	1.2 (27)	0.5127
Hemoptysis	(0)	(48)	>0.999
Active malignant disease	0.5 (16)	3.8 (32)	0.027*

The numbers in parentheses represent the number of patients
PTE, pulmonary embolism
*Significant difference (<0.05)

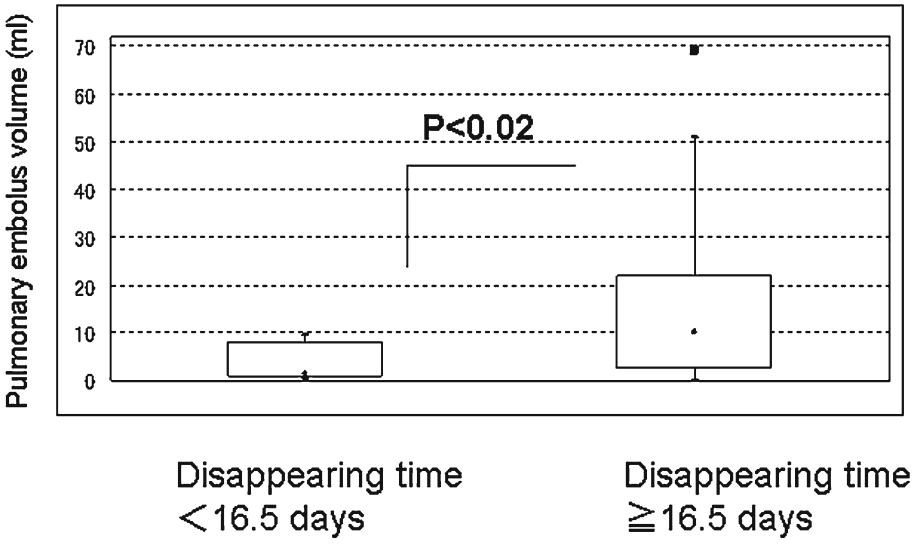

Fig. 3. Box plot showing PEV in patient groups with disappearance times of <16.5 days and ≥16.5 days. PEV was significantly smaller in patients with a disappearance time of <16.5 days

Of the 48 patients, 4 died of concurrent illness (colon cancer, $n = 1$; ovarian cancer, $n = 2$; rheumatoid arthritis, $n = 1$), and another after pulmonary thrombectomy. Three patients were lost to follow-up. As a result, 40 patients survived (80%) with a minimum follow-up of 2 months. Embolus volume was not significantly smaller in surviving patients than in dead patients ($P > 0.69$) (Fig. 4a). Survival rates did not differ irrespective of whether the embolus volume was ≥10 ml (Fig. 4b).

Logistic regression analysis showed that the embolus volume was not predictive for survival after PTE (Table 4) or clinical probability of PTE as determined by Wells' criteria (Table 5).

No significant correlations were seen with PaO_2 ($P = 0.16$; $\rho = -0.24$). A weak negative correlation was found between PEV and $PaCO_2$ ($P = 0.04$; $\rho = -0.37$). A significant positive correlation was identified with the D-dimer level ($P = 0.002$; $\rho = 0.46$). There was no correlation between PEV and Wells' score ($P = 0.12$; $\rho = -0.003$). These results are shown in Fig. 5.

Discussion

The method for diagnosis of PTE has shifted from pulmonary angiography or scintigraphy to MDCT. However, few previous studies have investigated relations between the degree of pulmonary arterial obstruction on MDCT and patient outcomes.[3–6,9,10] We measured embolus volume as a quantitative index rather than as a semiquantitative CT obstruction index. To the best of our knowledge, this is the first study to correlate embolus volume to clinical symptoms and patient outcomes.

