A Case of Massive Pulmonary Embolism With ST Elevation in Leads V1-4

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A 35-year-old man was referred to the emergency department after having a short syncopal episode while waiting for a Doppler scan of the lower extremities for a 4-week history of a painful right leg. He had no significant past medical history and was a non-smoker. On presentation he had severe chest pain and dyspnea associated with diaphoresis, and was hemodynamically unstable. His initial electrocardiogram (ECG) showed ST segment elevations in leads V1-4, mimicking an anteroseptal myocardial infarction. However, the angiography showed the coronary arteries were normal and the right main pulmonary artery was partially occluded by large pulmonary emboli. The ECG changes were recorded in detail which also pointed to the diagnosis of pulmonary embolism (PE). This case shows how a PE can mimic an anteroseptal myocardial infarction on ECG, and the physiopathology of the ST elevation in PE was discussed. (Circ J 2009; 73: 1157–1159)

Key Words: Electrocardiogram; Myocardial infarction; Precordial ST segment elevation; Pulmonary embolism

Pulmonary embolism (PE) can be difficult to diagnose on clinical grounds as it often presents with nonspecific symptoms! Clinical instability can delay or prevent the execution of an effective diagnostic strategy, with resulting delay in providing appropriate therapy. Prompt diagnosis of PE heavily relies on clinical suspicion. The electrocardiogram (ECG), one of the first examinations to be performed in cases of suspected PE, has been widely studied. ECG abnormalities can be seen in 70–80% of patients, varying from the typical S1Q3T3 pattern to no specific changes. However, only very rarely does a PE produce an ECG pattern mimicking acute myocardial infarction (AMI). We describe such a case in the following report. Furthermore, the angiography showed the coronary arteries were normal and the right main pulmonary artery was partially occluded by large pulmonary emboli, despite the acute phase ECG pattern suggestive of AMI, and we recorded in detail the ECG changes which also pointed to the diagnosis of PE. It shows the importance of recognition of various ECG changes in patients with PE.

Case Report

A 35-year-old Chinese man was admitted to the hospital because of a 4-week history of a painful right leg. The patient had no significant past medical history and took no medications. The family history was unremarkable for any acute or chronic medical problems. He had no drug allergies and no history of past surgeries, smoking and drinking. He was referred to the radiology department for a Doppler scan of lower extremities. While waiting for the examination, he suddenly collapsed while standing and lost consciousness for approximately 3 min. He recovered spontaneously and had severe chest pain and dyspnea associated with diaphoresis. The patient was then transferred to the cardiology emergency department.

In the emergency department, his blood pressure was 72/43 mmHg, heart rate was regular at 80 beats/min and his respiratory rate was 23 breaths/min. An arterial blood gas test showed an arterial oxygen pressure of 71 mmHg (oxygen saturation was 90%) and a carbon dioxide pressure of 36 mmHg while the patient was breathing 6L of oxygen by face mask. Physical examination revealed a well developed, well nourished man, who knew his identity, whereabouts and the date, and focal neurologic findings were not present when examined. The chest was clear to auscultation and percussion bilaterally. Findings of heart and abdominal examinations were unremarkable, and lower extremities were without edema.

Initial investigations revealed that concentrations of complete blood cell count, serum electrolytes, glucose, blood urea and creatinine were normal. The initial ECG (Figure 1A) showed a normal sinus rate of 80 beats/min with incomplete right bundle branch block (RBBB), ST elevations in leads V1-4, s waves in leads I and V5, q wave in lead III. An initial diagnosis of acute anterior myocardial infarction was made. The inotropic agent dopamine was initiated to maintain adequate tissue perfusion, and the patient was administered aspirin and clopidogrel, and low-molecular-weight heparin. The thrombolytics were not administered initially because the patient’s family could not agree with the treatment. However, there was no previous history of angina, and he had no coronary risk factors, such as smoking, hypertension, diabetes mellitus, or hyperlipidemia. After 1 h, the ECG (Figure 1B) showed the ST elevations in V1 and V3,4 back to normal except lead V2, s waves in leads I and V5, q wave in lead III, and progressive deep T waves inversion in leads III, V1,2. A PE was suspected.
A transthoracic echocardiogram done in the emergence department showed a normal left ventricle function, a distended right ventricle with free wall hypokinesia and mild pulmonary hypertension (40 mmHg). A Doppler scan of the lower extremities showed deep venous thrombosis in the patient’s right lower extremity (popliteal vein). One million U of urokinase was delivered in 1 h to a peripheral vein. The patient was hemodynamically stable 12 h after the urokinase infusion.

Serial cardiac enzymes were within reference range. Over the following 42 h serial ECG (Figures 1A–D) recordings showed progressive deep T waves inversion in leads III and V1–4. Coronary angiography showed the coronary arteries were normal. The pulmonary angiography revealed the right main pulmonary artery was partially occluded by large pulmonary emboli (Figure 2).

An intravenous filter was placed in the inferior vena cava (IVC filter) to prevent further pulmonary thromboemboli. The patient remained well, taking lifelong warfarin. The ECG (Figure 1E) after 3 months was basically normal except low QRS voltage in lead III.

Discussion

PE, defined as ‘the implantation of material into branches of the pulmonary arterial bed’, usually consists of clots dislocated from peripheral veins. However, they might also consist of neoplastic cells, fat emboli, amniotic fluid, air bubbles, and other exogenous materials, such as talc, cornstarch particles, or pieces of catheters. Most commonly the clots migrate from deep veins of the pelvis and the lower extremities. The formation of venous thrombosis is usually due to one or more of the following factors: endothelial injury, hypercoagulability, or stasis of blood.

