Novel management strategy for patients with suspected pulmonary embolism

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Aims A simple management strategy is required for patients with acute pulmonary embolism which allows a rapid and reliable diagnosis in order to start timely and appropriate treatment.

Methods and results Two hundred and four consecutive patients with suspected pulmonary embolism were managed according to a standardized protocol based on the clinical pretest probability and the initial haemodynamic presentation (shock index = heart rate divided by systolic blood pressure). Patients with a high pretest probability and a positive shock index (≥1; n=110) underwent urgent transthoracic echocardiography. Based on the presence or absence of right ventricular dysfunction, reperfusion treatment was initiated immediately. Patients with a negative shock index (<1; n=183) underwent diagnostic evaluation including pretest probability, D-dimer, and spiral computed tomography (CT) as first-line tests. Echocardiography was performed only when a central pulmonary embolism was found in the spiral CT (n=33). According to our strategy, 98 patients met the diagnostic criteria of pulmonary embolism: 75 patients (all shock index <1) were treated with heparin alone, 16 (seven had a shock index ≥1) with thrombolysis, four (all shock index ≥1) with catheter fragmentation, and three (all shock index ≥1) with surgical embolectomy. The all-cause mortality rate at 30 days was 5%, and at 6 months 11%. Right ventricular dysfunction on baseline echocardiography was not associated with a higher mortality rate at 6 months (logrank 2.4, P=0.12).

Conclusions The novel management strategy for patients with suspected pulmonary embolism resulted in a rapid diagnosis and treatment with a low 30-day mortality. In patients with pulmonary embolism and a positive shock index, time-consuming imaging tests can be avoided to reduce the risk of sudden death and not to delay reperfusion therapy.

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KEYWORDS Pulmonary embolism; Right ventricular function; Diagnosis; Therapy

Introduction

Overall mortality in patients with acute pulmonary embolism remains high despite modern diagnostic and reperfusion strategies. In the largest international registry of 2454 patients, 3-month mortality was 17% whereby 45% of deaths were attributed to pulmonary embolism.1 Right ventricular dysfunction is present in about 30% of patients with acute pulmonary embolism and normal blood pressure.2 Therefore, transthoracic echocardiography has emerged as important diagnostic tool for assessing the degree of right
ventricular dilation and the severity of right ventricular systolic dysfunction. In the international registry, echocardiographic right ventricular dysfunction was identified as the single most important prognostic factor for in-hospital death, and 75% of deaths in patients with pulmonary embolism and right ventricular dysfunction occurred in the first days after diagnosis. In these patients, overall mortality at 3 months was twice as high as in patients with preserved right ventricular function in the baseline echocardiogram.

Nevertheless, none of the available reperfusion strategies has ever shown a mortality benefit for patients with acute pulmonary embolism. So far, less than 500 patients have been enrolled in small randomized trials comparing thrombolysis with heparin alone. A mortality benefit for thrombolysis has not been shown.3–11 Thrombolysis is indicated in patients with pulmonary embolism-related shock. Data to support this indication are obtained from a study of eight patients, when four patients who received thrombolysis survived, and four patients who were treated with heparin alone died within 48 h after pulmonary embolism diagnosis.3 However, there are no controlled data for catheter fragmentation or surgical embolectomy in comparison to thrombolysis or heparin alone.

Thus, there is a need for a simple management strategy for patients with acute pulmonary embolism, which provides a rapid and reliable diagnosis and which identifies high risk patients who need treatment without delay. As a consequence, a standardized management strategy for patients with suspected pulmonary embolism was developed by a disease management group. This strategy was based on the initial haemodynamic presentation of the patients and allowed the correct diagnostic and therapeutic algorithm to be defined. The proposed management strategy takes recent data on Doppler echocardiography1,2,12–14 and spiral computed tomography15–19 into account, which play a major role in pulmonary embolism diagnosis and risk stratification. The aim of the present study was to test the outcome of consecutive patients with suspected pulmonary embolism who were diagnosed and treated according to our management strategy.

Methods

Disease management project ‘acute pulmonary embolism’

Clinical departments involved in the care of patients with acute pulmonary embolism at the University Hospital in Bern participated in the disease management project (see Appendix). Flow charts were designed for clinical pretest probability (Fig. 1), diagnostic (Fig. 2) and treatment strategy (Fig. 3) and accepted in their final versions by the disease management group. The management strategy was approved by the local ethics committee.

