SHORT COMMUNICATION

Acute massive pulmonary embolism mimicking non-ST-elevation acute coronary syndrome

Prashanth Panduranga *, Mohammed El-Deeb

Department of Cardiology, Royal Hospital, Muscat, Oman

Received 23 January 2013; accepted 14 February 2013
Available online 18 March 2013

KEYWORDS
Pulmonary embolism; Acute coronary syndrome; T-wave inversion; Syncope; Echocardiography

Abstract Non-ST-elevation acute coronary syndrome frequently presents with negative T-wave inversion. In acute pulmonary embolism precordial T-wave inversion occurs due to right ventricular strain. We herein report a case of a 48-year-old woman presenting with syncope secondary to massive main pulmonary artery embolism which was initially diagnosed as acute coronary syndrome due to negative anterior T-wave inversion and raised troponin. In addition, her room air saturation was normal and electrocardiogram showed T-wave inversion in inferior wall leads as well. We present this case of massive pulmonary embolism with varied presentation which was initially misdiagnosed as acute coronary syndrome.

© 2013 Production and hosting by Elsevier B.V. on behalf of Egyptian Society of Cardiology.

1. Introduction

Acute coronary syndrome (ACS) frequently presents with negative T-wave inversion in anterior or inferior leads.¹ In acute pulmonary embolism (PE) precordial T-wave inversion is a common clue to right ventricular (RV) strain.¹ We herein report a case of a 48-year-old woman presenting with syncope secondary to massive main PE which was initially diagnosed as ACS due to negative anterior T-wave inversion and raised troponin.

* Corresponding author. Address: Department of Cardiology, Royal Hospital, Post Box 1331, Muscat 111, Sultanate of Oman. Tel.: +968 92603746; fax: +968 24599841.
E-mail address: prashanthp_69@yahoo.co.in (P. Panduranga).
Peer review under responsibility of Egyptian Society of Cardiology.

2. Manuscript

A 48-year-old Arabic woman, hypertensive, presented with history of frank syncope. There was no tonic-clonic seizures witnessed, and she experienced no incontinence. She denied chest pain, cough, dyspnea or fever, but complained of palpitations. Clinically, she was afebrile, conscious, oriented with her neurological, cardiovascular, and chest examinations unremarkable. Her pulse was regular at 90 beats/minute, blood pressure was 122/72 mmHg without postural drop, and respiratory rate was 18 breaths/minute. The room air oxygen saturation was 99%. All her blood investigations were normal except for white cell count of 18 × 10⁹/L (normal = 3.6–11.1) and hs-troponin T which was 0.08 μg/L; 4-h later increased to 0.104 μg/L (normal < 0.014 μg/L). An electrocardiogram (ECG) showed sinus rhythm with T wave inversion in anterior (Fig. 1). In addition, there was T-wave inversion in inferior wall leads as well. Chest X-ray was clear. Transthoracic echocardiogram (TTE) was normal with left ventricular ejection fraction of 60%. Computed tomographic (CT) scan of her
head was normal. A provisional diagnosis of ACS/non ST-elevation myocardial infarction was made in view of raising troponin and T inversion in anterior and inferior leads even though she did not have any chest pain.

Coronary angiogram was performed, but revealed normal coronaries. Telemetry revealed no brady- or tachyarrhythmias. After admission, she started complaining of mild exertional dyspnea. In view of her exertional dyspnea, D-dimer assay and brain natriuretic peptide (BNP) were analyzed. Her D-dimer assay was 1.3 μg/ml (fibrinogen-equivalent units) (normal: 0.1–0.3) and BNP was 5155 pg/ml (normal = 20–250 pg/ml). Arterial blood gas (ABG) analysis showed PaO2 of 77 mmHg (normal: 83–108), PaCO2 of 26 mmHg (normal: 36–45), calculated alveolar-arterial gradient of 40 mmHg, pH of 7.45 (normal = 7.35–7.45) and oxygen saturation of 94%.

A repeat TTE demonstrated good left ventricular systolic function, minimal pericardial effusion, no evidence of aortic dissection, but her RV was dilated, dysfunctional with typical McConnell’s sign demonstrating akinetic RV free wall with apical contraction (Fig. 2A and B, arrowheads). There was mild tricuspid regurgitation with calculated RV systolic pressure of 48 mmHg. Interestingly, basal short-axis view demonstrated a large mobile mass (measuring 2.7 × 0.8 cm) arising from left pulmonary artery and prolapsing into main pulmonary artery suggestive of a thrombus (Fig. 2C, arrowheads). A CT chest was performed which showed extensive PE of both right and left main pulmonary arteries extending distally into lobar, segmental and sub segmental branches (Fig. 2D, E and F, arrowheads). There was no pulmonary infarction. A Doppler scan of the legs revealed no deep vein thrombosis. Her thrombophilia profile was normal. The patient was treated with low molecular weight heparin (LMWH) along with overlap warfarin and her dyspnea improved markedly over a week. At six-month follow-up she was doing well.

