Cardiac Imaging

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Abbreviations:
ANTELOPE = Advances in New Technologies Evaluating the Localization of PE
PE = pulmonary embolism
RVD = right ventricular dysfunction
RV/LV = right ventricle to left ventricle short-axis diameters

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Right Ventricular Dysfunction and Pulmonary Obstruction Index at Helical CT: Prediction of Clinical Outcome during 3-month Follow-up in Patients with Acute Pulmonary Embolism[1]

PURPOSE: To retrospectively quantify right ventricular dysfunction (RVD) and the pulmonary artery obstruction index at helical computed tomography (CT) on the basis of various criteria proposed in the literature and to assess the predictive value of these CT parameters for mortality within 3 months after the initial diagnosis of pulmonary embolism (PE).

MATERIALS AND METHODS: Institutional review board approval was obtained, and informed consent was not required for retrospective study. In 120 consecutive patients (55 men, 65 women; mean age ± standard deviation, 59 years ± 18) with proved PE, two readers assessed the extent of RVD by quantifying the ratio of the right ventricle to left ventricle short-axis diameters (RV/LV) and the pulmonary artery to ascending aorta diameters, the shape of the interventricular septum, and the extent of obstruction to the pulmonary artery circulation on helical CT images, which were blinded for clinical outcome in consensus reading. Regression analysis was used to correlate these parameters with patient outcome.

RESULTS: CT signs of RVD (RV/LV ratio, >1.0) were seen in 69 patients (57.5%). During follow-up, seven patients died of PE. Both the RV/LV ratio and the obstruction index were shown to be significant risk factors for mortality within 3 months (P = .04 and .01, respectively). No such relationship was found for the ratio of the pulmonary artery to ascending aorta diameters (P = .66) or for the shape of the interventricular septum (P = .20). The positive predictive value for PE-related mortality with an RV/LV ratio greater than 1.0 was 10.1% (95% confidence interval [CI]: 2.9%, 17.4%). The negative predictive value for an uneventful outcome with an RV/LV ratio of 1.0 or less was 100% (95% CI: 94.3%, 100%). There was a 11.2-fold increased risk of dying of PE for patients with an obstruction index of 40% or higher (95% CI: 1.3, 93.6).

CONCLUSION: Markers of RVD and pulmonary vascular obstruction, assessed with helical CT at baseline, help predict mortality during follow-up.

Pulmonary embolism (PE) is a common and potentially fatal cardiovascular disorder. Even when PE is properly treated with anticoagulant therapy, the mortality rate in hemodynamically stable patients varies from 2% to 7% (1–3). Mortality is thought to be caused in part by pressure overload of the right ventricle secondary to acute pulmonary arterial hypertension caused by PE. This initially results in right ventricular dysfunction (RVD), which may progress to right ventricular failure and circulatory collapse (4). It has been reported that a substantial proportion (40%) of normotensive patients with acute PE presents with echocardiographic signs of RVD. These patients with latent hemodynamic impairment have a 10% rate of PE-related shock and a 5% rate of in-hospital mortality (5). In contrast, normotensive patients without RVD have a more benign short-term prognosis (0% rate of PE-related shock or in-hospital mortality) (5). Thus, the presence of RVD is a marker for adverse clinical outcome in patients with acute PE. Echocardiography is recommended as a first-line examination to diagnose the signs of RVD. However, since helical computed tomography (CT) is now more and more routinely being used as the first-line technique to diagnose PE, assessment of RVD at CT would facilitate patient diagnosis.

In small studies with helical CT, the ratio of the right ventricle to left ventricle short-axis diameters (RV/LV) has been proposed as an accurate sign for the presence of RVD (6–9). In addition, other criteria have been proposed, including deviation of the interventricular septum and the ratio of the pulmonary artery to ascending aorta diameters (10). Also, the extent of PE (ie, the arterial thrombus load in the pulmonary arteries) has been proposed as an important parameter for predicting RVD (8,11,12). Thus, the aim of the present study was to retrospectively quantify RVD and the pulmonary artery obstruction index with helical CT on the basis of the various criteria proposed in the literature and to assess the predictive value of these CT parameters for mortality within 3 months in consecutive patients who are initially hemodynamically stable after the initial diagnosis of PE.

