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Enlarged Right Ventricle Without Shock in Acute Pulmonary Embolism: Prognosis

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Abstract

Background—An unsettled issue is use of thrombolytic agents in patients with acute pulmonary embolism who are hemodynamically stable, but have right ventricular enlargement.

Purpose—To assess in-hospital mortality of hemodynamically stable patients with pulmonary embolism and right ventricular enlargement.

Methods—Patients were enrolled in the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II). Exclusions included shock, critically ill, ventilatory support, myocardial infarction within 1 month, or ventricular tachycardia or ventricular fibrillation within 24 hours. We evaluated the ratio of the right ventricular minor axis to the left ventricular minor axis measured on transverse images during computed tomographic angiography.

Results—Among 76 patients with right ventricular enlargement treated with anticoagulants and/or inferior vena cava filters, in-hospital deaths from pulmonary embolism were 0 of 76 (0%) and all-cause mortality was 2 of 76 (2.6%). No septal motion abnormality was observed in 49 (64%), septal flattening in 25 (33%) and septal deviation in 2 (3%). None required ventilatory support, vasopressor therapy, rescue thrombolytic therapy, or catheter embolectomy. There were no in-hospital deaths due to pulmonary embolism. There was no difference in all-cause mortality comparing patients with and without right ventricular enlargement (relative risk = 1.04)

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Conclusion—In-hospital prognosis is good in patients with pulmonary embolism and right ventricular enlargement if not in shock, acutely ill, on ventilatory support, recent myocardial infarction or life threatening arrhythmia. Right ventricular enlargement alone in patients with pulmonary embolism, therefore, does not appear to indicate a poor prognosis or an indication for thrombolytic therapy.

Keywords

Pulmonary embolism; venous thromboembolism; right ventricular dysfunction

An unsettled issue is the prognosis and the use of thrombolytic agents in hemodynamically stable patients with acute pulmonary embolism who have evidence of right ventricular dysfunction¹. Right ventricular dysfunction in a patient with pulmonary embolism and normal systolic blood pressure has been classified as “impending hemodynamic instability”². At one university, thrombolytic therapy is given to all patients with pulmonary embolism who have right ventricular dysfunction unless contraindicated³. Right ventricular dysfunction has been used as an indication for surgical embolectomy in normotensive patients with pulmonary embolism⁴. Right ventricular dysfunction was defined as a right ventricular-to-left ventricular end-diastolic diameter ratio greater than 1 in the apical 4-chamber view, right ventricular end-diastolic diameter greater than 30 mm, or paradoxical septal systolic motion⁴. The need for a randomized trial in such patients, however, is recognized^{1,3,5}. In view of diverse opinion about the prognosis or whether thrombolytic therapy should be administered to stable patients with acute pulmonary embolism who have right ventricular enlargement⁵, we evaluated such patients who were enrolled in the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II) investigation. We tested the hypothesis that in-hospital mortality and the rate of life-threatening adverse events among patients with pulmonary embolism treated with anticoagulants and/or inferior vena cava filters may be higher in those with right ventricular enlargement than in those without right ventricular enlargement.

METHODS

Data are from the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II), which was a multicenter national collaborative investigation of the accuracy of multidetector computed tomographic (CT) angiography alone with venous phase imaging of the veins of the pelvis and thigh for the diagnosis of acute pulmonary embolism⁶. The data allow a retrospective cohort comparison of outcome between those with and those without right ventricular enlargement. In PIOPED II, 181 patients had a reference test diagnosis of pulmonary embolism and a CT angiogram that was of adequate quality for interpretation⁶. Among these patients, CT angiograms were available for analysis of right ventricular enlargement in 160 patients. All patients in PIOPED II were $\geq$ age 18 years of age. Exclusions included shock, critical illness, ventilatory support, myocardial infarction within 1 month, or ventricular tachycardia or ventricular fibrillation within 24 hours.

