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Clinical Manifestations of Acute Pulmonary Embolism: Henry Ford Hospital Experience, A Five-Year Review

Kenneth V. Leeper, Jr, MD,* John Popovich, Jr, MD,* Deborah Adams, BSN,* and Paul D. Stein, MD†

Clinical findings of 112 patients with angiographically proven pulmonary embolism over a five-year period were analyzed. Recent immobilization, chronic heart disease, deep venous thrombosis, malignancy, and recent surgery were the most frequent predisposing factors. Only 5% had no identifiable risk factors. Presenting syndromes were circulatory collapse (25%), pulmonary infarction (59%), and uncomplicated pulmonary embolism (18%). Dyspnea and pleuritic chest pain were the predominant symptoms. The combination of dyspnea, pleuritic chest pain, and hemoptysis occurred in 23% of the patients. The most frequent signs were tachypnea, tachycardia, and rales. Most of the patients demonstrated arterial oxygen tension (PaO₂) between 50 and 70 mm Hg, and 9% had a PaO₂ greater than 80 mm Hg. The most common chest x-ray findings were infiltration and consolidation, pleural effusion, and atelectasis. The most common electrocardiographic abnormalities were nonspecific ST and T-wave changes in 42% of patients. Lung scans were most frequently interpreted as indeterminate probability. A continuing reassessment of the features of pulmonary embolism may assist in selecting patients for confirmatory diagnostic studies. (Henry Ford Hosp Med J 1988; 36:29-34)

Pulmonary embolism is common yet frequently remains unrecognized. Consequently, this condition is underdiagnosed, especially in patients with a history of cardiac or pulmonary disease. The mortality rate from untreated pulmonary embolism is approximately 30%, while correctly diagnosed and treated patients have a mortality rate of approximately 8% (1). The clinical features and laboratory findings have been characterized as nonspecific in numerous studies (2-4), which is similarly true for the noninvasive diagnostic studies, including ventilation perfusion scans (5,6). Because of the nonspecificity of the noninvasive studies, pulmonary angiography has been used as the "gold standard" in establishing the diagnosis of pulmonary embolism (7,8). Some centers, however, have reported that only 30% of patients with suspected pulmonary embolism have had pulmonary embolism proven by angiography (9). Despite the rather grim impression of the nonspecificity of the clinical diagnosis, some investigators have shown that the clinical features of pulmonary embolism may be extremely useful in establishing a preliminary diagnosis (10,11), thereby eliminating the necessity of performing angiography on patients in whom the diagnosis is unlikely. In this investigation, we assess our own experience in an effort to strengthen our diagnostic capabilities.

Methods

A computer search of the hospital medical records and a review of the records in Interventional Radiology were undertaken to identify patients who underwent pulmonary angiography for suspected pulmonary embolism from 1980 through 1984. During this five-year period, 302 pulmonary angiograms were performed and 112 (37%) patients had pulmonary embolism identified by this procedure (Table 1). Charts of these 112 patients were reviewed for demographic data, symptoms, and signs. Reports of the chest radiographs, ventilation perfusion lung scans, and pulmonary angiograms were also reviewed.

Results

Patient population and predisposing factors

The patients with angiographically proven pulmonary embolism included 59 men and 53 women. Their ages ranged from 20 to 89 years.

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of Patients with Suspected Pulmonary Embolism Undergoing Angiography</th>
<th>Number of Positive Angiograms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980</td>
<td>24</td>
<td>12</td>
</tr>
<tr>
<td>1981</td>
<td>78</td>
<td>18</td>
</tr>
<tr>
<td>1982</td>
<td>60</td>
<td>25</td>
</tr>
<tr>
<td>1983</td>
<td>65</td>
<td>27</td>
</tr>
<tr>
<td>1984</td>
<td>75</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
<td>302</td>
<td>112</td>
</tr>
</tbody>
</table>

Accepted for publication: November 10, 1987.
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The most frequent predisposing factors included recent immobilization and chronic heart disease which occurred in 29% and 28% of the patients with proven acute pulmonary embolism, respectively. Deep venous thrombosis, documented by invasive or noninvasive methods, was observed in 23% of patients with acute pulmonary embolism. Eight patients had positive venograms, all demonstrating clots above the knee. Thirteen of the 18 patients who had noninvasive studies had clots in the femoral vein, and five had equivocal studies suggesting femoral vein involvement. Malignancy and recent surgery were predisposing factors in 24% and 21% of patients, respectively. Less frequent predisposing factors included a history of pulmonary embolism, trauma (usually from long bone fractures), or the use of estrogen-containing drugs. Obesity as the only identifiable risk factor was present in 7% of the patients (Table 2). Many of the patients had multiple risk factors. However, 5% of the patients had no recognized risk factor.

