**Introduction**

Acute pulmonary embolism (PE) is a life-threatening disease, which mostly results from deep venous thrombi of lower extremities. The symptoms and signs are vague and similar to many other diseases. Here, we describe an elderly patient who developed typical symptoms and objective findings of acute coronary syndrome after a neurologic operation. Despite an initial electrocardiogram (ECG) suggestive of posterior myocardial infarction (MI) or unstable angina (UA), his coronary arteries were patent on coronary angiography. Further examinations showed the presence of acute PE. The usefulness of bedside echocardiography should be considered to distinguish acute coronary syndrome from PE in these patients.

**Case Report**

A 67-year-old man, a current smoker, was admitted because of progressive low back pain with gait disability for at least 1 year. A review of past medical illness revealed gout and hypercholesterolemia for the previous 3 months. A series of examinations demonstrated normal coronary arteries. Further investigations demonstrated a final diagnosis of acute pulmonary embolism. Acute pulmonary embolism with simultaneous recent neurosurgery was a therapeutic dilemma because of the risk of postoperative hemorrhage threatening neurologic function. After treatment with enoxaparin and close monitoring of his neurologic condition, his symptoms were eliminated. Clinicians must keep in mind a differential diagnosis of pulmonary embolism in a postoperative high-risk patient. [International Journal of Gerontology 2009; 3(4): 251–255]
The symptoms could not be relieved by rest. Physical examination revealed a heart rate of 106/min, respiratory rate of 22/min, blood pressure of 100/62 mmHg, and room air oxygen saturation of 93% by pulse oximetry. The breathing sounds were clear, but the heart sound displayed a 3/6 grade blowing mid-systolic murmur at the fifth intercostal space near the sternum. There was no ankle edema. An ECG taken immediately showed sinus tachycardia with new onset of ST depression without T wave inversion at V1 to V6 (R/S > 1 at V1–2) and a S1Q3T3 pattern (Figure 1A). Initially, acute posterior MI or UA was suspected. A thrombolytic agent was not indicated owing to his recent major operation. Because of refractory chest pain after morphine and sublingual nitroglycerin therapy, primary coronary catheterization was planned. Surprisingly, the coronary angiography showed normal coronary arteries, and left ventriculography displayed no wall motion abnormality (Figures 2A and 2B). The arterial blood gas analysis presented 129 mmHg of PaO2 under 35% oxygen therapy without an acid-base imbalance. Because the patient had persistent chest pain and jaw numbness, an echocardiography was performed that demonstrated paradoxical ventricular septal wall motion and D-shaped left ventricle in cross section. A peak systolic pulmonary arterial pressure of 50 mmHg and a moderate tricuspid regurgitation were also found. It made us highly suspect acute PE. Right heart catheterization was planned immediately, which revealed a mean pulmonary arterial pressure of 45 mmHg. The pulmonary arteriography manifested multiple large filling defects in bilateral pulmonary arteries (Figure 2C). Acute PE was diagnosed, but emergency thrombolytic therapy was not administered because of relative stable hemodynamics and because the patient was recovering from a major operation. Anticoagulant (enoxaparin, 1 mg/kg) was given, and the patient was admitted to the intensive care unit for close monitoring of vital signs and neurologic condition. Further examinations included the lung ventilation–perfusion scintigraphy, demonstrating multiple large perfusion defects in bilateral lobes compatible with a high probability of PE. The D-dimer level was as high as 5,108 ng/mL. The peripheral venous echogram revealed many thrombi in the bilateral femoral and popliteal veins. Chest pain/tightness, jaw numbness and tachycardia were relieved 3 days after anticoagulant therapy. The follow-up ECG showed a remission of the ST depression and S1Q3T3 pattern (Figure 1B). There was no elevated serum cardiac biomarker during hospitalization. According to the above-mentioned examinations, oral warfarin therapy was prescribed for 3 months. There was no recurrence of PE after stopping anticoagulant therapy.