Patients

A total of 204 consecutive patients with suspected pulmonary embolism admitted to the emergency department between April 2000 and October 2001 (18 months) were enrolled in the present study. Patients were stratified according to the clinical pretest probability which was assessed during clinical examination using a modification of the flow chart proposed by Wells and coworkers.20 Systolic blood pressure and heart rate were measured, and the shock index (heart rate divided by systolic blood pressure) was calculated in all patients for further stratification. A positive shock index was defined as ≥1 (=haemodynamically
Diagnostic strategy in patients with suspected pulmonary embolism and a shock index <1

In 183 patients with suspected pulmonary embolism and a negative shock index, the diagnostic strategy included D-dimer, pretest-probability, and spiral computed tomography as first-line tests (Fig. 2). In six of 145 patients with D-dimer >500 µg l⁻¹, spiral computed tomography was not feasible due to contraindications for radiographic contrast agents: four patients were negative for pulmonary embolism with a normal/near-normal ventilation perfusion scan, two were positive for pulmonary embolism with a high probability ventilation perfusion scan. During 3-months of follow-up, there was no clinical venous thromboembolism among the 99 patients with initially negative test results. They were excluded from the study.

In 33 patients with central pulmonary embolism on spiral computed tomography and with a shock index <1, transthoracic echocardiography was performed to assess right ventricular dysfunction (n=26). Echocardiography was optional in patients with non-central pulmonary embolism and a negative shock index.

Diagnostic strategy in patients with suspected pulmonary embolism and a shock index ≥1

All 21 patients with a shock index ≥1 had a high pretest probability for pulmonary embolism (Fig. 1). Emergent echocardiography was performed by a resident cardiologist within 30 min after admission in all of them (Fig. 3). In the presence of echocardiographic right ventricular dysfunction, reperfusion therapy was started immediately. In seven of the 21 patients with shock index ≥1 and with preserved right ventricular function on the echocardiogram, a diagnosis of pulmonary embolism was discarded as the cause of haemodynamic instability. In these patients, a major pulmonary embolism was excluded by spiral computed tomography (two had aortic dissection, two decompensated left heart failure, two systemic bacterial infection, one decompensated chronic pulmonary obstructive disease).

Transthoracic echocardiography

According to the management strategy (Fig. 3), echocardiography was performed in 54 patients (21 with shock index ≥1, and 33 with shock index <1) and central pulmonary embolism on spiral computed tomography. In addition, echocardiography
was carried out in 25 patients with non-central pulmonary embolism in the spiral computed tomogram.

Echocardiography was performed using an Acuson Sequoia™ C256 system (Mountain view, California, U.S.A.) with a 3.5 MHz probe and three-lead electrocardiographic monitoring. Right ventricular end-diastolic diameter was measured either from the apical or subcostal four-chamber view. The maximal distance between the endocardium of the right ventricular free wall and the interventricular septum, perpendicular to the long axis of the ventricle, was measured at the beginning of the QRS complex. Systolic right ventricular function was assessed by examining right ventricular free wall motion using a four-point scoring system (normal systolic function, mild, moderate, and severe systolic dysfunction). Tricuspid pressure gradient as a surrogate of right ventricular pressure overload: Right ventricular diameter >40 mm and/or the presence of moderate to severe right ventricular systolic dysfunction, and/or a tricuspid systolic systolic gradient >50 mmHg.

D-dimer, ventilation perfusion scan, compression sonography of the leg veins, pulmonary angiography, and spiral CT

Quantitative VIDAS D-dimer levels were measured in all patients on admission, and a value <500 µg l⁻¹ was used as the cut-off value to exclude pulmonary embolism in patients with a low and moderate pretest probability. Ventilation perfusion scans were performed in six patients and interpreted as described elsewhere. The criterion for the diagnosis of deep venous thrombosis by compression ultrasonography of the leg veins (n=55) was noncompressibility of the vein. Pulmonary angiography was performed in non-conclusive situations (n=2) or in haemodynamically unstable patients scheduled for catheter fragmentation (n=4).

Spiral computed tomography was performed in all patients with a negative shock index and increased D-dimer values. Pulmonary embolism was diagnosed when at least one pulmonary artery showed an intravascular filling defect. Central pulmonary embolism was diagnosed when a filling defect in one of the major pulmonary arteries (i.e. the main stem or the left and right main or the lobar pulmonary artery) was seen. Accordingly, non-central pulmonary embolism was diagnosed in

Fig. 3 Management strategy for 204 patients with suspected pulmonary embolism (PE). RV=right ventricular.
the presence of filling defects located distally to the lobar pulmonary arteries.