### Presenting syndromes of acute pulmonary embolism

The syndrome of circulatory collapse (shock or syncope) occurred in 25% of the patients. The pulmonary infarction syndrome (hemoptysis with or without pleuritic pain or pleuritic pain without hemoptysis) occurred in 59% of patients. Uncomplicated pulmonary embolism (dyspnea alone or nonpleuritic pain alone) occurred in 18% of patients (Table 3).

### Symptoms and physical signs

The symptoms and physical signs of the 112 patients with pulmonary embolism are listed in Tables 4 and 5. Dyspnea, the most frequent symptom, occurred in 87% of the patients. Pleuritic chest pain occurred in 59%. An acute onset of either dyspnea or pleuritic chest pain occurred in 73% of the patients with pulmonary embolism. Cough, hemoptysis, syncope, nonpleuritic chest pain, calf pain, and palpitations each occurred in less than 30% of the patients (Table 3).

Tachypnea, the most frequent physical sign, occurred in 81% of the patients. Tachycardia and rales were the next most frequent physical findings at 67% and 46%, respectively (Table 5). An accentuated pulmonic closure sound, shock, fever (temperature > 37.8°C), pleural rub, and cyanosis were less common, with each noted in less than 20% of the patients (Table 5). Either dyspnea or tachypnea (> 20 breaths/min) occurred in 96% of patients. The combination of dyspnea and pleuritic chest pain occurred in 47% of patients. However, dyspnea, pleuritic chest pain, and hemoptysis occurred in 23% (Table 6).

### Arterial blood oxygen

Eighty-nine (79%) of the 112 patients with proven pulmonary embolism had arterial blood gases drawn while breathing room air. The distribution of levels of the arterial PO$_2$ is shown in the Figure. The most frequent range of arterial PO$_2$ was between 30 and 50.
and 59 mm Hg, which occurred in 33% of the patients. An arterial PO₂ greater than 80 mm Hg was present in 9%, and a PO₂ less than 40 mm Hg occurred in 6% of the patients.

Electrocardiographic features

Of the 112 patients studied, 83 had identifiable electrocardiograms at the time of the embolic event. The most common abnormality was nonspecific ST segment or T-wave changes, which occurred in 57% of patients. A normal electrocardiogram was seen in 10% of patients. An S₃T₃ pattern occurred in 7% of patients. A new right bundle branch block was noted in 14 (17%) of 83 patients, and six of these patients presented with either shock or syncope. Left axis deviation (≤ −30°) and right axis deviation (> 90°) occurred in 7% and 10% of the patients. New arrhythmias that seemed to accompany the onset of symptoms occurred in 12%, with atrial flutter or fibrillation being most common in 10% of the patients.

Chest roentgenographic and lung scan findings

The most commonly reported chest x-ray finding was infiltration (43%), followed by pleural effusion (37%). Atelectasis was noted in 19%. A normal chest x-ray was present in 18%. Ventilation and perfusion scans were obtained prior to pulmonary angiography in 46 (41%) of 112 patients. Of these scans, 22 (48%) were interpreted as indeterminate probability, 19 (41%) as high probability, and five (11%) as low probability.

Pulmonary angiography

Only 112 (37%) of the 302 patients who underwent pulmonary angiography had demonstrable pulmonary emboli. No angiographically related deaths occurred among these 112 patients. Of the 112 patients with pulmonary embolism, 108 had bilateral angiograms in which the most frequent lobar locations for emboli were the right lower lobe (36%) and the left lower lobe (33%). Right atrial, right ventricular, and pulmonary artery pressures were not consistently recorded. Of the 108 bilateral angiograms, 62% had bilateral involvement. One patient had a pulmonary digital subtraction angiogram which demonstrated a right lower lobe embolus.

