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Case Report

Chest Pain Revealing Acute Coronary Syndrome Associated with Pulmonary Embolism

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Abstract

Acute pulmonary embolism (PE) is potentially a life threatening emergency that needs prompt management to reduce preventable deaths. Symptoms like dyspnea and chest pain often lack specificity and overlap with acute coronary syndrome (ACS). Importantly, electrocardiographic changes associated with PE are reported to be variable with some ECG patterns mimicking ACS, posing problems in the differential diagnosis. We report the case of an acute coronary syndrome associated with pulmonary embolism revealed by atypical chest pain. Acute pulmonary embolism was suspected and a computed tomography of the chest with angiography was performed immediately and showed massive bilateral thrombosis of the pulmonary artery.

Keywords: *Pulmonary Embolism; Thrombosis; Acute Coronary Syndrome*

Introduction

Acute pulmonary embolism (PE) is potentially a life threatening emergency that needs prompt management to reduce preventable deaths. Symptoms like dyspnea and chest pain often lack specificity and overlap with acute coronary syndrome (ACS). Importantly, electrocardiographic changes associated with PE are reported to be variable with some ECG patterns mimicking ACS, posing problems in the differential diagnosis.

Case Presentation

We report the case of an acute coronary syndrome associated with pulmonary embolism revealed by atypical chest pain. A 68-year-old man with a history of high blood pressure presented and type 2 diabetes, presented to the emergency department for progressive shortness of breath on exertion for the seven previous days associated with atypical chest pain that has been evolving for 12 hours before his admission. His physical examination was unremarkable except for crackles in the lower lung fields. Laboratory assessment revealed markedly elevated kinetics of ultrasensitive T-troponin level, raising from 0.07 ng/ml to 0. 09 ng/ml NV (< 0.014 ng/ml) within 3 hours. A 12-lead ECG showed sinus rhythm with precordial V1-4 diffuse T wave inversion and a QS aspect in the lower derivations. Based on the above results, the diagnosis of ACS without ST segment elevation complicated of heart failure was retained. A coronary angiogram was performed withing 13 hours of his presentation to the emergency department, showing significant stenosis of the middle segment of the left anterior descending coronary artery. An active dilatation of the stent was performed. The day following the angioplasty, the pain disappeared but the dyspnea worsened with oxygen saturation of 80% and persistence of sinus tachycardia. According to prediction

scores such as Wells or Geneva, the patient had a low to moderate clinical probability, his D-dimer dosage was 2.98 mg/L (normal < 0.5 mg/L). Acute pulmonary embolism was suspected and a computed tomography of the chest with angiography was performed immediately and showed massive bilateral thrombosis of the pulmonary artery. The patient's evolution was marked by refractory cardiogenic shock accompanied by multiorgan system failure and death.

Discussion

Pulmonary embolism is a frequent and potentially serious pathology, requiring urgent and adequate management. The diagnosis of pulmonary embolism is not always easy, despite the use of clinical probability scores, such as the Wells and Geneva score, allowing it to be classified as low, intermediate or low probability. strong and to guide further examinations thereafter. The chest CT angiography is the gold standard in the diagnosis of pulmonary embolism. Numerous studies have shown the safety of excluding PE on the basis of a negative result, in the event of a low clinical probability but positive D-dimers or in the event of a high clinical probability. In parallel with the improvement of nuclear medicine imaging technology, ventilation/perfusion (V/P) tomographic scintigraphy, currently allows the acquisition of three-dimensional images in a better resolution and visualization of perfusion defects of small sizes, which increases diagnostic performance compared to conventional (V/P) scintigraphy. Echocardiography is not recommended as a first-line diagnosis for PE because of its low sensitivity, which is around 60 to 70%. TTE finds its place in the diagnosis of severe forms, in particular when the patient is too unstable to perform CT angiography or there is a state of cardiogenic shock or signs of right heart failure. The PESI score (Pulmonary Embolism Severity Index and its simplified version (sPESI), allows low-risk patients to be identified with at least as much accuracy as the combination of ultrasound and laboratory parameters.

Clinical symptoms such as dyspnea, chest pain and faintness are not very specific, may also be reported in acute coronary syndromes, aortic dissection, pneumothorax and pericarditis; thus posing the problem of differential diagnosis. Elevation of biological markers such as troponin and pro BNP can be found both in pulmonary embolism and in coronary syndromes. D-dimers which are useful in the exclusion of pulmonary embolism, in cases of low or intermediate clinical probability according to the European recommendations for pulmonary embolism, may also be moderately elevated in coronary syndromes and aortic dissection. As for the electrical abnormalities accompanying pulmonary embolism, they have been well described for decades in the literature in the form of isolated cases or short series where the differential diagnosis of pulmonary embolism vs acute coronary artery disease has been raised. Their sensitivity and specificity are not cutting edge. The classic S1Q3T3 aspect, is the first sign that was described by Mc Ginn 1935, with high specificity but low sensitivity for the diagnosis of pulmonary embolism. Sinus tachycardia and incomplete right bundle branch block are most commonly seen in patients with pulmonary embolism, in the study by Rodger., *et al*. covering 246 cases. Another study of 190 patients conducted by Sukhia., *et al*. concluded that the combination of at least two of the following five criteria (S1, Q3, S1Q3, sinus tachycardia and supraventricular tachyarrhythmia) has a sensitivity of 78% and a specificity of 96% for the diagnosis of pulmonary embolism if the context clinical orientation. T-wave inversion has also been described in the anterior and inferior territory.