Discussion

Acute PE is a common disease and has a high mortality rate of nearly 30% without therapy. Mortality can be reduced by accurate diagnosis followed by effective anticoagulant therapy. Because of nonspecific manifestations of acute PE, it can lead to an erroneous diagnosis and compromise the patient’s outcome. The most common symptoms are dyspnea with or without exertion, pleuritic pain, and cough. The most common signs are tachypnea, tachycardia, and rales1. In addition, the ECG is usually nonspecific, and around one-third of patients have normal findings. The common ECG findings of suspicious PE are sinus tachycardia, T wave inversion in precordial leads V1 through V4, and transient right bundle branch block. The S1Q3T3 pattern also suggests PE and is found in around 50% of patients2. Typical chest pain and jaw numbness rather than dyspnea combined with ECG ST depression (no T wave inversion) in the precordial lead, which mimics acute coronary syndrome, was rarely reported. We present an elderly patient who had received lumbar laminectomy and developed acute PE on the fifth postoperative day with an initial impression of acute posterior MI or UA. The identifiable risk factors of acute PE include immobilization, stroke, paresis, surgery in the past 3 months, paralysis, history of venous thromboembolism, central venous instrumentation in the past 3 months, and chronic heart disease1,3–5. In women, risk factors such as obesity (body mass index > 29 kg/m2), heavy cigarette smoking (>25 cigarettes/day), and hypertension have also been identified6. Laminectomies were rarely reported to cause PE7. For our patient, the probable etiologies of acute PE were immobility during operation and chronic tobacco use. Interestingly, he experienced typical anterior compressive-like chest pain associated with jaw numbness and cold sweating rather than dyspnea that made us highly suspect posterior MI or UA on ECG presentation. Thrombolytic therapy was postponed because of a high risk of postoperative hemorrhage threatening neurologic impairment. Primary coronary catheterization was only indicated in this emergent
There were no critical obstructive coronary diseases on coronary angiogram. Could we have made a more accurate diagnosis before the angiogram was performed?

On reviewing the course of management, cardiac ultrasound could have contributed to a differential diagnosis in such patients. Early thrombolysis is a guideline for saving lives in those with ST elevation during

**Figure 1.** (A) Electrocardiogram (ECG) showing sinus tachycardia with new onset of ST depression without T wave inversion at V1 to V6 (arrow sites) and a $S_1Q_3T_3$ pattern. (B) Follow-up ECG showing normal sinus rhythm with remission of ST depression and $S_1Q_3T_3$ pattern.
MI, but is not recommended in non-high-risk patients of PE. The common echocardiographic signs of PE are an enlarged right ventricle, paradoxical ventricular septal wall motion, the McConnell sign (right ventricle, free wall hypokinesis with sparing of the apex), and pulmonary hypertension with a tricuspid regurgitant jet velocity $>2.6$ m/sec. For our patient, although the presentation of the McConnell sign was lacking, which had been described in instances of massive PE, paradoxical ventricular septal wall motion made us highly suspect acute PE. With relatively stable hemodynamics, anticoagulant therapy was the first-priority aim, followed by closely monitoring the neurologic condition. On the other hand, if massive acute PE with right ventricular dysfunction occurred, surgical embolectomy or percutaneous mechanical thrombectomy were considered as therapeutic alternatives. Pneumatic compression stocking prophylaxis is an alternative means and can effectively decrease the incidence of deep venous thrombosis and PE in high-risk patients undergoing multilevel lumbar laminectomies with instrumented fusions. Finally, our patient was strongly advised to abstain from smoking and warfarin was administered for 3 months according to the guideline.

Although the initial management of acute coronary syndrome and PE may be similar, some critical conditions are different. For example, thrombolysis is the first-line therapy in high-risk PE with cardiogenic shock and persistent hypotension. Administration within 48 hours of symptom onset is associated with the greatest benefit, but it can still be effective 6–14 days after onset of symptoms. However, thrombolytic therapy is indicated in patients with ST elevation MI within 12 hours of symptoms onset. Besides, low molecular weight heparin only cannot be effective for high-risk PE with unstable hemodynamics. Those with PE would need anticoagulation therapy for at least 3 months, but it is not a standard therapy with MI.
In conclusion, this case report suggests that acute PE mimicking acute coronary syndrome may develop in elderly patients undergoing major operations with multiple atherosclerotic risk factors. Despite that ECG is unreliable for diagnosis of acute PE, it can show the initial presentation of a right ventricle strain, and further investigation is necessary to differentiate the diagnosis. We emphasize that doctors must evaluate PE in all patients suffering from typical chest pain with ischemic ECG changes.

References