Measurement of PEV showed relatively good reproducibility. Factors confounding reproducibility are image artifact, partial volume averaging, and window width/level setting. Streak artifact originating from a densely enhanced superior vena cava may overlap with the embolus, causing its contour to be unrecognizable. Connective tissue surrounding the pulmonary artery makes distinction among embolus, arterial wall, and connective tissue difficult. As a result, a nonfloating large embolus is more prone to be associated with a large

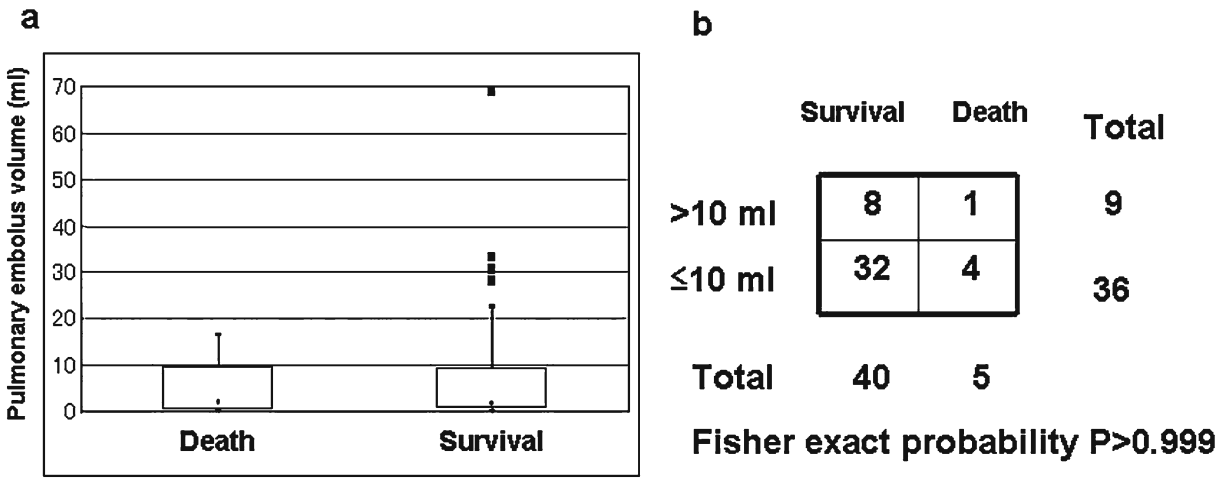

Fig. 4. Box plot (**a**) and two-by-two contingency table (**b**) showing the relation between embolus volume and prognosis. Embolus volumes were not significantly different between survived patients and succumbed patients (**a**). Survival rates were not significantly different between patient groups with embolus volume >10 ml and ≤10 ml (**b**)