The modification of the ST segment in the acute phase of pulmonary embolism remains a rare sign, only a few cases have been described. In our case, the electrical modification involved the anteroseptal territory. The physiopathological explanation of this phenomenon is currently unknown, and hypotheses have been put forward in this direction: 1) mechanical ischemia (coronary compression by dilation of the right ventricle); 2) coronary spasm secondary to hypoxia; 3) paradoxical embolism at the coronary level; 4) acute decrease in left ventricular preload. Acute coronary syndrome is an emergency that is both diagnostic and therapeutic, management is a race against time "time is muscle", which is why drug treatment, often associated with interventional treatment, is quickly initiated. This treatment comprising among other things a curative dose of anticoagulants constitutes in principle the basis of treatment of non-severe pulmonary embolism. Our case, which joins the cases described in the world literature, confirms that pulmonary embolism can indeed be the great simulator of thoracic pathology, the presence of a modification of the ST segment has directed us to wrong towards acute coronary artery disease [1-18].

Conclusion

In conclusion, this case report underscores the difficulties in the differential diagnosis of these two diseases. It should bear in mind the limitation of a 12-lead ECG in differentiating PE from ACS. PE presenting with T-wave inversion can mimic ACS and lead to misdiagnosis.

Data Availability

No data were used to support this study.

Conflicts of Interest

No conflict of interest.

Funding Statement

This study did not receive any financial support.

Consent

Consent to publish this case was obtained from the patient next of kin.

Bibliography

1. Wells PS., *et al*. "Derivation of a simple clinical model to categorize patient's probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer". *Thrombosis and Haemostasis*83.3 (2000): 416-420.

2. Wicki J., *et al*. "Assessing clinical probability of pulmonary embolism in the emergency ward: a simple score". *Archives of Internal Medicine* 161.1 (2001): 92-97.

3. Van Belle A., *et al*. "Effectiveness of managing suspected pulmonary embolism using an algorithm combining clinical probability: D-dimer testing, and computed tomography". *The Journal of the American Medical Association*295.2 (2006): 172-179.

4. Righini M., *et al*. "Diagnosis of pulmonary embolism by multidetector CT alone or combined with venous ultrasonography of the leg: a randomised non-inferiority trial". *Lancet* 371.9621 (2008): 1343-1352.

5. Stein PD., *et al*. "SPECT in acute pulmonary embolism". *The Journal of Nuclear Medicine* 50.12 (2009): 1999-2007.

6. Torbicki A., *et al*. "The Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology In: guidelines on the diagnosis and management of acute pulmonary embolism". *European Heart Journal* 29.18 (2008): 22766-22315.

7. Aujesky D., *et al*. "Validation of a model to predict adverse outcomes in patients with pulmonary embolism". *European Heart Journal* 27.4 (2006): 47-81.

8. Fedullo PF and Tapson VF. "The evaluation of suspected pulmonary embolism". *The New England Journal of Medicine* 349.13 (2003): 1247-1256.

9. Konstantinides S. "Clinical practice: acute pulmonary embolism". *The New England Journal of Medicine* 359.26 (2008): 2804-2813.

10. Cavallazzi R., *et al*. "Natriuretic peptides in acute pulmonary embolism: a systematic review". *Intensive Care Medicine* 34.12 (2008): 2147-2156.

11. Konstantinides S., *et al*. "The task force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC)". *European Heart Journal* 35.43 (2014): 3033-3080.

12. Chan TC., *et al*. "Electrocardiographic manifestations: pulmonary embolism". *The Journal of Emergency Medicine* 21 (2001): 263-270.

13. McGinn S and White PD. "Acute cor pulmonale resulting from pulmonary embolism". *The Journal of the American Medical Association*104 (1935): 1473-1480.

14. Rodger M., *et al*. "Diagnostic value of the electrocardiogram in suspected pulmonary embolism". *The American Journal of Cardiology*86.7 (2000): 807-809.

15. Sukhja R., *et al*. "Electrocardiographic abnormalities in patients with right ventricular dilatation due to acute pulmonary embolism". *Cardiology* 105.1 (2006): 57-60.

16. Kosuge M., *et al*. "Electrocardiographic Differentiation Between Acute Pulmonary Embolism and Acute Coronary Syndromes on the Basis of Negative T Waves". *The American Journal of Cardiology* 99.6 (2007): 817-821.

17. Witting MD., *et al*. "Simultaneous T wave inversions in anterior and inferior leads: an uncommon sign of pulmonary embolism". *The Journal of Emergency Medicine* 43.2 (2012): 228-235.

18. Brahic H., *et al*. "Embolie pulmonaire massive et sus-décalage du segment ST dans les dérivations antérieures: piège diagnostique". *Annales de Cardiologie et d'Angéiologie*59 (2010): 107-110.

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