The diagnosis of PE is often difficult to establish. PE differ considerably in size and number, and the underlying disorders (eg, underlying malignancy, trauma, hypercoagulable state resulting from protein C or S deficiency, antithrombin III deficiency, and obesity) are numerous. Thus, the clinical picture of PE is variable, which accounts for the frequent failure to recognize its presentation. Indeed, most emboli are not recognizable on clinical grounds because they are too small to produce cardiorespiratory symptoms and the lung is devoid of pain fibers! Because only the parietal pleura has pain fibers, typical pleuritic pain occurs only when an embolus is complicated by pulmonary infarction! It therefore follows that most instances of PE are clinically silent. However, massive PE is life-threatening if left untreated, with rapid progression and deterioration. Massive PE consists of shock or hypotension (defined as systolic blood pressure, 90 mmHg or a pressure drop of >40 mmHg.
for 15 min if not caused by new onset arrhythmia, hypovolaemia, or sepsis). The increased right ventricular afterload leads to increased right ventricular myocardial work and oxygen consumption. The combination of hypoperfusion and increased demand on the right ventricle leads to severe ischemia and dysfunction, which has been suggested as the important factor causing the electrocardiographic changes of PE.

Electrocardiographic changes associated with PE were first described by McGinn and White in 1935. They described the presence of the classic S1Q3T3 pattern in 7 patients with acute cor pulmonale. Since then, several ECG abnormalities have been associated with the diagnosis of PE. The 12-lead ECG has been studied to assess for potential use in the diagnosis of PE and in determining the severity and prognosis of the disease. Chou suggested that typical EKG findings in PE are as follows: (a) an S1Q3 or S1Q3T3 pattern; (b) rightward shift of the QRS axis; (c) transient, incomplete or complete RBBB; and (d) T wave inversion in the right precordial leads (2). Sinus tachycardia is the most common EKG abnormality of PE. Other more rare findings are: (a) displacement of the transitional zone to the left; (b) left axis; (c) QR pattern in V1; (d) R/S in V1>1 mm; (e) ‘staircase’ ascent of ST segments in lead I or II; (f) ST elevation in lead III; (g) ST depression or elevation in right precordial leads; (h) ST and T-wave changes in the left precordial leads; (i) a P pulmonale pattern; (j) atrial dysrhythmias, including atrial flutter, atrial fibrillation, atrial tachycardia, and atrial premature contractions; and (k) first degree AV block. Sreeram et al found that RBBB, S waves in leads I and aV1, Q waves in leads III and aVr, right axis deviation or indeterminate axis, low QRS voltage in frontal plane, and T wave inversions were associated with PE in a study performed in 49 consecutive patients. However, Rodger et al studied the ECG of 246 patients (49 cases and 163 controls) and found that only sinus tachycardia and incomplete RBBB were significantly more common in confirmed PE patients vs controls.

In our case we recorded in detail the ECG changes. The initial ECG showed incomplete RBBB, s waves in leads I and V5–6, q wave in lead III, ST elevations in leads V1–4. The ST segment elevation and the evolution to ST regress and negative T-waves in leads V1–4 mimicking an anteroseptal myocardial infarction in our patient made proper recognition very difficult. However, scrutinous interpretation could have led to earlier diagnosis because some electrocardiographic findings were not typical for an evolving myocardial infarction and hinted to PE: (a) The most characteristic change in the QRS in myocardial infarction that develops initially presenting with ST elevation is the evolution of Q waves in the leads overlying the infarct zone – leading to the term Q-wave infarction. However, the evolution of Q waves in leads V1–4 with ST elevations did not appear in our patient. (b) The initial ECG had no reciprocal changes in lead inferior leads. (c) Our patient had many of the typical electrocardiographic findings associated with PE described by Chou. Specifically, our patient had an S1Q3T3 pattern, an incomplete RBBB, and T wave inversion in leads V1–3. All these findings, combined with the clinical suspicion of PE in the patient with painful right lower extremity and without any coronary risk factors, could have led to an earlier diagnosis of PE, and earlier start of anticoagulant treatment.

This present case shows how a PE can mimic an anteroseptal myocardial infarction on ECG and present a diagnostic challenge for the clinician. The reasons for the ECG changes including ST segment elevations and inverted T waves in the precordial leads are unclear. The electrocardiographic findings in our patient might be the combined result of hemodynamic, anatomic, metabolic and autonomic effects of acute PE by itself. First, right ventricular dilatation has been suggested as one of the major factors causing the electrocardiographic changes of PE. Acute stretching of right bundle branch when the heart abruptly dilates, is reflected in the ventricular conduction delay with appearance of incomplete or complete RBBB as in our patient. Second, the hypoxaemia secondary to PE and right ventricular overload might induce right ventricular myocardial ischemia and hence mechanical dysfunction. Third, elevated right heart pressures with concurrent right ventricular dilatation will summate, by means of the laplace law, to markedly increase wall tension. This means more right ventricular work and oxygen consumption. Fourth, at some extent of vascular obstruction, the right ventricle will be unable to generate a systolic pressure high enough to preserve pulmonary perfusion and will ultimately fail. This results in turn in an acute reduction of left ventricular preload and acute lowering of the cardiac output. The right ventricular hypertension together with a dilated right ventricle and leftward shift of the interventricular septum further add to the diminished left ventricular preload and resultant decreased cardiac output. This results in a decrease in the aortic root pressure and coronary flow. Its combination with other reasons might even induce right ventricular infarction. All the reasons could explain the ST elevation in the right precordial leads in our case. No overall conclusion has been reached as yet.

In conclusion, this case shows how PE can mimic anteroseptal AMI on ECG. In PE the pattern of ST elevation is extremely rare, and we recorded in detail the ECG changes which can suggest acute cardiac injury.

References