Case Report

Concurrent pulmonary embolism and acute coronary syndrome with dynamic electrocardiographic changes

Abstract

Concomitant occurrence of pulmonary embolism and acute coronary syndrome is rare. The early diagnosis and treatment of acute coronary syndrome with right ventricular myocardial ischemia during acute pulmonary embolism (APE) are crucial. The irreversible right ventricular myocardial dysfunction is a major risk factor for mortality from APE. In this case report, we present a 66-year-old female patient with APE who had a significant right coronary artery (RCA) lesion, which was successfully treated with angioplasty and stent implantation.

Acute pulmonary embolism (APE) is a relatively frequent cardiovascular emergency that generally appears with chest pain and shortness of breath. The overall inhospital mortality rate of APE has been reported to be 7% to 11% and raises to 25% among those with shock and to 65% among those with cardiopulmonary arrest [1,2]. Life-threatening severe right ventricular dysfunction may develop because of acute obstruction of pulmonary arterial bed. Irreversible functional impairment of right ventricle is a major risk factor for mortality. Therefore, acute coronary syndromes (ACSs), which accompany APE and affect the right ventricle functions in particular, should be determined and treated in the early period.

In the present case, a patient diagnosed with APE was successfully treated by percutaneous intervention of the RCA for the concurrent ACS.

A 66-year-old woman was admitted to our emergency department with complaints of shortness of breath and burning chest pain for 4 hours. She also described swelling and pain in her left leg that occurred after a 4-hour bus trip a week ago. She was transferred to the coronary care unit for the differential diagnosis of the chest pain. On her physical examination, blood pressure was 180/100 mm Hg, and heart rate was 104 beats per minute and regular. The respiratory rate was 30 per minute, and oxygen saturation was 90% in room air. Her electrocardiogram (ECG) revealed a sinus rhythm with incomplete right bundle-branch block, negative T waves, and 0.5-mm ST-segment depression in leads V₁ to V₆ (Fig. 1). Bedside transthoracic echocardiographic examination revealed an enlarged right ventricle with akinetic mid and basal segments and hyperkinetic apical segment (McConell’s sign). There was moderate tricuspid insufficiency, and the estimated pulmonary artery pressure was 45 mm Hg. Because of the absence of shock or resistant hypotension, fibrinolytic therapy for APE was not considered, and she was scheduled for elective multislice computed tomography (MSCT) imaging. She was treated initially with enoxaparine (0.1 mg/kg, BID), acetylsaliclyc acid (300 mg/d), and metoprolol (50 mg/d). Biochemical analysis showed increased d-dimer (2500 ng/mL), troponin I levels (1.2 U/L), and normal creatinine kinase/creatinine kinase-MB levels. On the second day of her hospitalization, she had burning chest pain that impaired hemodynamic stability (systolic blood pressure, 80-90 mm Hg) and was accompanied by paroxysmal atrial fibrillation (PAF) (Fig. 2). Because the PAF episodes became frequent and impaired the hemodynamic stability, coronary angiography was performed to eliminate an acute coronary event. Coronary angiography revealed 60% to 70% tubular narrowing of the mid segment of the left anterior descending artery (LAD) and 95% to 99% narrowing of the mid proximal segment of the RCA (Fig. 3A). Percutaneous transluminal coronary angioplasty (PTCA) was performed for the severe obstruction in RCA. The obstruction in the RCA was dilated via a 2.0 × 14 mm mercury balloon (Abbott Laboratories, Abbott Park, IL), and a 2.75 × 16 mm bare metal stent (20 atm, 3.3 mm; Flexmaster [JoMed GmbH, Rangendirgen, Germany]) was implanted (Fig. 3-B). After the intervention, the patient was hemodynamically stable, and the MSCT pulmonary angiography was performed 3 days after the procedure. Multislice computed tomography imaging showed diffuse thrombus in both of the main pulmonary arteries extending through to the distal segment (Fig. 4). Doppler ultrasound examination also showed thrombus in the deep femoral vein of the left lower extremity. On the fifth day, PTCA was performed for the lesion in LAD. The patient was discharged on the eighth day with warfarin, acetylsaliclyc acid, clopidogrel, atorvastatin, metoprolol, and ramipril.

