Case Report

Acute Pulmonary Embolism Masquerading as Anteroseptal Myocardial Infarction

Worawut Roongsangmanoon MD*, Arthit Wongsoasup MD*,
Nattapun Rattanajaruskul MD*, Theeranan Angkananard MD*

** Department of Medicine, Srinakharinwirot University, Ongkharak, Nakhon Nayok, Thailand*

Pulmonary embolism (PE) is a potentially lethal condition. Prompt diagnosis of PE relies on high index of clinical suspicion. Despite multiple electrocardiographic findings described in association with PE, ST-segment elevation remains rare and represents a diagnostic challenge to differentiate it from acute myocardial infarction. The authors present a case of PE mimicking anteroseptal infarction on electrocardiography (ECG) which was successfully thrombolysed with streptokinase.

Keywords: *Pulmonary embolism, Electrocardiography, Myocardial infarction*

J Med Assoc Thai 2017; 100 (Suppl. 8): S221-S225
Full text. e-Journal: *http://www.jmatonline.com*

The ECG is often an initial diagnostic test for a patient who has a suspicion of having PE. As various electrocardiographic changes have been noted in the PE patient, it is important to recognize and understand the abnormalities. Electrocardiographic manifestations of PE are variable and range from the classic $S_1Q_3T_3$ pattern to such non-specific changes as sinus tachycardia. PE is an uncommon and often overlooked cause of ST-segment elevation on the ECG. The authors describe such a case in the following report. The objectives of this case report include a review of the literature pertaining to the various electrocardiographic manifestations of an acute PE, a demonstration of how acute PE may cause dramatic and uncharacteristic changes on the ECG, and a recognition of bedside echocardiography aids in distinguishing between the diagnosis of an acute PE and a myocardial infarction.

Case Report

A 60-year-old man presented with two episodes of syncope. He developed mild chest discomfort. He had no significant past medical history and was a non-smoker. The family history was unremarkable for sudden cardiac death. His blood pressure was 91/75 mmHg, his heart rate was regular at 76 beats per minute and his respiratory rate was 20 breaths per minutes. The oxygen saturation was 99% while the patient was breathing 10 liters of oxygen by face mask. The chest and cardiac examination were unremarkable. The focal neurological findings were not present and lower extremities were without edema. Electrocardiography (Fig. 1) showed normal sinus rhythm, right bundle branch block (RBBB) and ST-segment elevation in V_1 to V_3, S waves in leads I, V_5, V_6, Q wave in lead III. An initial diagnosis of acute anterior myocardial infarction was made. He was transferred to the catheterization laboratory. Coronary angiography demonstrated no significant stenosis. The patient developed marked hypoxemia, oxygen saturation fell to 91%. Echocardiography revealed a normally contracting left ventricle, a right ventricular dilatation with free wall hypokinesia and hypercontractility of the apical wall, a displacement of the interventricular

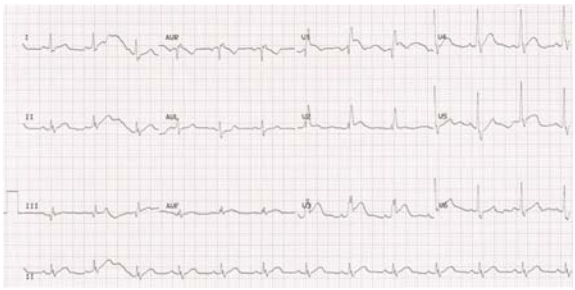

Fig. 1 ECG showed normal sinus rhythm, right bundle branch block (RBBB) and ST-segment elevation in V_1 to V_3, S waves in leads I, V_5, V_6, Q wave in lead III.

Correspondence to:
Roongsangmanoon W, Department of Medicine, Srinakharinwirot University, Ongkharak, Nakhon Nayok 26120, Thailand.
Phone: +66-37-395085 ext. 11001
E-mail: jrworawut@yahoo.co.th

septum towards the left ventricle (Fig. 2) and tricuspid insufficiency that did not permit quantification of pulmonary artery pressure. Computed tomography of pulmonary arteries revealed that the main pulmonary arteries of the right and left lung were partially occluded by large emboli (Fig. 3). The patient's complete blood cell count and hematological investigation of thrombophilia were normal. The initial troponin was negative.

The patient received thrombolytic treatment with a bolus of streptokinase 250,000 units and then as a continuous infusion of 100,000 units per hour for 24 hours. Chest discomfort improved dramatically.