We evaluated the ratio of the right ventricular minor axis to the left ventricular minor axis measured on transverse images of contrast enhanced multidetector CT angiograms obtained for the evaluation of suspected acute pulmonary embolism⁷⁻¹¹ (Figures 1:2). We did not perform multiplanar reformatted reconstructions¹²⁻¹³. The minor axes of the right ventricle and left ventricle were measured at their widest points between the inner surface of the free wall and the surface of the interventricular septum¹⁴. The maximum dimensions of the right ventricle and left ventricle may be found at slightly different levels^{11, 14}. Typically, the maximum right ventricular dimension was measured at the level of the tricuspid valve and the maximum left ventricular dimension was measured at the level of the mitral valve⁸. Papillary muscles were excluded from the measurement of left ventricular dimension¹⁴. Assessment of

the right ventricular-to-left ventricular dimension ratio was made independently by two readers (FM and AB). A right ventricular-to-left ventricular dimension ratio >1 was defined as right ventricular enlargement^{10,15-19}. The combination of septal dyskinesia with right ventricular enlargement was also evaluated. In patients with right ventricular enlargement, readers evaluated images for the presence of septal displacement, shown as either flattening^{14,20} or leftward displacement of the interventricular septum^{8,10,14,20}. These categories of septal displacement served as a surrogate for septal dyskinesia as seen of echocardiography²¹ and were evaluated separately.

Because of the possibility that comorbid conditions may have caused right ventricular enlargement, we evaluated patients with no prior cardiopulmonary disease as well as all patients. No prior cardiopulmonary disease was defined as no current asthma, pneumonia, history of chronic bronchitis, emphysema, chronic obstructive pulmonary disease, current or past history of right or left sided heart failure, lung cancer, or prior pulmonary embolism.

Statistical methods

Confidence intervals were calculated using the binomial distribution. Mortality rates were compared by chi square. The single unweighted kappa statistic was used to test for agreement between readers²². Relative risk and its 95% confidence interval was calculated with the program at www.sign.ac.uk/methodology/risk.xls.

RESULTS

Among 160 patients with pulmonary embolism by the reference standard in PIOPED II who had a CT angiogram adequate for interpretation of whether pulmonary embolism was present, and whose images were available for review for this study, 157 (98%) showed the left and right ventricles with adequate clarity for evaluation of the diastolic dimensions (Figure 3). Two of these received thrombolytic therapy. One of these patients was normotensive, but was treated with thrombolytic therapy because of the perceived high risk of right ventricular enlargement and hypokinesia of the right ventricle. The other patient was treated with thrombolytic therapy because of hypoxia in spite of the administration of oxygen. Treatment in the remaining 155 patients was with anticoagulants only in 138, anticoagulants plus inferior vena cava filter in 15 and inferior vena cava filter alone in 2.

Age and sex of the patients treated with anticoagulants and /or an inferior vena cava filter according to whether ventricular enlargement was present and whether they had prior cardiopulmonary disease is shown in Table 1. The age and sex distribution of patients with and without right ventricular enlargement were similar. The Wells' objective clinical assessment score in these patients is shown in Table 2²³. Patients with an enlarged right ventricle more often had a high probability clinical assessment than who did not have right ventricular enlargement. ($P \leq 0.05$).

The following results are those of reader 1. Right ventricular enlargement was observed in 78 of 157 (50%) patients with pulmonary embolism. Among those treated with anticoagulants and/or inferior vena cava filters, right ventricular enlargement was observed in 76. In these patients, in-hospital death from pulmonary embolism was 0 of 76 (0%)[95% confidence interval (CI) = 0 to 3.9%] and all-cause mortality was 2 of 76 (2.6%)(95% CI = 0.3 to 9.2%) (Figure 3, Table 3). One died of glioblastoma multiforme and the other died of carcinoma of the lung. No septal motion abnormality was observed in 49 (64%), septal flattening in 25 (33%) and septal deviation toward the left ventricle in 2 (3%). Among the 76 patients who had an enlarged right ventricle who were treated with anticoagulants and/or an inferior vena cava filter, the right ventricular-to-left ventricular dimension ratio was ≥ 1.1 in 56 (74%), ≥ 1.2 in 36 (47%), ≥ 1.3 in 24 (32%), ≥ 1.4 in 16 (21%), and ≥ 1.5 in 11 (14%)(Table 4). The association

with interventricular septal motion abnormalities is shown in Table 4. None of the patients had an in-hospital life threatening adverse event that required ventilatory support, vasopressor therapy, rescue thrombolytic therapy, or catheter embolectomy. Even if the patient who was treated with thrombolytic therapy because of right ventricular enlargement and hypokinesis would have died if he had been treated with anticoagulants, the death rate from pulmonary embolism in those with right ventricular enlargement would have been 1 of 80 (1.3%) and all cause mortality would have been 3 of 80 (3.8%).