Discussion

Pulmonary embolism is not a disease but a complication of deep venous thrombosis. The factors responsible for the development of deep venous thrombosis are obviously the major determinants for subsequent pulmonary embolization. As indicated in Table 2, recent immobilization, chronic heart disease, documented deep venous thrombosis, recent surgery, and malignancy were the most frequent predisposing factors. All of the patients in our study who had documented deep venous thrombosis by phlebography or venography had findings suggestive of proximal deep venous thrombosis (involvement of the veins of the thigh). Moser and LeMoine (12) demonstrated that proximal deep venous thrombosis carries a greater risk of embolization than distal (calf) venous thrombosis.

Chronic heart disease is a major risk factor for the development of thromboembolic disease, especially in patients observed with both atrial fibrillation and congestive heart failure (13). Chronic heart disease occurred in 28% of our patients, with most patients suffering from recurrent congestive heart failure.

Individuals who weigh 20% more than the standard weight for their age, sex, and frame have an increased incidence of thromboembolic disease (14). Adiposity in women was noted to be a factor for significant pulmonary embolism at autopsy (15). Obesity was the only identifiable risk factor in 7% of the patients in our study.

The association between malignancy and recurrent thromboembolic disease was first described by Trousseau (16). It has been suggested that recurrent pulmonary embolic events may presage the diagnosis of an occult neoplasm (17). In a review by Gore et al (17), an increased incidence of cancer of the breast, gastrointestinal tract, lung, and uterus was observed for two years following the diagnosis of deep venous thrombosis and pulmonary embolism. A shift in patients undergoing pulmonary angiography for suspected pulmonary embolism, from patients with primary cardiovascular disease to patients with neoplastic disease, has been reported (18). Malignancy was noted to be a major risk factor (24% of patients) in the development of pulmonary embolism in our review, which was comparable to the incidence in patients with chronic heart disease (28%). Lung, pancreas, gastric, and colonic neoplasms were the usual tumors associated with pulmonary embolic events in our patients.

The determination of an identifiable predisposing risk factor is important in formulating a clinical diagnosis of pulmonary embolism.
embolism. Of 215 patients with angiographically proven embolism and no preexisting cardiac or pulmonary disease, Stein et al (11) found that predisposing risk factors occurred in 76% of patients. In the urokinase pulmonary embolism trial (UPET) (19), 94% of patients with documented emboli had one or more recognized predisposing conditions. Only 5% of the patients in our study had no identifiable risk factors.

Syndromes

Categorizing pulmonary embolism in terms of syndromes helps to clarify the presenting clinical diagnostic patterns. The syndromes of pulmonary embolism include the circulatory collapse syndrome, pulmonary infarction syndrome, and uncomplicated pulmonary embolism syndrome (12).

By recognizing the various presenting syndromes of acute pulmonary embolism, the clinician can estimate the hemodynamic impact of the embolic event. In the study by Stein et al (11), the pulmonary angiographic severity scores were higher in syndromes characterized by circulatory collapse and the uncomplicated embolism syndrome. In contrast, the angiographic index of severity was lower in patients with the pulmonary infarction syndrome. Thames et al (20) found that most of the patients with syncope demonstrated 50% or greater obstruction of pulmonary blood flow.

Symptoms and physical findings

Several clinical studies have demonstrated the difficulty of diagnosing pulmonary embolism based on the history and physical examination (2,3). Nevertheless, some clues have been found based upon common but nonspecific signs and symptoms. Among patients with pulmonary embolism without preexisting cardiac or pulmonary disease, Stein et al (11) found that 96% had either dyspnea, tachypnea, or clinical signs of deep venous thrombosis. Our experience paralleled these observations, with either dyspnea or tachypnea occurring in 96% of our patients. The absence of dyspnea and/or tachypnea would be strong evidence for exclusion of the diagnosis.