MATERIALS AND METHODS

Patients

For this retrospective study, we used the patient data and CT image database previously obtained in a prospective cohort of 510 patients suspected of having PE (the Advances in New Technologies Evaluating the Localization of PE [ANTELOPE] study). This cohort was originally studied in a multicenter trial designed to investigate the safety of relying on negative results at helical CT to rule out PE. A detailed description of the study population and the results of the diagnostic trial have been published previously (13). In brief, study participants were consecutive inpatients and outpatients who were suspected of having PE at clinical presentation and who had not yet undergone diagnostic testing for PE. Exclusion criteria were age younger than 18 years, pregnancy, initiation of thrombolytic therapy because of hemodynamic instability, and/or failure to obtain written informed consent. All patients underwent contrast material–enhanced helical CT of the pulmonary arteries to detect or exclude PE.

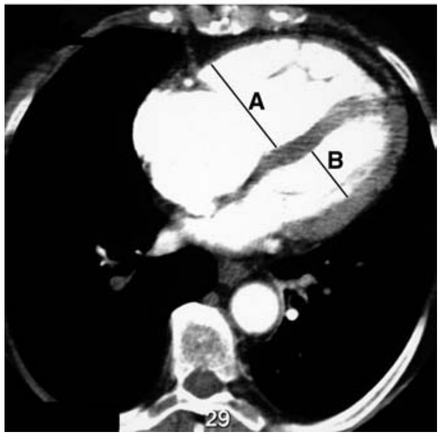

Figure 1. Transverse contrast-enhanced CT scan shows maximum minor axis measurements of the right ventricle *(A)* and left ventricle *(B)*. Note the flattening of the interventricular septum. RV/LV ratio = 1.81.

The institutional review boards of all participating institutions approved the ANTELOPE prospective study and the present retrospective study, and informed consent was obtained from all patients for the prospective cohort study but was not required for the retrospective study.

During the inclusion period of the ANTELOPE study (between April 1999 and May 2000), 704 consecutive patients were suspected of having acute PE at clinical presentation at the participating centers. Of these 704 patients, 512 were included, and the other 192 patients met one or more of the stated exclusion criteria. (Five of the 192 patients were excluded because they were hemodynamically unstable, and thrombolytic therapy had been initiated.) Helical CT performed in 510 patients (in one of the 512 patients, contrast agents were contraindicated; another patient could not maintain a supine position) demonstrated PE in 124 (24%) patients. These 124 patients were all initially hemodynamically stable and were all treated by means of anticoagulant therapy (13). Scans from three CT examinations could not be retrieved (none of the three patients died during the follow-up period), and one CT scan did not depict the cardiac ventricles (but the obstruction index in this patient could be measured).

Thus, results of CT in 120 patients (mean age, 59 years ± 18 [± standard deviation]; range, 18–89 years) were available for our retrospective analysis. There were 55 men (mean age, 58 years ± 16; range, 18–86 years) and 65 women (mean age, 60 years ± 20; range, 20–89 years). There was no significant difference in age between the male and female patients (P = .635).

Imaging Studies

CT was performed by using single–detector row helical CT scanners. Scanning parameters were 120 kV, 210 mAs, 5-mm collimation, a pitch of 1, and a reconstruction interval of 3 mm. Tube rotation time was 1000 msec, and a table feed of 5 mm per second was used. A 16-cm volume in the caudocranial direction was scanned from the top of the diaphragm to a level slightly above the aortic arch during a single 32-second breath hold. In dyspneic patients, scanning was performed during shallow, gentle respiration. Standardized dose rate and total dose of nonionic contrast agent were used, and image acquisition was started after a scanning delay of 15–20 seconds after the start of the intravenous injection of contrast medium. Film hard-copy images were obtained at standard mediastinal settings (window width, 350 HU; window level, 50 HU) and lung settings (window width, 1500 HU; window level, −500 HU).

CT Signs of RVD

The hard-copy CT images in patients with positive diagnosis of PE were selected and read by two observers (P.M.T.P. and R.W.v.d.M., with 9 years and less than 1 year of experience in examining thoracic CT scans, respectively) in a consensus reading who were unaware of the clinical signs and symptoms and the patient's condition at the time of initial presentation. They were unaware of the clinical outcome, as well. Although P.M.T.P. was one of the investigators in the ANTELOPE study, he did not interpret the CT scans for that study. R.W.v.d.M. was a resident in radiology. The observers were aware that PE had been previously diagnosed.