Among patients with pulmonary embolism and no right ventricular enlargement who were treated with anticoagulants and/or an inferior vena cava filter, in-hospital death from pulmonary embolism occurred in 1 of 79 (1.3%) (95% CI = 0.03 to 6.9%) and all cause mortality was 2 of 79 (2.5%) (95% CI = 0.3 to 8.9%). The patient who died, but not of pulmonary embolism, died of respiratory failure from pneumocystis pneumonia associated with human immunodeficiency virus. There was no difference in the rate of death from pulmonary embolism or all cause mortality comparing patients with right ventricular enlargement and those without right ventricular enlargement. The relative risk of all cause mortality comparing those with right ventricular enlargement with those without right ventricular enlargement was 1.04 (95% CI = 0.15 to 7.19).

Regarding patients with no prior cardiopulmonary disease and pulmonary embolism, 54 had right ventricular enlargement and were treated with anticoagulants or an inferior vena cava filter. There were no in-hospital deaths among these 54 patients. Right ventricular enlargement was accompanied by septal flattening in 19 (35%) and by septal deviation to the left in 1 (2%) (Table 5). The right ventricular-to-left ventricular dimension ratio was ≥ 1.2 in 27 of these patients and ≥ 1.3 in 17 patients with no prior cardiopulmonary disease (Table 5).

The kappa statistic for agreement between readers of whether there was right ventricular enlargement was 0.745 indicating good agreement. The kappa statistic for agreement on flattening or leftward displacement of the interventricular septum was 0.165 indicating only slight agreement. Reader 2 measured a right ventricular-to-left ventricular dimension ratio >1 in 66 of 157 (42%) patients. Among those treated with anticoagulants and/or an inferior vena cava filter, right ventricular enlargement was observed in 64. In-hospital death from pulmonary embolism was 0 of 64 (0%) (95% CI = 0 to 7.1%) and all cause mortality was 2 of 64 (3.1%) (95% CI = 0.6 to 11.8%). Among patients treated with anticoagulants and/or an inferior vena cava filter in whom both readers agreed that there was right ventricular enlargement, death from pulmonary embolism was 0 of 60 (0%) (95% CI = 0 to 7.5%) and all cause mortality was 2 of 60 (3.3%) (95% CI = 0.6 to 12.5%).

DISCUSSION

The data do not support the hypothesis that an enlarged right ventricle in stable patients with acute pulmonary embolism may lead to a higher in-hospital mortality or higher rate of life-threatening adverse events. There was no difference in the rate of death from pulmonary embolism or all cause mortality comparing patients with right ventricular enlargement and those without right ventricular enlargement. In the patients with no prior cardiopulmonary disease, patients with possible prior right ventricular enlargement were excluded. None of the patients with right ventricular enlargement had an in-hospital adverse event that required ventilatory support, vasopressor therapy, rescue thrombolytic therapy, or catheter embolectomy.

Selection of a right ventricular-to-left ventricular dimension ratio >1 as indicative of right ventricular enlargement was conservative and has been used by several ^{10,15-19}. Others defined right ventricular enlargement or dysfunction on the basis of a right ventricular-to-left

ventricular dimension ratio >0.9 , although measurements were made from a reconstructed CT four chamber view^{13,24}. A right ventricular-to-left ventricular dimension ratio >0.6 has also been used to define right ventricular overload⁵. Irrespective of the right ventricular-to-left ventricular dimension ratio which ranged from >1.0 to 2.9 in our study, there were no deaths from pulmonary embolism.

The agreement between reader 1 and 2 of interpretation of right ventricular enlargement was good ($\kappa = 0.745$). Others, using a similar method with 3 readers, showed an intraclass correlation of 0.51 ⁹. The κ statistic that we showed for agreement of whether there was flattening or bowing of the interventricular septum showed only slight agreement ($\kappa = 0.165$). Others showed fair agreement on the presence of septal bowing ($\kappa = 0.54$)⁹.

Without electrocardiographic gating, transverse images of the heart may not be truly diastolic images. However, the positive predictive value of measurements on CT transverse sections of an RV/LV dimension ratio >1 or septal deviation was 7 of 7 (100%), based on echocardiography as a reference standard in 23 patients and pulmonary angiography in 2 patients¹⁰. Sensitivity was 7 of 9 (78%) and specificity was 16 of 16 (100%)¹⁰. In another investigation, similar comparisons were made in 14 patients with massive pulmonary embolism¹⁸. The positive predictive value for right ventricular dilatation or septal deviation was 11 of 11 (100%). Right ventricular dilation or septal deviation was shown by CT in 11 of 12 (92%)¹⁸. Specificity was 2 of 2 (100%).