The interobserver agreement for spiral computed tomography results was 97% (kappa 0.95, \(P<0.0001\)). Pulmonary occlusion rate on spiral computed tomography was assessed in all patients with pulmonary embolism using the semi-quantitative Miller index based on the modification of Remi-Jardin and coworkers.25

Reperfusion treatment

All patients with right ventricular dysfunction and without a high bleeding risk were treated with thrombolysis. In patients >70 years with a shock index ≥1 and contraindications for thrombolysis according to the guidelines from the European Society of Cardiology,36 catheter fragmentation or surgical embolectomy was performed. In patients >70 years with a shock index <1 and contraindications for thrombolysis, heparin alone was given (Fig. 3).

Thrombolysis was performed with tissue plasminogen activator (r-tPA) with a bolus of 15 mg followed by a continuous infusion of 85 mg over 2 h. In patients with a high pretest probability and a shock index ≥1, catheter fragmentation with a rotational pigtail catheter26 was performed immediately after right ventricular dysfunction was diagnosed by echocardiography, i.e. 41±16 min after admission. Surgical embolectomy27 was performed as described previously.

Follow-up procedure of patients with pulmonary embolism

Written informed consent was obtained from all patients with pulmonary embolism for the follow-up examination. The 6-month follow-up was completed by interview, or by phone from their general practitioner, in all 98 patients with pulmonary embolism. Mean follow-up time was 15±6 (range 6–23) months. In case of death, rehospitalization or outpatient therapy during follow-up, medical reports were obtained.

Statistical analysis

Nominal data comparison between the treatment groups was performed using a Chi-square test. Continuous data are given as mean±SD, and differences between treatment groups were examined by the Student \(t\)-test. Primary endpoint measure was all-cause mortality calculated at 30 days, 3 and 6 months using the Kaplan–Meier method. Data were considered significant at \(P<0.05\).

Results

Diagnosis of pulmonary embolism was confirmed in 48% of all patients with suspected pulmonary embolism, compared to 16–48% in patients with suspected pulmonary embolism from previous trials.28–35 Of the 98 patients with pulmonary embolism, 21 (13%) had a positive shock index on admission. The presence of dyspnoea, syncope, deep venous thrombosis, or right heart thrombi was higher among patients who were treated with reperfusion strategy (i.e. thrombolysis, catheter fragmentation, or surgical embolectomy) than with heparin alone (Table 1). Contraindications for thrombolysis according to the guidelines of the European Society of Cardiology36 included patients >70 years with a negative shock index <1. Thus, patients who were treated with heparin alone were older than those treated with reperfusion. Haemodynamic and respiratory changes were more severe in patients who underwent thrombolysis, catheter fragmentation, or surgical embolectomy compared with those treated with heparin alone (Table 2).

Prevalence of right ventricular dysfunction among patients who underwent echocardiography was 62%. Pulmonary occlusion rate as assessed by spiral computed tomography was higher and right ventricular pressure overload as determined by echocardiography more prevalent in patients treated with reperfusion than those with heparin alone (Table 2).

In 23 patients with right ventricular dysfunction, the time interval between hospital admission and start of reperfusion therapy was 78±34 min compared with 32±12 min in patients with a positive shock index ≥1 (\(n=14\)).

Therapeutic management in patients with a negative shock index <1 (Fig. 3)

Thrombolysis was performed in nine of 26 patients with central pulmonary embolism and right ventricular dysfunction. Eight of nine patients without shock but with right ventricular dysfunction showed clinical and echocardiographic improvement of right ventricular function within 12 h after thrombolysis. The remaining 17 patients with central pulmonary embolism in the spiral computed tomogram and echocardiographic evidence of right ventricular dysfunction received conventional heparin alone. In 51 patients with non-central pulmonary embolism on spiral computed tomography, conventional heparin (\(n=52\)) or low molecular-weight heparin alone (\(n=23\)) was given.
Therapeutic management in patients with a positive shock index ≥1 (Fig. 3)

In 14 of the 21 patients with suspected pulmonary embolism and a shock index ≥1, reperfusion therapy was started on the basis of right ventricular dysfunction in the echocardiogram. In all 14 patients, pulmonary embolism was confirmed after initiation of reperfusion therapy: In seven patients, pulmonary embolism was confirmed by spiral computed tomography after initiation of thrombolysis, in four using pulmonary angiography during catheter fragmentation, and in three during surgical embolectomy. Six of seven patients with shock who were treated with thrombolysis, and all four patients who underwent catheter fragmentation, as well as one of three patients with surgical embolectomy had rapid clinical and echocardiographic improvement of right ventricular function.