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 may be found at

TABLE 1
Survival Time among 18 Patients Who Died during the 3-month Follow-up Period

Patient No.	Cause of Death	Day of Death*
1	PE	0
2	PE	1
3	PE	2
4	PE	5
5	PE	6
6	PE	7
7	PE	17
8	Myocardial infarction	20
9	Myocardial infarction	63
10	Malignancy	3
11	Malignancy	5
12	Malignancy	16
13	Malignancy	43
14	Malignancy	45
15	Malignancy	49
16	Diverticulitis	9
17	Respiratory failure	51
18	Neurologic damage after cardiopulmonary resuscitation	8

* The day of inclusion is considered day 0.

different levels. The RV/LV ratio was then calculated. CT scans were considered to show no RVD if the ratio was 1.0 or less, modest RVD if the ratio was greater than 1.0 but less than or equal to 1.5, and severe RVD if the ratio was greater than 1.5, as recommended in the literature (7–9). Deviation of the interventricular septum was evaluated on a three-point scale, as follows: score of 1, normal septum (ie, convex toward the right ventricle); score of 2, flattened septum; and score of 3, septum deviation convex toward the left ventricle (7,8).

The diameters of the main pulmonary artery and ascending aorta (the diameters of the inner lumina) were measured at a single predefined transverse scanning level, that is, that at which the right pulmonary artery is in continuity with the main pulmonary artery and sweeps across the midline. The pulmonary artery to ascending aorta diameter ratio was then calculated (10).

Degree of Vascular Obstruction

We quantified the vascular obstruction index (ie, the percentage of vascular obstruction of the pulmonary arterial tree caused by PE) by using the scoring system of Qanadli et al (11). In brief, this index is defined as the number of segmental artery branches that are blocked and corrected by a factor of one for partial blockage or a factor of two for completely obstructive PE. With this scoring system, the highest possible score is 40 (thrombus completely obstructing the pulmonary trunk), which corresponds to a 100% obstruction index.

Three-month Follow-up

All patients with acute PE were administered intravenous unfractionated heparin for at least 5 days; the aim of this treatment was a prolongation of activated partial thromboplastin time by a factor of 1.5–2.5. Vitamin K antagonists were given for a period of at least 3 months with the aim of an international normalized ratio of 2.0–3.0. During the follow-up period, all patients received routine clinical care from their physicians. All patient deaths during the 3 months of follow-up had been prospectively registered and evaluated by an independent adjudication committee that had full access to all available clinical and diagnostic patient data. The adjudication committee determined and recorded whether a patient 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 commercially available software (SPSS version 10.0 for Windows; SPSS, Chicago, Ill). The association between death and the RV/LV ratio, the pulmonary artery to ascending aorta ratio, and the obstruction index was determined by using Cox regression analysis corrected for age. Only deaths caused by PE were considered; deaths from other causes were censored. Regression coefficients and *P* values are reported for continuous variables; the hazard ratio and confidence intervals are reported for the obstruction index as a categoric variable. The RV/LV ratio, the pulmonary artery-to-ascending aorta ratio, and the obstruction index are expressed as mean ± standard deviation, and 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 obstruction index among the patients, who were 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 correction for multiple post hoc testing.

RESULTS

Clinical Follow-up

Three-month follow-up was completed in all patients. During the follow-up period, 18 (15%) patients died. According to the independent adjudication committee, death was related to PE in seven patients (definitely related to PE in six patients, most probably related to PE in one patient), none of whom had a history of cardiac failure or chronic obstructive pulmonary disease. Death in the remaining 11 patients was assumed to be directly related to malignancy ($n = 6$), myocardial infarction ($n = 2$), respiratory failure ($n = 1$), diverticulitis ($n = 1$), or neurologic damage after cardiopulmonary resuscitation ($n = 1$). Table 1 lists the duration of survival after presentation for these 18 patients.