Definitions of right ventricular dysfunction vary (Table 6,7). Goldhaber and associates²⁵ and Wolfe and associates²⁶, based on echocardiograms, defined right ventricular dysfunction as right ventricular hypokinesis. Others used right ventricular enlargement as one of the criteria^{4,5,11,13,15-17,27-29}. Some used only right ventricular enlargement to define right ventricular dysfunction^{5,11,27,28}. Others used right ventricular enlargement or dyskinesia of the interventricular septum^{4,15}. Konstantinides and associates²⁸ required absence of inspiratory collapse of the inferior vena cava in addition. Some evaluated prognosis based on right ventricular enlargement, without defining it as dysfunction¹³. Others evaluated prognosis based on right ventricular enlargement, and other findings as an indicator of right ventricular afterload stress and/or pulmonary hypertension¹⁵⁻¹⁷. Some evaluated prognosis based on right ventricular enlargement and interventricular septal dyskinesia as indicators of acute cor pulmonale²⁹.

Among patients with pulmonary embolism who died in-hospital, the right ventricular dimension on average was larger and the left ventricular dimension smaller than among those who survived^{7,30}. The reported mortality rate from pulmonary embolism among patients with an enlarged right ventricle ranged from 7.8% to 16.7%^{11,16} and depended on the severity of right ventricular enlargement¹¹ (Table 6). Ghaye and associates, among patients in an intensive care unit who had severe pulmonary embolism-related conditions, showed a 5% all cause in-hospital mortality among patients with a right ventricular-to-left ventricular dimension ratio $=1.0$, 10% mortality with right ventricular-to-left ventricular dimension ratio $=1.3$, 20% mortality with right ventricular-to-left ventricular dimension ratio $=1.7$ and 30% mortality with right ventricular-to-left ventricular dimension ratio $=1.9$ ⁷. All-cause mortality in patients with pulmonary embolism and right ventricular enlargement has been reported in 15.6% to 23.5%^{13,16,29}. However, some showed that right ventricular enlargement was not a predictor of mortality⁹. In-hospital adverse events (escalation of therapy including cardiopulmonary resuscitation, mechanical respiratory support, catecholamine treatment for hypotension, rescue thrombolysis or surgical embolectomy)¹² or admission to an intensive care unit⁸ were observed more frequently in patients with right ventricular enlargement or dysfunction than those without.

Whether patients with right ventricular enlargement and /or dysfunction who are not in shock and are treated only with anticoagulants have a higher death rate from pulmonary embolism, all-cause mortality, or higher rate of in-hospital adverse events than those treated with thrombolytic agents is controversial. Prior literature showed mixed results (Table 6). Mortality from pulmonary embolism in such patients who were treated with anticoagulants ranged from 0 to 11.1%^{5,15,25} and all cause mortality in the 2 studies in which it was reported, was the same as the mortality from pulmonary embolism^{5,25}. No statistically different mortality rate was shown in those treated with thrombolytic agents^{5,25} although Goldhaber and associates showed a favorable trend with thrombolytic agents²⁵. All-cause mortality in patients with an enlarged right ventricle and/or right ventricular dysfunction who were not in shock in general ranged from 0 to 11.1%^{5,17,25,29} (Table 7). One group, however, found a 43% all cause mortality in those treated with anticoagulants²⁷. Only one group showed a significantly lower all cause mortality among patients treated with thrombolytic agents¹⁷. In-hospital adverse events were less frequent in those treated with thrombolytic agents (24.6% versus 10.2%)²⁸ (Table 7). A lower rate of adverse events (4.6%) among surviving patients with right ventricular enlargement who were treated with anticoagulants was reported by Grifoni and associates than by Konstantinides^{15,28}.

Strengths of this investigation are the incorporation of all of the Standards for Reporting Diagnostic Accuracy (STARD) criteria^{31,32}. Recruited patients were inpatients and outpatients of both sexes with a wide range of ages and associated illnesses. Therefore, the STARD criteria for a representative population were met³¹.