3. Discussion

Symptoms of acute PE are nonspecific and may mimic ACS. Syncope as an initial presentation of PE occurs in 10% of patients and there is an increased incidence of main pulmonary artery embolus in patients with syncope than in patients without syncope. The main mechanisms for syncope in PE are vasovagal reaction and sudden RV pressure overload with failure along with brady- or tachy-arrhythmias. ABG analysis is not diagnostic for PE, but can show hypoxemia, hypocapnia with widened alveolar-arterial gradient and respiratory alkalosis. Since respiratory alkalosis can left-shift the Hemoglobin-O2 dissociation curve, the pulse oximetry saturation can wrongly be seen as normal. In a study, of 312 patients with documented PE, 12, 14 and 35% had normal alveolar-arterial gradient, PaO2 and PaCO2, respectively.

Recent American Heart Association (AHA) statement defines massive PE (5%) as acute PE with sustained hypotension (systolic blood pressure < 90 mmHg), pulselessness, or persistent profound bradycardia (heart rate < 40 bpm with signs or symptoms of shock) and sub massive PE as acute PE without systemic hypotension, but with either RV dysfunction (RV dilation by echocardiography or CT, RV systolic dysfunction by echocardiography, BNP elevation and/or ECG changes) or myocardial necrosis (elevated troponin). Patients without any of the above features are classified as low-risk PE. These new guidelines reiterate the importance of ECG, echocardiography and cardiac biomarkers in the diagnosis and management of PE.

Even though, ECG has limited sensitivity for PE, early precordial T-wave inversion is a common clue to RV strain. The common ECG findings in PE are anteroseptal T-wave inversion/ST-elevation or depression along with complete or
incomplete right bundle branch block, sinus tachycardia, low QRS-complex voltage, an S1Q3T3 pattern, and right axis deviation. In addition to sudden RV strain, other factors for T-inversion include hypoxemia and the release of catecholamine’s, serotonin, and histamine. In a study comparing PE and ACS patients with negative precordial T waves, negative T waves in leads III, and V1 (also V2) strongly suggested acute PE with a sensitivity of 88% and specificity of 99%. Lead III faces the inferior aspect of the RV, and leads V1 and V2 represent anterior RV. In another study, simultaneous T-wave inversions in anterior and inferior leads were associated with PE but were seen in 4–11% of cases. Jankowski et al. reported that negative T waves in leads V1–3 and inferior leads II, III, aVF (OR 1.3 [1.14–1.68]) significantly indicated acute PE with a positive predictive value of 85% and specificity of 87%.9–11

With regard to biomarkers, in low-risk PE patients, the highly sensitive D-dimer assays have a sensitivity of 98% and a negative predictive value of 100% for PE. However, these assays have a low specificity (~40%) and high false-positive results (~50%), which decrease their clinical usefulness. Acute RV myocardial stretch leads to elevation of serum troponin levels in up to 50% of patients with a moderate to large PE. In one study, significantly elevated levels of N-terminal pro-BNP were independently associated with central PE and were a predictor of death from PE. TTE and transesophageal echocardiography have a sensitivity of 50% and specificity of 90% for diagnosing PE. TTE can visualize right heart thrombi in 5% of patients with acute PE and generally does not detect emboli in the main pulmonary arteries which is seen by transesophageal echocardiography with high specificity (>90%). McConnell’s sign of akinetic RV free wall with apical sparing when present has a sensitivity and specificity of 77% and 94%, respectively for diagnosing PE. While saddle PE may usually cause RV dysfunction, up to 45% of stable patients with saddle PE have normal RV function.

In the case presented, the patient presented to the emergency department with syncope. There were no risk factors for PE and she was remarkably fit for her age. PE was not considered initially in view of any significant symptoms or signs related to the respiratory system. In addition, room air saturation and echocardiogram were deceptively normal. An ABG analysis was not performed initially. In the absence of any risk factors for PE application of prediction scores like Wells or Geneva would have shown a low probability for PE. Negative T-wave
inversion on ECG with troponin raise led to the diagnosis of non-ST-elevation acute coronary syndrome. Persistence of her unexplained dyspnea led to investigate for PE and an elevated D-dimer and BNP necessitated a repeat echocardiogram which demonstrated classical signs of PE. Interestingly, TTE showed evidence of the thrombus floating in main pulmonary artery, which is uncommon. Even though the diagnosis was delayed, she did receive full dose of LMWH since admission, as her initial diagnosis was acute coronary syndrome.

In conclusion, PE presenting with negative T-wave inversion can mimic ACS and mislead physicians. Simultaneous T-wave inversions in anterior and inferior leads are important clues suggesting PE rather than ACS. In addition, physicians must be aware that PE can present as syncope without risk factors, respiratory symptoms or desaturation on pulse oximetry. They should take clue from the combination of multiple investigations including D-dimer, BNP, troponin T, ECG and echocardiography to come to the diagnosis of PE, as defined in the AHA guidelines.

Disclosures/funding

None.

References