RV/LV Ratio

The mean RV/LV ratio was 1.17 ± 0.37 (Table 2). There was a statistically significant relationship between the RV/LV ratio and PE-related mortality, with a regression coefficient of 1.55 ($P = .04$, Table 3). The mean RV/LV ratio was 1.17 ± 0.37 (Table 2). When the patients were divided into three groups (patients who died of PE, patients who survived, and patients who died of a cause unrelated to PE), results of the overall test for equality for the three groups together showed a statistically significant difference in the mean RV/LV ratios ($P = .018$). 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 (1.54 ± 0.18 vs

TABLE 2
CT Measurements in Patients with Proved PE

Parameter	All Patients (n = 120)	RV/LV Ratio >1.5 (n = 18)	RV/LV Ratio >1.0 and ≤1.5 (n = 51)	RV/LV Ratio ≤1.0 (n = 51)	P Value*
Maximum diameter (mm)†					
Right ventricle	46.9 ± 8.3	57.8 ± 7.4	48.2 ± 7.1	41.8 ± 4.9	<.001
Left ventricle	42.1 ± 7.8	31.4 ± 4.5	40.5 ± 5.5	47.4 ± 6.0	<.001
RV/LV ratio†	1.17 ± .37	1.86 ± .31	1.20 ± .14	0.89 ± .10	<.001
Interventricular septum‡§					
Normal	71 (59)	0 (0)	24 (47)	47 (92)	<.001
Displacement grade 2	24 (20)	4 (22)	16 (32)	4 (8)	
Displacement grade 3	25 (21)	14 (78)	11 (22)	0 (0)	
PA/Ao ratio†‖	0.92 ± .14	0.92 ± .13	0.88 ± .12	0.95 ± .16	.076
Obstruction index (%)†	31.8 ± 22.9	62.8 ± 20.8	32.0 ± 21.5#	20.9 ± 13.2	<.001
Obstruction index categoric‡					
<40%	83 (69)	2 (11)	35 (70)	45 (88)	<.001
≥40%	37 (31)	16 (89)	15 (30)	6 (12)	

* Refers to overall test for equality of means or proportions.
† Data are mean ± standard deviation.
‡ Data are numbers of patients, and numbers in parentheses are percentages.
§ Grade 2 = flattened, grade 3 = deviated toward the left ventricle.
‖ PA/Ao ratio = ratio of inner lumen pulmonary artery diameter to aorta diameter.
\# In one patient, the obstruction index could not be determined because of complete pneumectomy of one lung.

1.14 ± 0.03, P = .005) and that in patients who died of a cause unrelated to PE (1.17 ± 0.08, P = .033). There was no significant difference in the mean RV/LV ratio between patients who died of causes unrelated to PE and patients who survived (P = .796) (Fig 2).

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 our study, 51 (42.5%) patients had an RV/LV ratio of 1.0 or less (no deaths from PE), 51 (42.5%) patients had an RV/LV ratio greater than 1.0 but less than or equal to 1.5 (four deaths from PE, 8%), and 18 (15%) patients had an RV/LV ratio greater than 1.5 (three deaths from PE, 17%). Results of analysis of variance testing with least significant difference as a post hoc test showed a significant increase in age (P < .001) for patients in both groups with an elevated RV/LV ratio compared with patients with a RV/LV ratio of 1.0 or less. There was no significant difference in age distribution between the two groups with elevated RV/LV ratios (P = .325). There was no significant difference in sex distribution (χ^2 test, P = .559) between the three groups. The positive predictive value for PE-related mortality with an RV/LV ratio greater than 1.0 was 10.1% (95% confidence interval: 2.9%, 17.4%), and the negative predictive value for an uneventful outcome with an RV/LV ratio of 1.0 or less was 100% (95% confidence interval: 94.3%, 100%). In our study population, an RV/LV ratio of 1.0 or less excluded mortality due to PE.

TABLE 3
Univariate Cox Regression Analysis of Death due to PE during 3-month Follow-up in Patients Treated with Anticoagulant Therapy

Parameter	Regression Coefficient	Standard Error	P Value
RV/LV ratio	1.55	0.74	.035
Obstruction index	0.041	0.016	.009
PA/Ao ratio*	1.36	3.08	.66

* PA/Ao ratio = ratio of inner lumen pulmonary artery diameter to aorta diameter.

CT-derived Vascular Obstruction Index

The mean value of the vascular obstruction index was 31.8% ± 22.9 (Table 2); in one case, a patient had undergone complete pneumectomy of one lung, and so this case was censored for this measurement. Results of Cox regression analysis showed a statistically significant relationship between the vascular obstruction index and PE-related mortality (P = .01, Table 3).