A weakness is that we did not follow patients with pulmonary embolism. Therefore, rates of recurrent pulmonary embolism and mortality after hospital discharge are not known. Recurrent pulmonary embolism was observed more frequently in hemodynamically stable patients with right ventricular dysfunction treated with anticoagulants than in those treated with thrombolytic agents²⁵. Another weakness was the inability to assess right ventricular hypokinesis from the CT images. Right ventricular hypokinesis was used in the assessment of right ventricular dysfunction in the patients evaluated by Goldhaber and associates, and Wolf and associates^{25,26}.

In conclusion, in-hospital prognosis is good in patients with pulmonary embolism and right ventricular enlargement, providing they are not in shock, acutely ill, receiving ventilatory support or had a recent myocardial infarction or life threatening arrhythmia. Right ventricular enlargement alone in patients with pulmonary embolism, therefore, does not appear to adversely affect prognosis or be an indication for thrombolytic therapy.

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Figure 1. Patient with right ventricular-to-left ventricular dimension ratio <1 shown on transverse image of contrast enhanced multidetector CT angiogram. Minor axis of right ventricle is shown by dotted line at top and minor axis of left ventricle is shown at bottom.

Figure 2. Patient with right ventricular/left ventricular dimension ratio >1 shown on transverse image of contrast enhanced multidetector CT angiogram. Minor axis of right ventricle is shown by dotted line at top and minor axis of left ventricle is shown at bottom. Flattening of the interventricular septum is shown.

Figure 3.

Enrollment and outcome. IVC = inferior vena cava; Data are based on measurements of reader 1.

Table 1
Age and Sex of Patients Treated With Anticoagulants and/or Inferior Vena Cava Filter

	Right Ventricular/Left Ventricular Dimension Ratio > 1		Right Ventricular/Left Ventricular Dimension Ratio ≤ 1	
	No Prior CPD	Prior CPD	No Prior CPD	Prior CPD
Age	57 ± 16	60 ± 16	58 ± 16	59 ± 20
Males	22/55 (40)	9/23 (39)	31/78 (40)	13/23 (57)
			21/56 (38)	55 ± 17
				34/79 (43)

All differences not significant
CPD= cardiopulmonary disease
Data are based on measurements of reader 1

Table 3
Patients Treated With Anticoagulants and/or Inferior Vena Cava Filter

Outcome	Right Ventricular/Left Ventricular Dimension Ratio > 1		Right Ventricular/Left Ventricular Dimension Ratio ≤ 1	
	No Prior CPD n/N (%)	Prior CPD n/N (%)	No Prior CPD n/N (%)	Prior CPD n/N (%)
PE Deaths	0/54 (0)	0/22 (0)	1/56 (1.8)	0/23 (0)
All Cause Deaths	0/54 (0)	2/22 (9)*	1/56 (1.8)	1/23 (4.3)
Life threatening adverse events	0/54 (0)	0/22 (0)	0/56 (0)	0/23 (0)
All differences not significant				

* 1 patient who died showed flattening of the interventricular septum. The other showed a normal appearing septum.

CPD= cardiopulmonary disease

PE= pulmonary embolism

Data are based on measurements of reader 1

Table 4

Right Ventricular/Left Ventricular Dimension Ratios and Interventricular Septal Motion in All Patients Treated with Anticoagulants and/or Inferior Vena Cava Filter

Right ventricular/Left ventricular Dimension ratio	Normal Septum	Septal Flattening	Septal Deviation	Total
≥1.0	49	25	2	76
> 1.1	31	23	2	56
≥ 1.2	18	16	2	36
> 1.3	10	12	2	24
≥ 1.4	7	8	1	16
> 1.5	5	5	1	11
≥ 1.6	4	3	1	8
> 1.7	1	2	1	4
≥ 1.8	1	2	1	4
> 1.9	1	2	1	4
2.2 - 2.9	1	2	0	3

Data are based on measurements of reader 1

Table 5

Right Ventricular/Left Ventricular Dimension Ratios and Interventricular Septal Motion in Patients With No Prior Cardiopulmonary Disease Treated with Anticoagulants and/or Inferior Vena Cava Filter

Right ventricular/Left ventricular Dimension ratio	Normal Septum	Septal Flattening	Septal Deviation	Total
>1.0	34	19	1	54
> 1.1	23	18	1	42
≥ 1.2	13	13	1	27
> 1.3	7	9	1	17
≥ 1.4	5	4	0	9
> 1.5	4	2	0	6
≥ 1.6	3	1	0	4
1.7 - 2.2	1	0	0	1