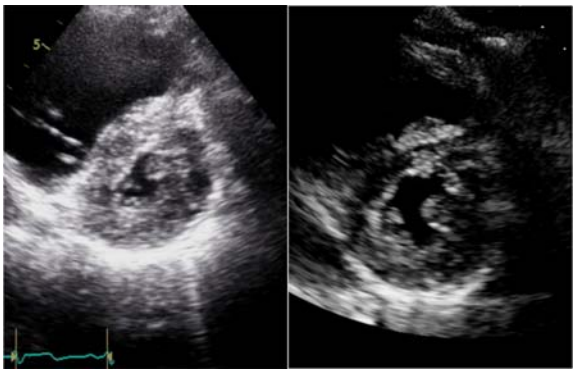

Fig. 2 Echocardiography (short-axis view) showed a right ventricular dilatation with a displacement of the interventricular septum towards the left ventricle (left). A less distended right ventricle with normal contraction was present after two weeks of treatment (right).

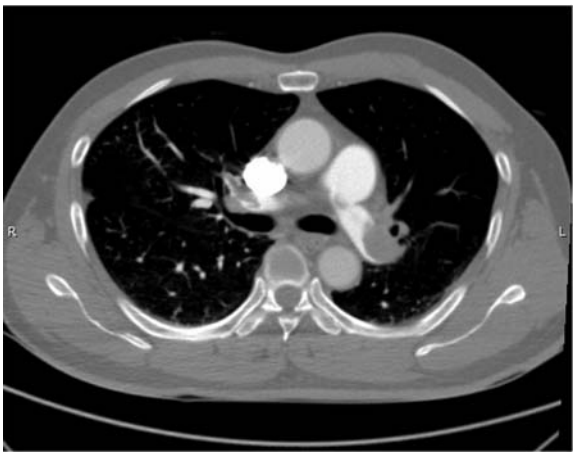

Fig. 3 Computed tomography of pulmonary arteries; the main pulmonary arteries of the right and left lung were partially occluded by large emboli.

The oxygen flow was reduced gradually during hospitalization according to oxygen saturation. The heart rate was reduced to around 60 beats per minute. Enoxaparin and warfarin were administered. The patient was discharged after 10 days of hospitalization in good condition. A less distended right ventricle with normal contraction was present after two weeks of treatment (Fig. 2).

Discussion

The clinical picture of PE is variable, which accounts for the frequent failure to recognize its presentation. Indeed, most of emboli are missed on clinical grounds because of non-specific symptoms. The ECG, one of the first examinations to be performed in cases of suspected PE, has relatively low sensitivity. An entirely normal ECG has been found in approximately 10% to 25% of the patients with PE[1,2]. The electrocardiographic changes associated with PE were first described in 1935 by McGinn and White who noted the traditional $S_1Q_3T_3$ pattern in acute corpulmonale[3]. Several ECG abnormalities have been associated with the diagnosis of PE, including arrhythmias (sinus tachycardia, atrial flutter, atrial fibrillation, atrial tachycardia, and atrial premature contractions), rightward QRS complex axis shift and other axis changes, P-pulmonale, S_1Q_3 or $S_1Q_3T_3$ pattern, transient RBBB, R >5 mm in V_1 or R/S in V_1 >1, "staircase" ascent of ST-segment in lead I or II, ST-segment elevation in lead III, ST-segment depression or elevation in right precordial leads, nonspecific ST-segment or T wave changes, and T wave inversions in the right precordial leads[4,5].

Sinus tachycardia is the most frequent rhythm and abnormality encountered on presentation[3,6]. Sinus tachycardia in the face of PE is likely related to the severity of hypoxemia or the physiologic demand to increase cardiac output. As left-sided stroke volume decreases, heart rate must increase to maintain cardiac output. Atrial arrhythmias are also seen in the patient with acute PE, these disturbances likely result from high atrial pressure and hypoxemia.

The classic $S_1Q_3T_3$ pattern, mistakenly considered pathognomonic for acute PE, is seen approximately 15% to 25% of patients diagnosed with PE[1]. This pattern is characterized by an S wave in lead I, a Q wave in lead III, and shallow T wave inversions in one or more of the inferior leads. In addition, the ST segments may be slightly elevated in the inferior leads. Although this finding is consistent with right-sided cardiac changes, it remains unclear if this finding

actually predicts PE. This finding should not be used as the sole criterion, particularly in patients lacking the clinical suspicious of PE. The $S_1Q_3T_3$ pattern usually resolves within 2 weeks.

