Acute pulmonary embolism mimicking inferior myocardial infarction

İnferiyor miyokart enfaktüsünü taklit eden akut pulmoner emboli

Sadık Volkan Emren, M.D., Mehmet Erdinç Arıkan, M.D., Oktay Şenöz, M.D., Eser Varış, M.D., Erol Akan, M.D.

Department of Cardiology, Katip Celebi University Faculty of Medicine, İzmir

Summary– Pulmonary embolism (PE) is a potentially life-threatening emergency that is sometimes difficult to diagnose due to nonspecific symptoms and findings. A remarkable number of cases of PE with ST elevation in anterior leads have been reported in the literature. However, PE with ST elevation in the inferior leads on ECG is very rare.

CASE REPORT

A 69-year-old male patient was admitted to a chest center with new-onset chest pain and sweating. He had no history of coronary artery disease. He was a smoker - 30 pack-years - and had poorly controlled hypertension. On the first physical examination, his blood pressure was 120/70 mmHg, pulse 80 bpm/rhythmic, and respiratory rate 20/min, and normal heart and lung sounds were present. Arterial blood gas analysis showed pH 7.42, \( PO_2 \) 59 mmHg, \( PCO_2 \) 38.3 mmHg, \( HCO_3^- \) 26.1 mmol/L, and Sat\( O_2 \) 92%. ECG revealed sinus rhythm with ST elevations in the inferior leads (Figure 1), and therefore, the patient was transferred to our center with the diagnosis of ST elevation myocardial infarction.

Abbreviations:

- ECG: Electrocardiogram
- PE: Pulmonary embolism
- RBBB: Right bundle branch block
few noncritical coronary artery plaques (Videos 1, 2 and 3*), with normal left ventriculography. Subsequently, an aortogram was performed to rule out aortic dissection, which may compress the right coronary ostium, and the results were reported as normal in size and morphology, with no evidence of false lumen observed (Video 4*). A 7F sheath was placed in the femoral vein, right heart catheterization was per-
formed, and an elevated pulmonary artery pressure of 45/23 mmHg was revealed. A pulmonary angiogram was then performed with a pigtail catheter from the pulmonary trunk and right pulmonary artery, respectively. Right pulmonary artery opacification showed occlusion at the level of the right lower lobe pulmonary artery (Figure 2a). Surprisingly, however, the selective injection with 20 ml/600 psi caused the occlusion to recanalize partially, due to fragmentation of the ‘plug’, which scattered to distal segmental arteries (Figure 2b). Diagnosis of acute PE was established. Low molecular weight heparin and oral warfarin were started on the same day, and heparin was stopped two days later when international normalized ratio (INR) was over 2.0. The patient was almost clinically free of symptoms after the procedure, and the ECG had reverted to normal after one hour in the coronary care unit (Figure 3). The echocardiogram showed good left ventricular systolic function with slightly enlarged right ventricle (30 mm). Echocardiography estimated right ventricular systolic pressure as 40 mmHg. Maximal troponin I value (1.1 mcg/L) was reached at 24 hours. Ultrasound of the lower extremity deep veins revealed right femoral vein thrombosis.

DISCUSSION

Pulmonary embolism (PE) is a potentially life-threatening emergency that is sometimes difficult to diagnose due to nonspecific symptoms and findings. More than 90% of cases present shortness of breath and chest pain. Chest pain is usually pleuritic, which is due to pulmonary infarction. Chest pain is also related with myocardial ischemia due to the increased right ventricular afterload and a disproportion between oxygen demand and supply. The ECG is variable in acute PE, and sinus tachycardia is the most common. Choue and coworkers described the typical ECG features of PE, which may be summarized as S1Q3T3 pattern, right axis deviation, transient incomplete or complete right bundle branch block (RBBB), and negative T waves in the right precordial leads. Sreeram et al. reported that three or more of the following findings support the diagnosis of PE: incomplete or complete RBBB, deep S waves in D1 and aVL, Q waves in DIII and aVF, clockwise deviation of transition zone to V5, right axis deviation, low QRS voltage in extremity leads, and negative T waves in the anterior or inferior leads. ST elevation is rarely related to PE on the basis of a few case reports.

Although the pathophysiology of ST elevation in PE is unknown, a thrombus embolizing from deep leg veins may also pass to the left side of the heart via patent foramen ovale or atrial septal defect, the final destination being the coronary arteries. However, in this situation, there is either blockage or filling defects in the affected coronary artery. The other explanation may be that the right ventricle cannot tolerate the acutely increased afterload burden, and focal or diffuse ischemia ensues due to strain, which may also trigger coronary spasm. On the other hand, there is a long list of conditions that may cause ST elevations, which must be kept in mind in the differential diagnosis, including Prinzmetal’s angina, acute pericarditis phase I (ST elevation in all leads), hyperkalemia, aortic dissection, left ventricular aneurysm, blunt chest trauma, Brugada syndrome, hypothermia, and J point elevation.

Our patient presented with pure chest pain, and the ECG pointed to an inferior ST elevation myocardial infarction. ST elevation in the inferior leads is an atypical finding in PE. The lack of a filling defect in the coronary arteries rules out paradoxical embolism. Because we had previously encountered a number of cases of PE who were shown to have ST elevations, we decided to proceed with right heart catheterization and pulmonary angiography. According to one scenario, if this patient had been given thrombolytic therapy, the thrombus -we would never know where- would probably dissolve, the patient would be evaluated as ‘recanalized’, and the angiogram would later label this patient as having ‘myocardial infarction with normal coronary arteries’!

In conclusion, acute PE should be suspected in every patient with ST elevation myocardial infarction and normal coronary arteries. ST changes may be in the inferior as well as the anterior leads.

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*Supplementary video files associated with this article can be found in the online version of the journal.

REFERENCES


Key words: Myocardial infarction; pulmonary embolism; right bundle-branch block; ST segment elevation.

Anahtar sözcükler: Miyokart enfarktüsü; pulmoner emboli; sağ dal bloku; ST segment yükselmesi.