Oral anticoagulation and vena caval filter

Overall, 82 of 90 patients (91%) received oral anticoagulation for at least 3 months, 25 of 86 patients (29%) were alive at 6 months and had ongoing oral anticoagulation due to an increased thromboembolic risk. A vena caval filter was implanted into seven patients due to contraindications for oral anticoagulation.

Table 1  Baseline characteristics in 98 patients with pulmonary embolism

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Heparin or LMWH alone</th>
<th>Thrombolysis</th>
<th>Embolectomy or catheter fragmentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>98</td>
<td>75 (77)</td>
<td>16 (16)</td>
<td>7 (7)</td>
</tr>
<tr>
<td>Age, years</td>
<td>61±18</td>
<td>64±18</td>
<td>52±14</td>
<td>56±15</td>
</tr>
<tr>
<td>Men (%)</td>
<td>51 (52)</td>
<td>40 (53)</td>
<td>8 (50)</td>
<td>3 (43)</td>
</tr>
<tr>
<td>Clinical pretest probability</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low†</td>
<td>14 (14)</td>
<td>13 (17)</td>
<td>1 (6)</td>
<td>0</td>
</tr>
<tr>
<td>Moderate</td>
<td>23 (23)</td>
<td>20 (27)</td>
<td>2 (12)</td>
<td>1 (14)</td>
</tr>
<tr>
<td>High</td>
<td>61 (62)</td>
<td>42 (56)</td>
<td>13 (81)</td>
<td>6 (86)</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyspnoea**</td>
<td>63 (64)</td>
<td>41 (55)</td>
<td>15 (94)</td>
<td>7 (100)</td>
</tr>
<tr>
<td>Chest pain</td>
<td>41 (42)</td>
<td>30 (40)</td>
<td>8 (50)</td>
<td>3 (43)</td>
</tr>
<tr>
<td>Syncope*</td>
<td>13 (13)</td>
<td>3 (4)</td>
<td>7 (44)</td>
<td>3 (43)</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignancy</td>
<td>13 (13)</td>
<td>11 (15)</td>
<td>2 (12)</td>
<td>0</td>
</tr>
<tr>
<td>Systemic hypertension</td>
<td>22 (22)</td>
<td>17 (23)</td>
<td>3 (19)</td>
<td>2 (29)</td>
</tr>
<tr>
<td>Deep venous thrombosis**</td>
<td>22 (22)</td>
<td>13 (17)</td>
<td>8 (50)</td>
<td>3 (43)</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>7 (7)</td>
<td>5 (7)</td>
<td>2 (12)</td>
<td>0</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>6 (6)</td>
<td>4 (5)</td>
<td>2 (12)</td>
<td>0</td>
</tr>
<tr>
<td>COPD</td>
<td>6 (6)</td>
<td>5 (7)</td>
<td>1 (6)</td>
<td>0</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2 (2)</td>
<td>2 (3)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Right heart thrombus**</td>
<td>3 (3)</td>
<td>0</td>
<td>1 (6)</td>
<td>2 (29)</td>
</tr>
</tbody>
</table>

*C*<0.0001, †*P*<0.001, **P*<0.05 between the three treatment regimens, respectively.

COPD=chronic obstructive pulmonary disease, LMWH=low molecular-weight heparin.

Table 2  Haemodynamic and echocardiographic findings in 98 patients with pulmonary embolism