RBBB is found approximately 25% of the patients with PE. The PE-related RBBB pattern is transient, often resolving with the restoration of normal right-sided cardiac hemodynamics. However, it may be resolved in 3 months to 3 years after the index PE[1]. RBBB may also be associated with ST-segment elevation and prominent, upright T waves in the right precordial leads (V_1 to V_2), potentially mimicking anterior infarct pattern[7].

The ST-segment may be either depressed or elevated in the patient with PE. Minimal ST-segment depression is a common finding on the ECG in such patients. The $S_1Q_3T_3$ pattern may be associated with ST-segment elevation in the inferior leads. The RBBB pattern may present with ST-segment elevation in the right precordial leads. Significant ST-segment elevation consistent with acute myocardial infarction is quite rare. The presence of anterior ischemia caused by PE manifests as T wave inversion in the right to mid precordial leads (V_1 to V_4)[8]. In a study of 80 patients hospitalized for PE, the authors concluded that the severity of the PE correlated with T-wave inversion in the precordial leads, being present in 85% of their patients with massive PE[8].

Sreeram and colleagues reviewed the ECG of 49 patients with proven acute PE. They concluded that PE should be considered when three or more of the following ECG changes occur; incomplete or complete RBBB, S waves in leads I and aVL of more than 1.5 mm, a shift in the transition zone in the precordial leads to V5, Q waves in leads III and aVF (not lead II), right-axis deviation or an indeterminate axis, a low voltage QRS complex in limb leads, T wave inversion in leads III and aVF or leads V1 to V4. Regarding this criteria, they only had a 26.7% sensitivity and 57.1% positive predictive value for PE[7].

The current case demonstrates how a PE can mimic an anteroseptal myocardial infarction on ECG and present a diagnostic challenge for the physician. The patient lacked any risk factors for venous thromboembolism. He had no tachycardia. The use of supplemental oxygen obscured hypoxemia on presentation. The ST-segment elevation in the precordial leads is a very rare ECG manifestation in the patients with PE and only few cases has been reported[9-11]. It is very difficult to differentiate from myocardial infarction. The RBBB is consistent with the findings of right ventricular strain or the apparent mimicking of an anteroseptal myocardial infarction. However, the initial ECG had no reciprocal changes in the inferior leads which were not typical for infarction. After the unremarkable coronary angiography, the patient had worsening hypoxemia with normal lung auscultation. The use of bedside echocardiography proved to be a very helpful tool in distinguishing between the diagnosis of a myocardial infarction and a PE in these settings.

The reasons for the ECG changes mimicking an anteroseptal infarction are unclear[12]. They may be the combined result of hemodynamic, anatomic, and autonomic effects of acute PE[13]. Acute stretching of right bundle branch when the right ventricle abruptly dilates, results in the conduction delay with appearance of RBBB as in the current patient. Right ventricular dilatation with concurrent acute increase of afterload causes markedly increased wall tension hence the possibility of right ventricular ischemia is increased[7]. The more right ventricular work and oxygen consumption result in myocardial ischemia and systolic dysfunction. The right ventricle fails to generate adequate output which ultimately produces an acute reduction of left ventricular preload and cardiac output. Moreover, a sudden right ventricular hypertension together with a dilated right ventricle and leftward displacement of the interventricular septum further diminishes left ventricular preload leading to decrease of cardiac output[14]. The reduced cardiac output compromises both systemic and coronary perfusion. The hypoxemia accompanying PE induces a catecholamine surge and further increases myocardial workload, worsening the ischemia. ECG abnormalities may be attributable to catecholamine- and histamine-mediated ischemia at the cellular level, or coronary vasospasm secondary to hypoxemia[10,13]. Paradoxical coronary embolism secondary to PE is another possible mechanism[11]. However, there was no evidence of coronary obstruction in the current patient.

Conclusion

Acute PE can mimic anteroseptal myocardial infarction on ECG. The physician must view the ECG and clinical evaluation as a whole. Combinations of various electrocardiographic abnormalities and their diagnostic value have been considered. The physician will likely miss the diagnosis if he or she relies on the ECG as the sole screening tool. Echocardiography has great value for the rapid identification of RV overload and should be used in such a confusing case to make

the correct diagnosis and enable appropriate therapeutic intervention.

What is already known on this topic?

The clinical picture and ECG manifestation of PE are variable. The ECG has relatively low sensitivity and specificity for diagnosis of PE.

What this study adds?