Acute pulmonary embolism is an urgent clinical condition resulting from acute obstruction of pulmonary artery and the branches as well. The patients with acute massive pulmonary embolism (PE) that progresses to shock and hypotension due to severe right heart failure die in a short time.
Inhospital mortality rate among overall APE cases is reported to be 7% to 11%, whereas it was 25% in those with shock and 65% in those with cardiopulmonary arrest [1,2].

Acute coronary syndrome concomitant with APE is quite rare, and it is difficult to diagnose because of similar clinical findings and symptoms. Acute coronary events observed in the course of APE can be evaluated in 3 parts in the guidance of the literature.

The first one is the right ventricular ischemia or infarction developed due to the increased right ventricular afterload that is secondary to massive pulmonary embolus. In these cases, in general, no significant coronary artery obstruction that might lead to the right ventricular ischemia has been determined [4,5]. However, based on the autopsies of 6 cases with massive PE accompanied by isolated right ventricle myocardial infarction, coronary arteries were determined to be normal in 1 case, whereas significant obstruction was determined in the right or left coronary arteries of the remaining 5 cases. It was stated that these obstructions might partly be responsible for the right ventricle necrosis in APE [6].

Paradoxical coronary embolism is the other rare cause of ACS that accompanies APE. Myocardial infarction secondary to APE has been reported in the patients who had not been diagnosed with patent foramen ovale (PFO) before [7-9]. Thrombus that resembles embolus was observed in the coronary arteries on coronary angiographies performed in these patients because of the changes in ST-T segment observed on ECG in the course of APE. Patent foramen ovale, which allows thrombus to pass into the left atrium from the right atrium, thereafter, into the coronary artery system, was determined on echocardiographic examinations.

The third cause that leads to ACS in APE is the presence of severe obstruction in coronary arteries. A patient who underwent coronary angiography because of acute chest pain and elevated troponin levels in the course of APE and who was performed a percutaneous coronary intervention for the significant obstruction in LAD has been reported in the literature [10]. In the present case, percutaneous coronary intervention was performed to the significant obstruction in the RCA, which led to ACS. Vasospasm due to increased catecholaminergic discharge and the activation

**Fig. 1** Electrocardiographic findings at the time of admission to the hospital: sinus rhythm, incomplete right bundle-branch block, 0.5-mm ST-segment depression, and negative T waves in leads V1 to V6 and negative T wave in D3.
Fig. 2  Paroxysmal atrial fibrillation that accompanied chest pain and hypotension on the second day of hospitalization.

Fig. 3  A, The image of 95% to 99% tubular narrowing in the mid proximal segment of the RCA. B, The image after PTCA and stent implantation.
of coagulation system are likely the causes of preexisting coronary artery obstruction to become symptomatic. Systolic functions of the right ventricular myocardium, which have been affected in APE, have worsened because of the increase in the severity of the obstruction in RCA. Disappearance of the arrhythmias and the hypotension after percutaneous revascularization process performed in the present case revealed the importance of acute coronary events in the course of APE and raised the thought that they should be diagnosed and treated as soon as possible.

Electrocardiographic findings in APE are generally normal but neither sensitive nor specific. Sinus tachycardia and nonspecific changes of ST-segment and T wave are the most frequently encountered findings. Inversion of T waves in precordial derivations that include right ventricular overload, S1Q3T3 pattern, transient right bundle-branch block, sudden atrial fibrillation, and other atrial arrhythmias are the strong ECG changes that indicate PE [11]. In the present case, there were incomplete right bundle-branch block, Q waves, and T wave inversion in D3 that indicates right ventricular overload. These ECG findings are the changes that are expected to be present in APE. T wave changes in anterior derivations may be either nonspecific changes encountered in APE or ischemic changes developed due to the obstruction in LAD. Furthermore, frequent PAF attacks observed in this case may be due to the increased sympathetic activity in APE; however, it should be kept in mind that they might also occur because of the acute ischemic coronary events in the patients with chest pain.

Elevated troponin levels in APE have been found to be associated with mortality [12]. Rapid and severe increase in right ventricle pressure leads to myocardial ischemia in right ventricle as well as troponin release. In this case, troponin level was determined to be increased at the time of admission. This elevation resulted either from the increase in right ventricular pressure or from the right ventricular ischemia because of ACS.

It should be kept in mind that acute coronary events might accompany APE, which progresses with severe impairment in right ventricular function, in the presence of refractory atrial arrhythmia and chest pain. Early diagnosis and treatment are mandatory, particularly in patients who have coronary artery involvement that leads to right ventricular ischemia.

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References