When the patients were divided into three groups (patients who died of PE, patients who survived, and patients who died of a cause unrelated to PE), results of the overall test for equality for the three groups together showed a statistically significant difference in the mean obstruction indexes (P = .001). 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 (60.4% ± 28.4 vs 29.3% ± 20.8, P < .001) or patients who died of a cause unrelated to PE (36.8% ± 26.3, P = .027). There was no significant difference in the average obstruction index between patients who died of causes unrelated to PE and patients who survived (P = .280) (Fig 3).

When the previously recommended cutoff value of 40% for the pulmonary vascular obstruction index was used (11), patients with an index of 40% or greater had an 11.2-fold increased risk of dying of PE (despite anticoagulant treatment) relative to patients with an index of less than 40% (hazard ratio, 11.2; 95% confidence interval: 1.3, 93.6). Six of 37 patients with an obstruction index of 40% or greater died of PE versus one of 83 patients with an obstruction index of less than 40%. The positive predictive value for PE-related mortality with an obstruction index of 40% or greater was 16.2%

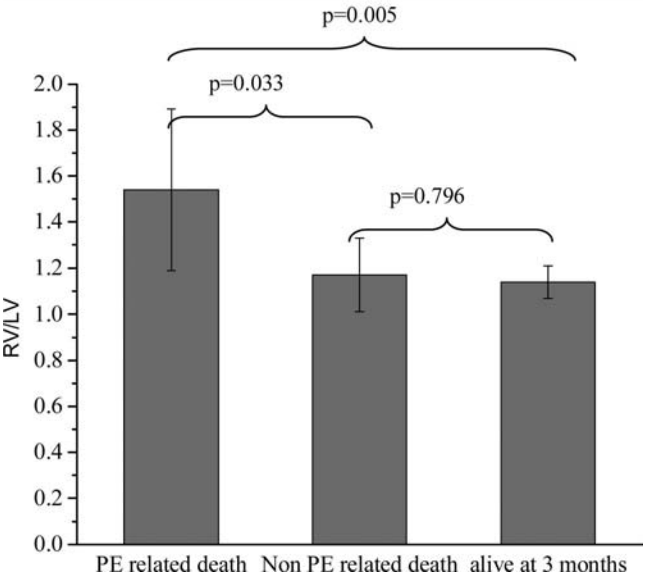

Figure 2. Graph of mean values of the RV/LV ratio relative to clinical outcome. Bars represent means and lines represent 95% confidence intervals. The mean RV/LV ratio was significantly higher in patients who died of PE than in the other patients (analysis of variance, $P = .018$). Brackets show significant differences between groups.

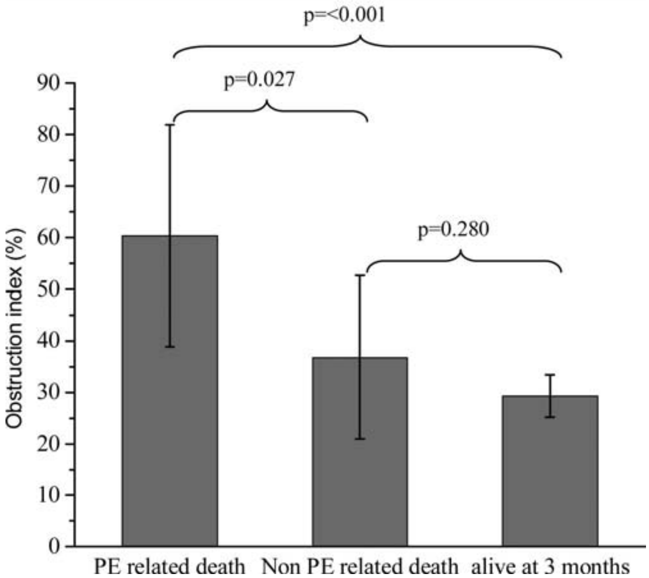

Figure 3. Graph of mean values of obstruction index relative to clinical outcome. Bars represent means and lines represent 95% confidence intervals. The mean obstruction index was significantly higher in patients who died of PE than in the other patients (analysis of variance, $P = .001$). Brackets show significant differences between groups.

(95% confidence interval: 4.1%, 28.3%). The negative predictive value for an uneventful outcome with an obstruction index of less than 40% was 98.8% (95% confidence interval: 96.4%, 100%).