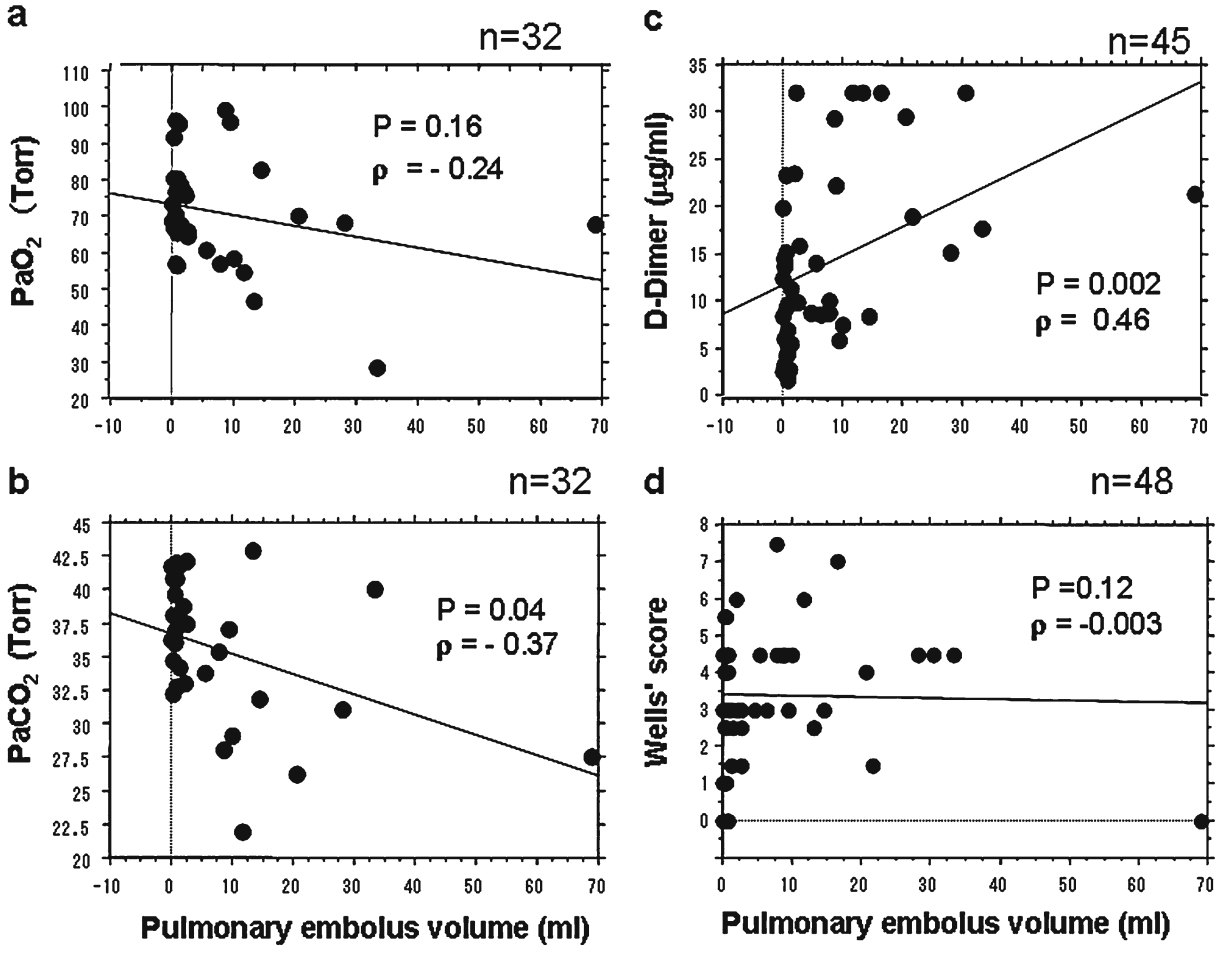

Fig. 5. Correlations between PEV and clinical indexes. There was no correlation between PEV and PaO_2 (**a**), although there was a weak negative correlation between PEV and $PaCO_2$ (**b**) and a positive correlation between PEV and the D-dimer level (**c**). There was no significant correlation between PEV and Wells' score

Table 4. Logistic regression analysis of survival after acute pulmonary embolism

	Survival after PTE	
Predictor	Regression coefficient	*P*
Pulmonary embolus volume, ml	0.841	0.4482
Wells' score	–0.383	0.6164
Age	–0.120	0.1930
Concurrent malignancy: present	2.556	0.3673
Concurrent disease: present	–36.630	0.9927
Sex		
Female	–49.568	0.9910
Male	15.975	0.9967
Respiratory symptoms: present	–29.999	0.9949
Lower limb symptoms: absent	28.766	0.5023
Deep vein thrombosis: present	–0.404	0.5116

Table 5. Logistic regression analysis of disease probability judged by Wells' score as high, intermediate, or low probability

	Disease probability: high vs. intermediate		Disease probability: high vs. low	
Predictor	Regression coefficient	*P*	Regression coefficient	*P*
Pulmonary embolus volume, ml	–0.508	0.9764	–0.513	0.9762
Concurrent malignancy: present	–0.381	0.9998	1.411	0.9994
Concurrent disease: present	–26.191	0.9950	–55.804	0.9896
Prognosis: survived	1.548	0.9995	49.257	0.9939
Sex				
Female	–30.147	0.9994	46.552	0.9937
Male	2.087	0.9879	16.059	0.9967
Respiratory symptoms: present	–29.999	0.9855	–58.919	0.9797
Lower limb symptoms: absent	28.766	0.9998	72.881	0.9753
Deep vein thrombosis: present	–0.404	0.9985	–14.781	0.9945

variation in PEV (Fig. 1). Partial volume averaging is always a problem when measuring volume from tomographic data. We tried to overcome this problem by decreasing the slice thickness and overlapping. The window width and level must be carefully set when measuring embolus volume. Previous studies have shown that window settings might hamper or emphasize the visibility of small, isolated segmental and subsegmental emboli.[7] We set the window level at 100–150 HU.[7] We set a more narrower window width than Storto et al.[7] because a wide setting (e.g., 600 HU) blurs the contours of the embolus and makes inhomogeneous pulmonary venous enhancement more prominent, which is confusing with an embolus.