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Heparin or LMWH alone</th>
<th>Thrombolysis</th>
<th>Embolectomy or catheter fragmentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>98</td>
<td>75 (77)</td>
<td>16 (16)</td>
<td>7 (7)</td>
</tr>
<tr>
<td>Heart rate, beats min⁻¹</td>
<td>89±19</td>
<td>83±17</td>
<td>107±9*</td>
<td>111±19*</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>116±24</td>
<td>122±19</td>
<td>98±21*</td>
<td>96±28*</td>
</tr>
<tr>
<td>Shock index</td>
<td>0.9±0.6</td>
<td>0.7±0.2</td>
<td>1.4±1.0*</td>
<td>1.3±0.7*</td>
</tr>
<tr>
<td>PO₂, mmHg</td>
<td>67±21</td>
<td>71±21</td>
<td>58±17</td>
<td>53±16**</td>
</tr>
<tr>
<td>PCO₂, mmHg</td>
<td>34±6</td>
<td>36±6</td>
<td>29±6†</td>
<td>30±4**</td>
</tr>
<tr>
<td>Pulmonary occlusion rate (modified Miller index), %</td>
<td>62±34</td>
<td>50±32</td>
<td>95±6*</td>
<td>97±4†</td>
</tr>
<tr>
<td>RV end-diastolic diameter, mm</td>
<td>43±7</td>
<td>41±7</td>
<td>47±5†</td>
<td>48±6**</td>
</tr>
<tr>
<td>Tricuspid systolic gradient, mmHg</td>
<td>49±18</td>
<td>44±18</td>
<td>60±13**</td>
<td>55±15</td>
</tr>
<tr>
<td>Moderate/severe right ventricular systolic dysfunction, n/n (%)</td>
<td>49/79 (62)</td>
<td>26/56 (56)</td>
<td>16/16* (100)</td>
<td>7/7* (100)</td>
</tr>
</tbody>
</table>

*C*<0.0001, †*P*<0.001, **P*<0.05 compared to treatment with heparin alone, respectively.
anticoagulation (n=4) or failure of anticoagulation (n=3).

Adverse events (Table 3)

All-cause mortality at 30 days (n=5) was 5%, and all five deaths occurred in the hospital, and were attributed to acute right ventricular failure from pulmonary embolism. One of these patients was treated with heparin alone, despite having a shock index <1, central clots in the spiral computed tomogram and right ventricular dysfunction. Reperfusion therapy was not considered due to concomitant ischaemic stroke. All-cause mortality at 3 and 6 months was similar in patients who received heparin alone and those treated with reperfusion (Table 3). The presence of right ventricular dysfunction on baseline echocardiography was not associated with a higher mortality at 3 (logrank 0.7, \( P=0.41 \)) and 6 months (logrank 2.4, \( P=0.12 \)).

Non-fatal major bleedings occurred in four patients during the initial hospital stay. One patient in the heparin-group had gastrointestinal bleeding, one patient underwent surgical revision for rupture of the liver and spleen following cardiopulmonary resuscitation, and one patient had diffuse tracheal bleeding following intubation (both from thrombolysis group). Another patient underwent surgical revision for liver rupture following cardiopulmonary resuscitation and surgical embolectomy. A drop in haemoglobin of more than 3 g/l was found in seven of 23 patients who received reperfusion treatment had.

At 6-month follow-up, there were no differences between the heparin and the reperfusion groups with regard to venous thromboembolism and the presence of persistent dyspnoea NYHA class ≥II.

Discussion

A novel management strategy was developed by a disease management group and was prospectively evaluated in consecutive patients with suspected pulmonary embolism. The unique feature of the proposed strategy is the diagnosis and treatment algorithms were standardized according to the initial haemodynamic presentation of the patient in order to avoid delays in initiating appropriate treatment. Diagnostic and therapeutic steps were defined by three flow charts (Figs. 1–3), allowing a general application of the proposed strategy to patients with suspected pulmonary embolism.
This study implies that echocardiography has a direct impact on treatment strategy in patients with pulmonary embolism. Echocardiography is useful in haemodynamically unstable patients to support the diagnosis of pulmonary embolism in order to start reperfusion therapy in those with right ventricular dysfunction without delay. On the other hand, echocardiography helps to identify patients with central pulmonary embolism who may (right ventricular dysfunction) or may not (no right ventricular dysfunction) benefit from thrombolysis in the absence of shock. Patients with a shock index <1 and a small clot burden on spiral computed tomography are at low risk and not likely to benefit from a reperfusion therapy even right ventricular dysfunction may have been present. Treatment strategy in these patients includes heparin alone, and echocardiography was not performed due to its low impact on treatment strategy and cost effectiveness. As a result, none of 51 patients with non-central pulmonary embolism and shock index <1 died within 6 months from pulmonary embolism.