There was a significant ($P < .001$) but weak correlation between the RV/LV ratio and the vascular obstruction index, OBS, determined with the regression equation: RV/LV ratio = 0.0099 · OBS + 0.85 (Pearson $r^2 = 0.38$). The positive predictive value for PE-related mortality among patients with an RV/LV ratio greater than 1.0 and an obstruction index of 40% or greater was 18.8% (95% confidence interval: 5.0%, 32.6%), and the negative predictive value for an uneventful outcome among patients with an RV/LV ratio of 1.0 or less or an obstruction index of less than 40% was 98.9% (95% confidence interval: 96.7%, 100%).

Displacement of the Interventricular Septum

Displacement of the interventricular septum was noted in 49 of 120 patients, as follows: flattening of the septum in 24 (20%) patients and inversion convex toward the left ventricle in 25 (21%) patients. 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. We found no significant relationship between the shape of the interventricular septum and PE-related mortality ($P = .20$). There was a relationship between the RV/LV ratio and septum displacement. The shape of the interventricular septum was abnormal in all 18 patients with severe RVD, in 27 (54%) patients with modest RVD, and in only four (8%) patients without RVD ($P < .001$).

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 0.92 ± 0.14. We did not find a significant relationship between this ratio and PE-related mortality ($P = .66$) or between this ratio and the RV/LV ratio ($P = .08$).

DISCUSSION

Results of this study showed that the presence and severity of RVD and the extent of obstruction of the pulmonary arterial tree as assessed at helical CT help predict mortality within 3 months of clinical presentation in initially hemodynamically stable patients with PE. It is important to note that both the absence of RVD as assessed by means of an RV/LV ratio of 1.0 or less and an obstruction index of less than 40% were predictive for an uneventful outcome. In contrast, the deviation of the interventricular septum and the ratio between the pulmonary artery and ascending aorta diameters seemed to have no prognostic relevance in acute PE. It is of interest that clinically relevant parameters of RVD can be obtained even from CT examinations that are not gated to the electrocardiogram.

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 (4,5,14). As an alternative, blood tests, including measurement of plasma brain natriuretic peptides (15,16) or cardiac troponins T and I (17), have been proposed as prognostic indicators for benign versus complicated courses. ten Wolde and colleagues (15) showed that the adjusted odds ratio of a brain natriuretic peptide level greater than 21.7 pmol/L for death related to PE was 14.1 (95% confidence interval: 1.5, 131.1), while in another study, a cutoff level of a brain natriuretic peptide level of less than 50 pg/L enabled identification of 95% of patients with a benign clinical course

(16). Konstantinides et al (17) found that 35%–40% of patients with PE had elevated cardiac troponin levels that were associated with overall mortality and a complicated course.

Some limitations of our study should be considered. First, imaging was performed with single–detector row helical CT scanners. At the time of investigation, multi–detector row CT scanners were not yet available in the participating hospitals. To our knowledge, there currently are no reports of studies available in the literature in which RV/LV ratios assessed with single–detector row technology have been compared with those assessed with multi–detector row CT scanners. We admit that this newer CT technology could have been helpful for better imaging of the subsegmental arteries. It remains to be studied whether the results will be different at multi–detector row CT, since subsegmental arteries have only a minimal influence on the obstruction index. Second, 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.

Regardless of the small numbers, our findings are in agreement with those of Wu et al (12), who suggested that the quantification of a clot at CT pulmonary angiography is an important predictor of patient death in the setting of PE, whereas authors of a previous study with an even smaller number of PE-related deaths (18) did not find a significant relationship between RVD or the extent of vascular obstruction of the pulmonary artery circulation and clinical outcome. Our study should be followed with a larger study to confirm our findings. Future studies on the prognostic role of RVD and obstruction index assessment at helical CT for PE should also include the determination of brain natriuretic peptide and troponin measurements in individual patients to determine their complementary role in stratifying the heterogeneous group of patients with PE. On the basis of these tests, a patient group suitable for a shorter stay in the hospital or even for full treatment out of the hospital (16) could be selected. Furthermore, more aggressive therapy, including thrombolytic therapy (19), may be warranted in those patients with RVD or a high degree of vascular obstruction, although additional evidence from properly designed clinical trials is still awaited.

We conclude that both the RV/LV ratio and the pulmonary vascular obstruction index as assessed at helical CT are potentially useful tools to predict mortality in patients with initially hemodynamically stable PE at clinical presentation.

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