Pulmonary Embolism Presenting with Evolving Electrocardiographic Abnormalities Mimicking Anteroseptal Myocardial Infarction: A Case Report

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Introduction

The diagnosis of pulmonary thromboembolism (PTE) with changes shown by electrocardiography (ECG) is a challenge in the clinical practice due to rare pathognomonic findings. Although several ECG changes can be observed in the acute phase of PTE, ST segment elevation is a rare occasion [1–3]. This report describes a woman who showed dynamic ST segment elevation suggestive of anteroseptal acute myocardial infarction (AMI).

Case Report

A 50-year-old woman was admitted to the emergency department with shortness of breath and epigastric pain radiating to the back and syncopal attack while waiting in the hospital for a control visit. She recovered spontaneously and felt epigastric pain thereafter. She had been injured after a traffic accident 2 weeks previously with fractures of the right scapula and 4th–9th ribs and had been discharged from hospital without surgical intervention. The patient’s medical history was significant for diabetes mellitus and hypertension of 10 years. Her prescribed medications included insulin and ramipril. The family history was unremarkable. On admission her pulse rate was regular (100/min), blood pressure was 100/65 mm Hg and tachypneic with a respiratory rate of 24 breaths/min. Physical examination revealed rales...
on the right lower lobe of lung and a 2/6 systolic murmur in the mesocardi...
in PTE and suggested that the diagnosis of PTE could be made on the basis of several ECG abnormalities including an S_1 Q_3 T_3 pattern, qr pattern in V_1, rSr' pattern in leads V_1–2, rS pattern in leads V_3–5, T wave inversion in leads V_1–5, ST segment elevation in leads V_1–3 and ST segment depression in lead D1 in the majority of conditions. A pseudoinfarction pattern occurs in 3–11% with acute PTE [5, 7]. Also Lynch et al. [8] analyzed 115 patients with acute PTE and concluded that left axis deviation (<30°) at the time of acute PTE was twice as frequent as right axis deviation. Other reported findings were right axis deviation, leftward displacement of the transitional zone, p pulmonale and atrial dysrhythmias including atrial fibrillation, atrial flutter, atrial tachycardia and premature atrial beats [5]. Additionally, ECG findings in the rightsided precordial leads in acute PTE have been rarely documented previously. Slurring of the ascending limb of the S wave in leads V_4 R and V_1–2 was shown in a previous report [9]. Chia et al. [10] described ECG findings of ST segment elevation and a qs or qr pattern in 3 of 4 patients with PTE in leads V_4–6 R similar to the findings of Akula et al. [11] in which the ST segment elevation was greater than 1 mm and depths of the q waves in leads V_4–6 R were 1.5–4 mm; those abnormalities were mostly normalized within 6 weeks due to the transient nature of ECG abnormalities. Tan et al. [12] and Andersen et al. [13] found that ST segment elevation was smaller than 0.5 mm and q wave depth varied between 0.5 and 1.5 mm in normal subjects.

Although ST segment elevation with the complaint of chest pain made us to focus on acute coronary syndrome initially, the diagnosis of acute PTE was also considered because besides the recent history of trauma and reduced mobility, several ECG abnormalities are also compatible
with PTE. A new-onset RBBB that had not been present 2 weeks previously and the initial ECG showing sinus tachycardia are the most common findings in PTE, but can also be seen in acute coronary syndrome; right precordial leads revealed ST segment elevations (2 mm) and q waves (2 mm) in V₄₋₆R, an abnormal finding reported in PTE, previously. Third, atrial tachycardia evolved during the course, an atrial rhythm abnormality seen in PTE. Fourth, after unfractionated heparin infusion, synchronous with symptomatic recovery, ST segment depression and recovery of sinus rhythm were observed. Fifth, ST segment depression in V₁₋₃ was seen in ECG with the resolution of RBBB before the discharge.

Due to the presence of atypical ECG changes for acute PTE in our patient, AMI was considered initially in the differential diagnosis, and a coronary angiogram was performed before other noninvasive tests. Other evaluations like echocardiography could be helpful in this case. Transthoracic echocardiography is a readily available bedside test that can be performed in the emergency department on admission and is helpful to differentiate massive PTE and anteroseptal AMI, but rarely provides direct visualization of PTE. The echocardiography was performed on our patient after a coronary angiogram that was consistent with massive PTE. If echocardiography had been performed immediately, coronary angiography could have been avoided in our patient.

**Conclusion**

This case emphasized the rare association of PTE with ST segment elevation, especially in the anteroseptal derivations. This case also showed the usefulness of the echocardiogram in differentiating AMI from massive PTE. Therefore, we recommend that physicians should remember the presence of PTE in patients with chest pain or dyspnea, even when ECG points to AMI.