Data are based on measurements of reader 1

Table 6
Mortality and Adverse Events: Pulmonary Embolism and Right Ventricular Enlargement

First Author (Reference)	RV Enlargement and Dysfunction Definition	Patient group	Duration	All- cause mortality RV large n/N (%)	All-cause mortality RV Not Large n/N (%)	PE mortality; RV large n/N (%)	PE mortality RV Not Large n/N (%)
Schoepf (13)	RV/LV >0.9 Reformatted CT 4-chamber view	All PE	30 day	43/276 (15.6)	12/155 (7.7) ¹		
Van der Meer (11)	RV/LV > 1 and ≤ 1.5	All PE	90 day [*]			4/51 (7.8)	0/51 (0) ²
Van der Meer (11)	RV/LV>1.5	All PE	90 day [*]			3/18 (16.7)	
Kasper (16)	RV/LV>1 or RVEDD >30 mm or ≥2 of following: TR jet >2.8 m/s or >2.5m/s if no inspir collapse of IVC, RPA>12 mm/m2, RV Wall > 5mm, No inspir collapse of IVC	All PE	In-hosp	16/87 (18.4)	13/230 (5.7) ³	11/87 (12.6)	2/230 (0.9) ³
Veillard-Baron (29)	RV/LV area >0.6 plus septal dyskinesia	No prior CPD	In-hosp	22/98 (22.5)			

* All PE deaths were within 17 days.

¹ p≤ 0.025
² p≤ 0.05
³ p≤ 0.001

RV = right ventricle, RVEDD = right ventricular end-diastolic dimension, LV = left ventricle, PE = pulmonary embolism, CT = computed tomogram, TR = tricuspid regurgitation, RPA = right pulmonary artery, IVC = inferior vena cava, CPD = cardiopulmonary disease, hosp = hospital, inspire= inspiratory

Table 7
Mortality and Adverse Events: Pulmonary Embolism and Right Ventricular Enlargement Not in Shock

First Author (Reference)	RV Enlargement and Dysfunction Definition	Follow-up Duration	All-cause mortality Anticoag n/N (%)	All-cause mortality n/N (%)	PE Mortality Anticoag n/N (%)	PE Mortality n/N (%)	In-Hosp Adverse Events ^{**} Anticoag n/N (%)	In-Hosp Adverse Events ^{**} n/N (%)	In-Hosp Adverse Events ^{**} n/N (%)
Konstantinides (28)	RV enlargement + no inspir collapse of IVC *	30 days	3/138 (2.2)	4/118(3.4)			34/138 (24.6)		12/118 (10.2)
Sukhija (27)	RV/LV area > 0.9 or RV ≥ 4.5cm	In-hosp	17/40 (43)	2/6 (33)					
Hanel (5)	RV/LV dimension>0.6	In-hosp	0/64 (0)	4/64 (6.3)	0/64 (0)	1/64 (1.6)			
Goldhaber (25)	RV ED area enlarged RV hypokinesis	14 days	2/18 (11.1)	0/18 (0)	2/18 (11.1)	0/18 (0)			
Konstantinides (17)	RV/LV > 1, paradoxical septal wall motion, no inspir collapse of IVC, or TR jet >2.8 m/s or >2.5 m/s if no inspir collapse of IVC	30 days	61/550 (11.1)	8/169 (4.7) ₂					
Veillard-Baron (29)	RV/LV area >0.6 +septal dyskinesis No prior card-pulm dis	In-hosp	1/32(3.1%)						
Grifoni (15)	RV/LV dimension>1 Or RVEDD >30 mm or paradoxical septal systolic motion or pulmonary acceleration time < 90 ms or RV/atrial gradient > 30 mm Hg	In-hosp			3/65 (4.6)		3/65 (4.6)		

* Study included 31% of patients with right ventricular dysfunction and remainder with pulmonary hypertension from pulmonary embolism.

** Shock, ventilatory support, cardiopulmonary resuscitation lytics, catheter fragmentation

₁ P=0.004

₂ P=0.016

Anticoag = anticoagulants, lytics = thrombolytic agents, RV = right ventricle, LV = left ventricle, PE = pulmonary embolism, CT = computed tomogram, TR = tricuspid regurgitation, RPA = right pulmonary artery, IVC = inferior vena cava, hosp = hospital, ED = end-diastolic, inspire = inspiratory, card-pulm = cardiopulmonary, dis = disease, RVEDD = right ventricular end-diastolic dimension