DESQUAMATIVE INTERSTITIAL PNEUMONIA MASQUERADING AS ACUTE LIFE-THREATENING PULMONARY EMBOLISM

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Severe right ventricular strain in a patient with acute onset of pleuritic chest pain, hemodynamic collapse, and hypoxemia is strongly suggestive of an acute life-threatening pulmonary embolism.1 Right ventricular strain and elevated pulmonary artery pressures, however, are not restricted to acute pulmonary embolism. These features can also be present in other conditions, including severe interstitial lung disease and an extensive right ventricular infarct.2

We present a patient with clinical and echocardiographic features of massive pulmonary embolism who was taking warfarin for recently diagnosed pulmonary embolism. Thrombolysis was considered as a possible treatment but was not undertaken. After respiratory and cardiovascular stabilization, a pulmonary angiogram did not reveal any large embolus. Desquamative interstitial pneumonitis and bronchopneumonia were found at autopsy. Thus, conditions with severe right ventricular strain and shock may masquerade as a large pulmonary embolus.1,3 Thrombolysis without further diagnostic imaging should be pursued cautiously in such cases.

Case Presentation

A 44-year-old man had acute onset of shortness of breath, right-sided pleuritic chest pain, and orthopnea. He had not had fever, chills, or productive cough. Pertinent medical history included splenectomy after a motor vehicle accident, peptic ulcer disease, and extensive deep venous thrombosis of the lower extremity with pulmonary embolism diagnosed 5 months before this visit.

Medications included warfarin, gabapentin, paroxetine, anagrelide, and rofecoxib. He was a heavy smoker and had not used any intravenous drugs. His sister had deficiency of protein S, an antithrombotic cofactor to protein C, causing hypercoagulability.

Physical examination revealed a slender ill-appearing man in respiratory distress. His body temperature was 37.8°C, blood pressure was 80/50 mm Hg, and heart rate was 120/min. Minimal rales at the right lung base were audible. On examination of the heart, the point of maximal impulse was shifted to the midline, and a loud S2 was audible. Jugular venous distention was not present. Percussion indicated an enlarged liver. The left thigh was tender and edematous, with a larger circumference than the right thigh. Stool was positive for occult blood.

A complete blood cell count revealed a white blood cell count of 15 x 10^9/L (80% granulocytes), a hemoglobin level of 79 g/L, and platelet count of 796 x 10^9/L. The international normalized ratio was 2.0. The serum iron level was 0.4 µmol/L (2 µg/dL; normal: 9.0-28.6 µmol/L [50-160 µg/dL]), the troponin level was 3.1 µg/L (normal: 0-5 µg/L), and the lactate level was 7.8 mmol/L (normal: 0.5-1.6 mmol/L). Arterial blood gas values (on room air) were pH 7.45, PCO2 25 mm Hg, and PO2 39 mm Hg. Results of a D-dimer assay were normal. A chest radiograph revealed a poorly defined opacity in the right lower lobe and elevation of the right hemidiaphragm. An electrocardiogram indicated a new right bundle branch block (RBBB). An echocardiogram indicated severe right ventricular strain, with dilated right atrium and ventricle, normal global left ventricular systolic function, and moderate tricuspid regurgitation. These findings were new compared with the study done a few months earlier. No evidence of ventricular or atrial clot was seen. Right ventricular systolic pressure was estimated at 39 mm Hg.

The patient was intubated and was given crystalloids and pressors. A massive pulmonary embolism was suspected, and heparin was started. The use of thrombolytic therapy was considered but was deferred.
because of concern for bleeding in a patient who was receiving anticoagulants, had a positive test for occult blood in the stool, and had a history of peptic ulcer disease. The next day, a Doppler sonogram revealed an old and chronic large clot in the left thigh. A ventilation-perfusion scan showed multiple matched defects and was considered low probability for pulmonary embolism. A pulmonary arteriogram showed no filling defect. The right and left pulmonary artery pressures were 41/18 and 55/18 mm Hg, respectively, consistent with pulmonary hypertension.

Within 72 hours, the patient’s condition deteriorated. He became less alert and severely hypotensive. Because of splenectomy and the possibility of sepsis and bacteremia, broad-spectrum antibiotics were administered. Blood cultures did not grow any microorganism. The serum cortisol level was normal, but the troponin level was elevated. Computed tomography of the chest revealed interstitial opacities of the right and left lower lobes. The patient remained persistently hypotensive despite treatment with crystalloids, dopamine, and norepinephrine. His condition eventually deteriorated to pulseless electrical activity and ventricular tachycardia. Resuscitation was unsuccessful, and he died on the fifth day of hospitalization. At autopsy, no pulmonary embolus was found. The lungs were firm, darkly pigmented, and focally congested. Sections showed early patchy bronchopneumonia, centriacinar emphysema, and desquamative interstitial pneumonitis.