The physician must view the ECG combining with thorough clinical evaluation. Acute PE can mimic anteroseptal myocardial infarction on ECG. The absence of reciprocal ST-segment depression indicates atypical for myocardial infarction. Several mechanisms have been proposed for ST-segment elevation in the patient with PE. Echocardiography has great value for the rapid differentiation between acute PE and myocardial infarction.

Potential conflicts of interest

None.

References

1. Panos RJ, Barish RA, Whye DW Jr, Groleau G. The electrocardiographic manifestations of pulmonary embolism. J Emerg Med 1988; 6: 301-7.
2. Hubloue I, Schoors D, Diltoer M, Van Tussenbroek F, de Wilde P. Early electrocardiographic signs in acute massive pulmonary embolism. Eur J Emerg Med 1996; 3: 199-204.
3. McGinn S, White PD. Acute corpulmonale resulting from pulmonary embolism. J Am Med Assoc 1935; 104: 1473-80.
4. Goldberger AL. Electrocardiography. In: Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J, editors. Harrison's principles of internal medicine. 19th ed. New York: McGraw-Hill; 2015: 1454.
5. Chou T. Pulmonary embolism. In: Electrocardiography in clinical practice. 2nd ed. Orlando, FL: Grune Stratton; 1986: 309-17.
6. Petruzzelli S, Palla A, Pieraccini F, Donnamaria V, Giuntini C. Routine electrocardiography in screening for pulmonary embolism. Respiration 1986; 50: 233-43.
7. Sreeram N, Cheriex EC, Smeets JL, Gorgels AP, Wellens HJ. Value of the 12-lead electrocardiogram at hospital admission in the diagnosis of pulmonary embolism. Am J Cardiol 1994; 73: 298-303.
8. Ferrari E, Imbert A, Chevalier T, Mihoubi A, Morand P, Baudouy M. The ECG in pulmonary embolism. Predictive value of negative T waves in precordial leads-80 case reports. Chest 1997; 111: 537-43.
9. Vranckx P, Ector H, Heidbuchel H. A case of extensive pulmonary embolism presenting as an acute myocardial infarction—notes on its possible pathophysiology. Eur J Emerg Med 1998; 5: 253-8.
10. Falterman TJ, Martinez JA, Daberkow D, Weiss LD. Pulmonary embolism with ST segment elevation in leads V1 to V4: case report and review of the literature regarding electrocardiographic changes in acute pulmonary embolism. J Emerg Med 2001; 21: 255-61.
11. Cheng TO. Mechanism of ST-elevation in acute pulmonary embolism. Int J Cardiol 2005; 103: 221-3.
12. Chou T, Knilans TK. Pulmonary embolism. In: Electrocardiography in clinical practice adult and pediatric. 4th ed. Philadelphia: Saunders; 1996: 167-70.
13. Spodick DH. Electrocardiographic responses to pulmonary embolism. Mechanisms and sources of variability. Am J Cardiol 1972; 30: 695-9.
14. Vlahakes GJ, Turley K, Hoffman JI. The pathophysiology of failure in acute right ventricular hypertension: hemodynamic and biochemical correlations. Circulation 1981; 63: 87-95.

โรคลิ่มเลือดอุดตันในหลอดเลือดแดงปอดที่ถูกวินิจฉัยเป็นหลอดเลือดหัวใจอุดตันเฉียบพลัน

วรวุฒิ รุ่งแสงมนูญ, อาทิตย์ วงษ์เสาวศุภ, ณัฐพันธ์ รัตนจรัสกุล, ธีรานันท์ อังคณานาฏ

โรคลิ่มเลือดอุดตันในหลอดเลือดแดงปอดนั้นมีอันตรายถึงชีวิต การวินิจฉัยควรเริ่มจากอาการและอาการแสดงที่สงสัย คลื่นไฟฟ้าหัวใจซึ่งเป็นการตรวจเบื้องต้นในโรคนี้เป็นไปได้หลายลักษณะ การยกตัวของส่วน ST พบได้ไม่บ่อย และทำให้ต้องวินิจฉัยแยกโรคกับหลอดเลือดหัวใจอุดตันเฉียบพลัน ผู้นิพนธ์นำเสนอผู้ป่วยโรคลิ่มเลือดอุดตันในหลอดเลือดแดงปอดที่คลื่นไฟฟ้าหัวใจมีลักษณะคล้ายกับโรคหลอดเลือดหัวใจอุดตันเฉียบพลันและได้รับการรักษาด้วยยาละลายลิ่มเลือด ต่อด้วยยาต้านการแข็งตัวของเลือดจนปลอดภัยกลับบ้านได้