In patients with suspected pulmonary embolism, the shock index, defined as heart rate divided by systolic blood pressure, is a simple parameter which allows an estimate to be made of the available time for the diagnostic approach: In patients with a negative shock index <1, there is sufficient time for imaging tests in order to accurately diagnose pulmonary embolism, whereas in those with a positive shock index ≥1, reperfusion treatment should be started without delay. In the present study it was shown that emergency transthoracic echocardiography is an accurate bedside test for the diagnosis or exclusion of pulmonary embolism. The diagnosis was correctly established in all 21 patients with a shock index ≥1. In the 14 of the 21 patients with a shock index ≥1 and echocardiographic evidence of right ventricular dysfunction, reperfusion treatment was initiated within a short time (32±12 min) after admission to hospital: seven patients received thrombolysis, one underwent catheter fragmentation, and three surgical embolectomy (Fig. 3). In the seven patients with shock but without right ventricular dysfunction, spiral computed tomography was negative for pulmonary embolism. Although unlikely, it is possible that subsegmental pulmonary embolism was present in a few of these patients, but probably did not contribute to the haemodynamic instability.

Incorporated in the diagnostic strategy, spiral computed tomography has been shown recently to be a diagnostic method of value in several trials. Sensitivity of spiral computed tomography to exclude minor pulmonary embolism is low. Thus, further diagnostic work-up was performed in only those 13 patients with a moderate to high pretest probability and a negative spiral computed tomogram (Fig. 2). On the other hand, patients with a low pretest probability, elevated D-dimer levels and a negative spiral computed tomogram were not further evaluated. As a consequence, no recurrent pulmonary embolism was observed during the 3-month follow-up among the 99 patients with initially negative test results. The risk of recurrent pulmonary embolism among patients with a negative spiral computed tomogram is similar to those with a negative pulmonary angiogram.15–18 Moreover, spiral computed tomography is the only non-invasive test which allows direct visualization of the pulmonary emboli to quantify clot burden. Assessment of the pulmonary occlusion rate also makes this technique useful for risk stratification.25 In patients with central pulmonary embolism and right ventricular dysfunction but with a shock index <1, reperfusion treatment might be considered according to the guidelines of the European Society of Cardiology although this indication has not yet been proven in a large controlled trial.36 In this study, all patients with central pulmonary embolism in the spiral computed underwent echocardiography to diagnose or exclude right ventricular dysfunction. The proportion of patients with a shock index <1 having signs of right ventricular pressure overload was identical (31%) to that reported by Grifoni et al.2 in a study of 209 patients with proven pulmonary embolism. In this trial, all patients with a shock index <1 and right ventricular dysfunction younger than 70 years were considered for thrombolysis when no contraindication was present (n=9) according to the guidelines of the European Society of Cardiology.36

Clinical implications

A large multicentre trial for patients with acute pulmonary embolism using an accurate risk stratification is overdue, because a number of hospitals perform reperfusion treatment in patients with pulmonary embolism and right ventricular dysfunction despite lacking evidence for a mortality benefit.37 The objective of this trial was not to compare the different treatment strategies in a randomized fashion but to test the new pretest probability and the diagnostic and therapeutic flow charts. Although reperfusion therapy was rapidly successful in most patients, the question whether this strategy has a benefit in all patients with pulmonary embolism and right ventricular dysfunction can only be answered with a randomized
The study. The goal was to apply recent data of echocardiography and spiral computed tomography in a standardized management strategy, and to investigate early and late mortality in consecutive patients with pulmonary embolism. Hospital mortality in the present trial was low (5%), and there was no increase in bleeding complications although a high proportion of patients were included with massive and submassive pulmonary embolism (14% with a shock index ≥1 and 26% with right ventricular dysfunction and a shock index <1). Indeed, there was no statistical significant difference between the mortality curves for patients with and without right ventricular dysfunction but there was a clear trend (logrank 2.4, *P* = 0.12; Fig. 4) with a higher mortality in those with right ventricular dysfunction. This negative result may be due to the limited number of patients in each group. On the other hand, reperfusion therapy in patients with right ventricular dysfunction has contributed to rapid normalization of right ventricular function possibly resulting in improved outcome in these patients.

The proposed diagnostic and treatment strategy is applicable only in hospitals where spiral computed tomography and emergent echocardiography are available.

**Conclusions**

The novel management strategy for patients with suspected pulmonary embolism resulted in a rapid diagnosis and treatment with a low 30-day mortality. In patients with pulmonary embolism and a positive shock index ≥1, time-consuming imaging tests can be avoided which increase risk of sudden death and delay initiation of reperfusion therapy. Echocardiography is a highly specific test for pulmonary embolism allowing treatment decisions in the presence of right ventricular dilation and systolic dysfunction. As long as data from a large-scale randomized reperfusion study in pulmonary embolism are not available, the proposed management strategy seems to be easily applicable and highly useful for patients with suspected pulmonary embolism.

**Appendix**

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References