Data collected from the UPET (19) revealed that the triad of pleuritic chest pain, hemoptysis, and thrombophlebitis was present in only 4% of patients with acute pulmonary embolism. The same study also showed that the triad of dyspnea, pleuritic chest pain, and hemoptysis was observed in only 28% of patients with pulmonary embolism. From our review, the combination of dyspnea, pleuritic chest pain, and hemoptysis was seen in 23% of patients. Stein et al (11) analyzed a subset of patients from the UPET study who had no history of previous cardiopulmonary disease. They found, even among patients with no cardiac or pulmonary disease, that the combination of dyspnea, pleuritic chest pain, and hemoptysis occurred in only 22% of patients with acute pulmonary embolism.

Arterial blood gases

Hypoxemia is a consistent and important clinical feature of acute pulmonary embolism and is present in most patients with pulmonary embolism (21). Bell et al (3) reported that the average PaO₂ was 60 mm Hg and that 10% of the patients had a PaO₂ greater than 80 mm Hg. McNeil et al (21) showed that no significant difference occurred in the PaO₂ of young patients with pleuritic chest pain who did or did not have pulmonary emboli.

In an editorial by Martin (22) concerning PaO₂ data from phase 2 of the UPET study, he contended that a PaO₂ above 80 mm Hg as being "normal" may be misleading without knowledge of the PaCO₂ and the alveolar-arterial gradient to allow for estimation of the contribution of hyperventilation to normoxemia. The Figure illustrates the varied ranges of partial pressure of oxygen in our patients with acute pulmonary embolism. As noted, most patients demonstrated moderate hypoxemia; however, 9% had a PaO₂ greater than 80 mm Hg, which is in accordance with Bell et al's study (3). However, the alveolar-arterial gradients were not evaluated.

Using their inert gas technique, Wagner et al (23) studied the causes of hypoxemia. Recent investigators, using this method, demonstrated that during the initial phase of the pulmonary embolic event hypoxemia was the result of perfusion of lung units with a low ventilation/perfusion ratio. Shunt as a major factor of hypoxemia was noted to occur later in the course of the embolic event (48 hours or more) and only if atelectasis or other factors leading to loss in lung volume were present (24).

Chest radiographic findings

A normal chest radiograph was found in 18% of the patients in our series. The presence of a normal chest radiograph was seen in 24% of 169 patients with angiographically proven pulmonary embolism in the National Heart, Lung and Blood Institute (NHLBI) urokinase-streptokinase pulmonary embolism trials (25).

In our review, the most frequent radiographic findings were infiltration, pleural effusion, and atelectasis. These parenchymal signs were more common in patients with the pulmonary infarction syndrome. Other chest radiographic abnormalities may include elevation of the hemidiaphragm on the side of the embolus, enlarged pulmonary arteries, a difference in vascularity of the two sides (Westermark's sign), and cardiomegaly secondary to right ventricular enlargement (25). Greenspan et al (26) evaluated whether the chest radiograph alone could be used to diagnose or exclude pulmonary embolism. These investigators found that the chest x-ray can provide additional information, especially to exclude other disease processes (rib fracture or pneumothorax). On the other hand, Stein et al (25) found frequent radiographic signs that may give clues to the diagnosis. In particular, an elevated hemidiaphragm was found in 26% of patients with pulmonary embolism. In our study, the chest x-ray report of an elevated hemidiaphragm was rare (only 5%), but this may have represented a lack of description of this finding. It would be important to consider a normal chest x-ray, in the clinical setting of acute dyspnea with hypoxemia and no evidence of bronchospasm, to be strongly suggestive of acute pulmonary embolism.

Electrocardiogram

In patients without prior cardiopulmonary disease, electrocardiographic abnormalities were present in 87% of 90 patients with angiographically documented emboli (27). The most common abnormalities were nonspecific T-wave changes in 42% of patients, with RST-segment abnormalities present in
41%. We also found nonspecific ST segment and T-wave changes to be the most common abnormality. Left axis deviation and right axis deviation occurred at equal frequency (7%) (27). Signs of cor pulmonale (S_qR, right bundle branch block, P-pulmonale, or right axis deviation) were noted in 26% of patients with submassive pulmonary embolism and in 32% with massive pulmonary embolism (27). Although we did not separate our patient group into massive or submassive categories, we noted that in six of 14 with syncope or shock a new right bundle branch block was present. In the UPET study (19), 23% of patients with submassive pulmonary embolism had normal electrocardiograms, whereas in our study 10% of patients had normal electrocardiograms. Although no specific electrocardiographic features are diagnostic of pulmonary embolism, these nonspecific ECG abnormalities in the proper clinical setting support the diagnosis.