We found that asymptomatic PE was more frequent in patients with malignancy (Table 1), and that the PEV was significantly smaller in patients with malignancy (Fig. 2b). Storto et al. noted that unsuspected pulmonary embolisms are found in a significant number of patients undergoing a routine MDCT study of the chest, with a higher prevalence among patients with malignancy.[7] Gladish et al. reported that incidental pulmonary emboli were seen in oncology patients and not detected because of their small size on initial CT image interpretation.[11] These results are concordant with the results of this study, but quantitative analysis of PEV was undertaken in our study only.

The prevalence of asymptomatic or incidental pulmonary embolism was not studied here. Others have reported that incidental pulmonary emboli are observed in about 2.6% of cases using a thick-section technique with 5- to 8-mm collimation contrast-enhanced CT of the chest.[12] Boswell et al. identified incidental PEs in 2.1% of patients, with most of the emboli in the second- to fourth-order lower lobe branches. They found large saddle embolus in nine patients (21% of positive cases),[13] whereas we found no large saddle emboli in our patients with malignancy.

A high frequency of asymptomatic pulmonary embolism has been reported in patients with DVT.[14] DVT and PE are related conditions and together have been categorized as venous thromboembolism. We assumed that symptomatic PTE involves a larger embolus than asymptomatic PTE. In this study, the embolus volume was

significantly greater in patients with respiratory symptoms (Fig. 2a). This is reasonable, as a large embolus volume results in greater extent of pulmonary artery obstruction.

Most pulmonary emboli are thought to migrate from the deep veins of the lower limbs to the lungs. We thus hypothesized that PEVs would be smaller in patients with lower limb symptoms than in patients without such symptoms, as symptomatic thrombus may be more adherent to the venous wall. Our results (Table 2) did not support this hypothesis, however, although the PEV was smaller—but not significantly so—in patients with lower limb symptoms.

Wells' score is the most validated and utilized clinical pretest probability score. This study was performed to determine whether PEV was a predictor of disease probability as estimated by Wells' scores. The linear regression analysis of Wells' score and PEV (Fig. 5d) showed no correlation, which is concordant with our results of the logistic regression analysis (Table 5). The analysis showed that embolus volume was not a determining factor for disease probability. This may mean that Wells' score is unaffected by embolus volume, which is a good feature for detecting even small PTEs. Conversely, subjective pretest probability of PTE by physicians (one item in Wells' score) was significantly associated with embolus volume, showing that this factor has a special value in Wells' score.

Embolus volume was not associated with patient survival in this study. Similar results were obtained by Pech et al.[6] Among 29 patients followed with CT, the embolus volume related to the disappearance time. Follow-up data from The Japanese Society of Pulmonary Embolism Research (JaSPER) study[15] revealed that the long-term survival rate was mainly determined by concurrent malignant disease. Miyahara et al. reported that recurrence and death rates during the remote period are considerably low, indicating the importance of treatment and secondary prevention during the acute stage.[15] Quantifying the embolus volume may become an effective tool for predicting the disappearance time of embolus and for planning follow-up intervals.

The lack of correlation between embolus volume and PaO_2 may be due to administration of supplemental oxygen. Clinical records were unclear regarding whether patients had been given oxygen. PEV correlated well with the D-dimer value. This result is not unexpected, as D-dimer is the breakdown product of cross-linked fibrin.

Metafratzi et al. found that ventilation and $PaCO_2$ are closely and inversely related to the CT obstruction index, and their results suggested that the extent of pulmonary artery obstruction regulates the level of hyperventilation response.[16] Similar results were obtained in our study: The embolus volume was inversely and significantly correlated with $PaCO_2$ (i.e., the extent of hyperventilation).

Some limitations of this study need consideration. The window width and level must be carefully set when measuring embolus volume. In addition, it is important to recognize special false-positive CT phenomena when detecting and measuring the embolus. In particular, some emboli may be confused with nearby mediastinal interstitial tissue of the specific site.[17] Finally, we did not evaluate the ratio of embolus volume to total pulmonary artery volume.

Conclusion

Our results raise the possibility that the embolus volume in acute PTE, representing a quantitative index from MDCT, is related to the presence of respiratory symptoms, the $PaCO_2$ value, and the heart rate. Furthermore, embolus volumes were shown to be smaller in patients with malignancy. Embolus volume may not directly contribute to mortality.

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