Acute pulmonary embolism mimicking inferior myocardial infarction

İnferiyor miyokart enfaktüsünü taklit eden akut pulmoner emboli

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Summary– Pulmonary embolism (PE) is a potentially life-threatening emergency that is sometimes difficult to diagnose due to nonspecific symptoms and findings. A 69–year-old male was admitted to our hospital with new-onset chest pain and sweating. The electrocardiogram (ECG) revealed sinus rhythm with ST elevations in the inferior leads. His angiogram showed noncritical coronary artery disease with a few plaques. Right heart catheterization was made, which revealed an elevated pulmonary artery pressure of 45/23 mmHg. A pulmonary angiogram was then performed, at first from the pulmonary trunk and then the right pulmonary artery, which showed occlusion of the pulmonary artery to the right lower lobe. This report emphasizes that 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.

Özet– Pulmoner emboli hayatı tehdit eden acil durumlardan biri olup, nonspesifik semptom ve bulgularından ötürü zor tanı konmaktadır. Altmış dokuz yaşında erkek hasta hastanemize yeni başlayan göğüs ağrısı ve terleme şikayeti ile başvurdu. EKG'de sinüs ritmi ve inferiyor derivasyonlarda ST segment yükselmesi saptandı. Anjiyografisinde plaklarla beraber kritik olmayan koroner arter hastalığı gözlendi. Sağ kalp kataterizasyonu uygulandı. Pulmoner arter basıncı 45/23 mmHg ölçüldü. Bundan sonra pulmoner anjiyogram uygulandı ve sağ akciğer alt lobunu besleyen pulmoner arter dalında tıkanıklık gözlendi. Bu olgu sunumunda, anteriyor derivasyonlarda olduğu gibi inferiyor derivasyonlarda ST elevasyonuyla başvuran ve normal koroner arter tespit edilen her hastada da pulmoner emboliden şüphelenilmesi gereği vurgulanmak istenmiştir.

Pulmonary embolism (PE) is a potentially life-threatening emergency that is sometimes difficult to diagnose due to nonspecific symptoms and findings.[1] The electrocardiogram (ECG) may show a wide spectrum of findings. A remarkable number of cases of PE with ST elevation in anterior leads have been reported in the literature.[2,3] However, PE with ST elevation in the inferior leads on ECG is very rare.

Abbreviations:

ECG	*Electrocardiogram*
PE	*Pulmonary embolism*
RBBB	*Right bundle branch block*

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 $SatO_2$ 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. As the ST elevations persisted with ongoing symptoms despite the use of sublingual nitrate in the ambulance, he was rushed directly to the catheter laboratory for primary percutaneous coronary intervention. Coronary angiography showed a

Received: August 18, 2013 *Accepted:* November 01, 2013

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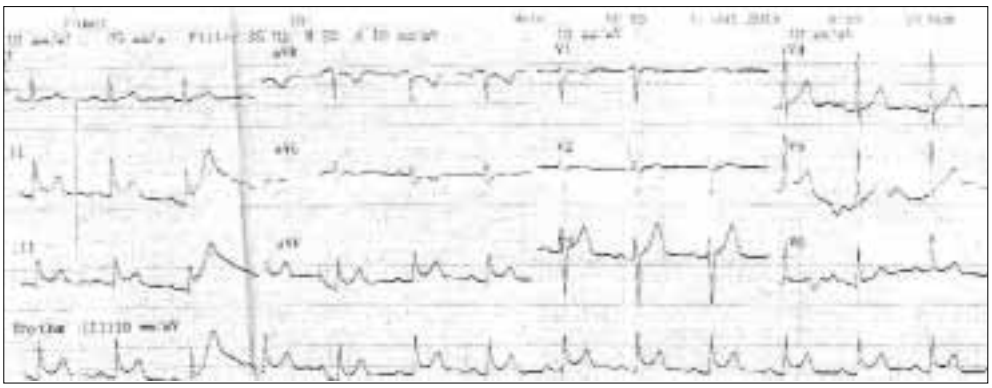

Figure 1. ECG revealed sinus rhythm with ST elevations in the inferior leads.