Arrhythmias occurred in 12% of our patients, most of which were atrial fibrillation or atrial flutter. This finding occurred more frequently in our study than in others, which we cannot explain. In 90 patients with pulmonary emboli without previous cardiopulmonary disease, no arrhythmia occurred (27). Of the 23 patients with acute pulmonary embolism and no heart disease in Szucs et al's study (28), only 4% had atrial flutter or atrial fibrillation. Even in patients who had previous cardiac or pulmonary disease, only 3% had premature atrial contractions, 5% had atrial fibrillation, and 9% had premature ventricular contractions (19).

**Lung scans**

The clinical utility of ventilation perfusion imaging has come under considerable controversy recently, especially in regard to focusing on the sensitivity and specificity of this noninvasive test (29). Several retrospective studies have compared scintigraphic findings with pulmonary angiographic findings, resulting in different criteria for the interpretation of ventilation perfusion scans in patients with pulmonary embolism (30,31). These studies showed that a scan pattern characterized as multiple mismatched perfusion defects, involving greater than 75% of the bronchopulmonary segment, is associated with a high probability of pulmonary embolism (90%). In a lung scan that demonstrates perfusion defects, matching with radiographic and ventilation abnormalities, the likelihood of pulmonary embolism is indeterminate (25% to 35%). A scan pattern which shows that all perfusion defects are less than 25% of the segment and are matched with ventilation abnormalities but not to chest radiographic abnormalities has a low likelihood of pulmonary embolism (less than 10%).

This scheme has been challenged by a prospective Canadian study (29), which suggests that low probability scan patterns may be associated with a higher incidence of venous thromboemboli than previously reported. Nevertheless, a completely normal ventilation perfusion scan virtually eliminates the diagnosis of pulmonary embolism (32,33).

Our institution is currently involved with a multicentered NHLBI study of the prospective investigation of pulmonary embolism diagnosis (PIOPED), which assesses the efficacy of ventilation perfusion scanning.

**Pulmonary angiography**

Pulmonary angiography continues to be the “gold standard” test for the diagnosis of pulmonary embolism (7,8). In the UPET study (19), in which over 800 pulmonary angiograms were performed, the morbidity was less than 1% and the mortality was less than 0.01%. A review of 1,350 angiograms performed at Duke Medical Center over an 11-year period revealed only three deaths (34). There were no angiographically related deaths in our series.

A negative pulmonary angiogram, despite even a strong clinical suspicion, excludes the diagnosis of pulmonary embolism. Among 167 patients with clinically suspected pulmonary embolism who had negative pulmonary angiograms and who were followed for six months, none had a clinical event suggestive of pulmonary embolism (35). Those who died and had postmortem examinations did not demonstrate pulmonary emboli.

**Summary**

We retrospectively reviewed the clinical features, laboratory data, chest radiographic reports, and lung scans of patients with angiographically proven pulmonary embolism to reassess the features of pulmonary embolism which may assist the clinician in selecting patients for confirmatory diagnostic studies. We realize evaluation of the clinical features of pulmonary embolism only in those patients with angiographic documentation creates a degree of bias when determining these clinical characteristics.

The clinical features of pulmonary embolism have been demonstrated in the literature as being nonspecific. However, the following clinical parameters should be sought in selecting patients with suspected pulmonary embolism for pulmonary angiography: 1) appropriate predisposing factors; 2) an acute, unexplained onset of dyspnea, tachypnea, or tachycardia; 3) arterial blood gases demonstrating hypoxemia or a widening of the alveolar-arterial oxygen gradient; 4) an electrocardiogram showing nonspecific ST segment and T-wave changes; 5) a normal chest radiograph in the presence of unexplained dyspnea or showing an elevated hemidiaphragm or parenchymal abnormalities; and 6) abnormal ventilation perfusion scans, which, whether of low or indeterminate probability, in the presence of the above findings warrant angiographic pursuit.

**Acknowledgment**

We thank Ida Borum for her assistance in the preparation of this manuscript.

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