Discussion

In our case presentation, the presence of new RBBB, right ventricular dysfunction, pulmonary hypertension, hypoxemia, and hemodynamic shock were all strongly suggestive of an acute and extensive episode of pulmonary embolism. However, the patient had desquamative interstitial pneumonitis and superimposed bronchopneumonia, not pulmonary embolism.

RBBB as a sign of right ventricular overload is an electrocardiographic feature of, but not unique to, pulmonary embolism. In conjunction with right-axis deviation, RBBB is seen in the following conditions: (1) right ventricular hypertrophy, due to elevated pulmonary artery pressure of any cause, or obstruction of the outflow tract of the right ventricle; (2) obstructive and restrictive pulmonary diseases due to cor pulmonale, lowering of the diaphragm, and decreased conductance of the pulmonary parenchyma; and (3) coronary artery disease with inferior or posterior wall myocardial infarction.

Echocardiography is an important tool in the diagnosis of acute and massive pulmonary embolism, in which tricuspid valve regurgitation and an increase in right ventricular end-diastolic diameter and systolic pressure may be present. The last 2 findings are usually seen irrespective of the findings on the electrocardiogram. A large atrial or ventricular clot may also, on occasions, be demonstrable by echocardiography.

Right ventricular strain and dilatation and pulmonary hypertension are not unique to pulmonary embolism. In patients with preexisting lung disease, the previously described electrocardiographic and echocardiographic findings can be easily misinterpreted as indicating pulmonary embolism. Patients with moderate to severe restrictive lung disease have evidence of increased thickness and enlargement of the right ventricular wall, as well as pulmonary hypertension. Shivkumar et al found that enlargement and thickening occurred when forced vital capacity was 57% or less of its predicted value. Restrictive lung diseases range from interstitial lung disease (including asbestosis and sarcoidosis) to lung cancer, respiratory muscle dysfunction, scoliosis, and obesity. Desquamative interstitial pneumonitis, although not specifically pointed out in the study of Shivkumar et al, falls into the category of interstitial lung diseases and can generate such cardiac findings. Desquamative interstitial pneumonitis is predominantly a bronchiolitis of monotonous histological uniformity that occurs in smokers.

**Desquamative interstitial pneumonia is predominantly a bronchiolitis with uniform histological findings that occurs in smokers.**

Thrombolysis can prevent right ventricular collapse with ensuing respiratory failure and death in patients with life-threatening pulmonary embolism. As indicated by echocardiography, right ventricular function and pulmonary perfusion improve within the first 24 hours of thrombolysis, although the long-term effect of thrombolysis on reducing the mortality has not been clearly established.

The idea of requiring definitive proof of pulmonary embolism before thrombolytic treatment has been a topic of debate. Some clinicians may consider thrombolytic therapy in patients with hemodynamic instability solely on the basis of severe right ventricular dysfunction or hypokinesia shown by echocardiography. The consensus statement of the American College of Chest Physicians on pulmonary embolism is not unanimous on this issue. Some clinicians require unequivocal angiographic proof of pulmonary embolism before they will begin thrombolysis. Others think that in patients with high probability of pulmonary embolism, angiography can be avoided and a noninvasive test,
such as duplex sonography of the extremity for detecting deep venous thrombosis or echocardiography for assessing ventricular dysfunction, will suffice. In fact, some think that angiography may increase the risk of bleeding in patients who undergo subsequent thrombolysis and thus advocate noninvasive techniques for diagnosing life-threatening pulmonary embolism. Two other noninvasive tests than can be used are ventilation-perfusion scan and spiral computed tomography, neither of which has the sensitivity or the specificity of angiography. Unfortunately, these diagnostic tests, as well as pulmonary angiography, cannot be done effectively in a patient who is in unstable condition in the intensive care unit. Therefore, at times the only noninvasive test that can be used is bedside echocardiography, a tool that merits future investigation.

The cause of death in our patient was not a pulmonary embolus, chronic thromboembolism, or left ventricular failure. Bronchopneumonia and possibly sepsis superimposed on interstitial lung disease could have caused his death.

**Presence of right ventricular dysfunction, new right bundle branch block, hypoxemia, and shock do not always indicate extensive pulmonary embolism.**

This case conveys an important message to physicians and nurses: the presence of right ventricular dysfunction, new RBBB, and hypoxemia in conjunction with hemodynamic shock does not unequivocally imply occurrence of an extensive pulmonary embolus. Even when the probability of pulmonary embolism is high, clinicians should be aware of the limitations of noninvasive tests, such as echocardiography, in the diagnosis of life-threatening and massive pulmonary embolism. In fact, the presence of interstitial lung disease and possibly pneumonia could further complicate the diagnosis. Sole reliance on echocardiography, an excellent and efficient bedside test, may be inadequate.

**REFERENCES**

Desquamative Interstitial Pneumonia Masquerading as Acute Life-Threatening Pulmonary Embolism
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