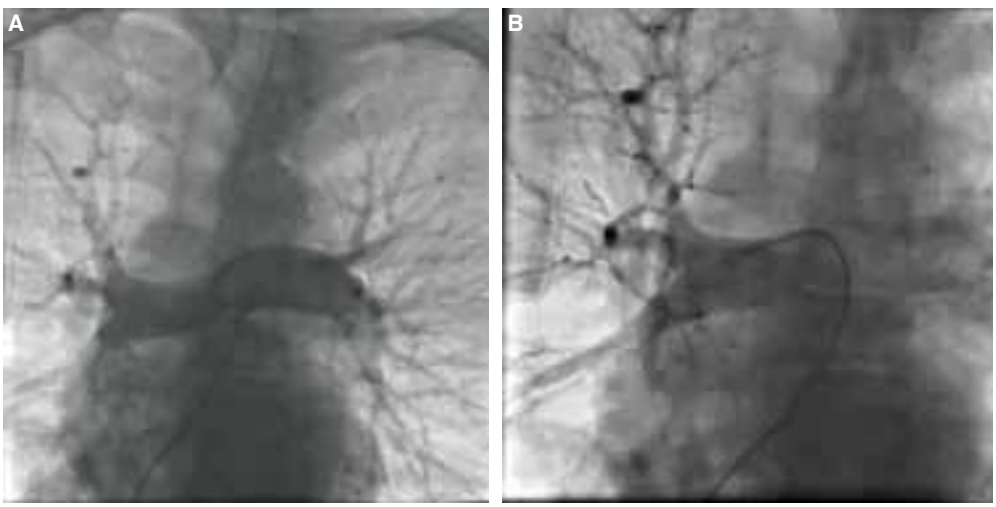

Figure 2. **(A)** Occlusion of the pulmonary artery to the right lower lobe. **(B)** Partial recanalization of the distal right pulmonary artery after selective injection.

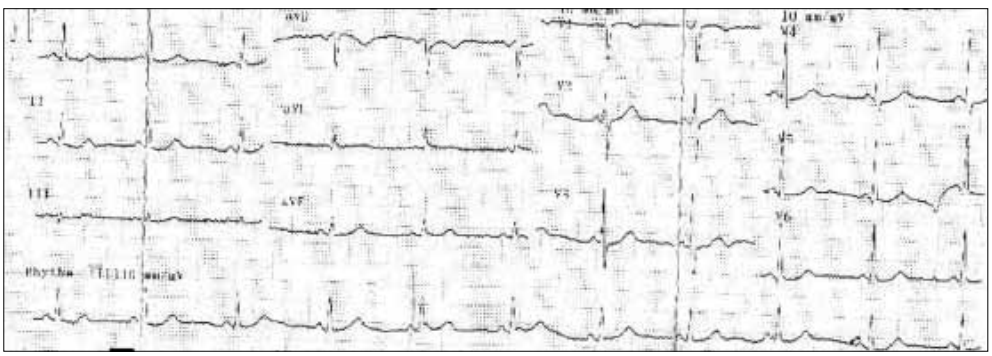

Figure 3. Electrocardiogram reverted to normal after initial therapy.

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.[1] More than 90% of cases present shortness of breath and chest pain.[4] Chest pain is usually pleuritic, which is due to pulmonary infarction.[5] Chest pain is also related with myocardial ischemia due to the increased right ventricular afterload and a disproportion between oxygen demand and supply.[3]

The ECG is variable in acute PE, and sinus tachycardia is the most common. Choue and coworkers[6] 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.[7] reported that three or more of the following findings support the diagnosis of PE: incomplete or complete RBBB, deep S waves in DI 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.[2,3,8-10]

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.[9] 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.[11]

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.

Conflict-of-interest issues regarding the authorship or article: None declared.

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

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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.