

## CASE REPORT

# A potential diagnostic pitfall in ST elevation: Acute pulmonary embolism or ST-segment elevation myocardial infarction

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**Abstract**

The diagnosis of acute pulmonary embolism (APE) is a great challenge for physicians due to its nonspecific symptoms, and often missed or misdiagnosed as acute coronary syndrome. Electrocardiographic (ECG) abnormalities are seen in majority of patients with APE. Recently, APE with ST-segment elevation (STE) in leads  $V_1$ - $V_3/V_4$ , mimicking ST-segment elevation myocardial infarction (STEMI), has been described. However, coronary angiography showed that the patient's coronary arteries were mostly normal. Herein, we describe a case of APE presenting with STE in  $V_1$ - $V_4$ , along with severe stenosis of the left anterior descending (LAD) artery.

**KEYWORDS**

electrocardiogram, myocardial infarction, pulmonary embolism, ST-segment elevation

## 1 | CASE REPORT

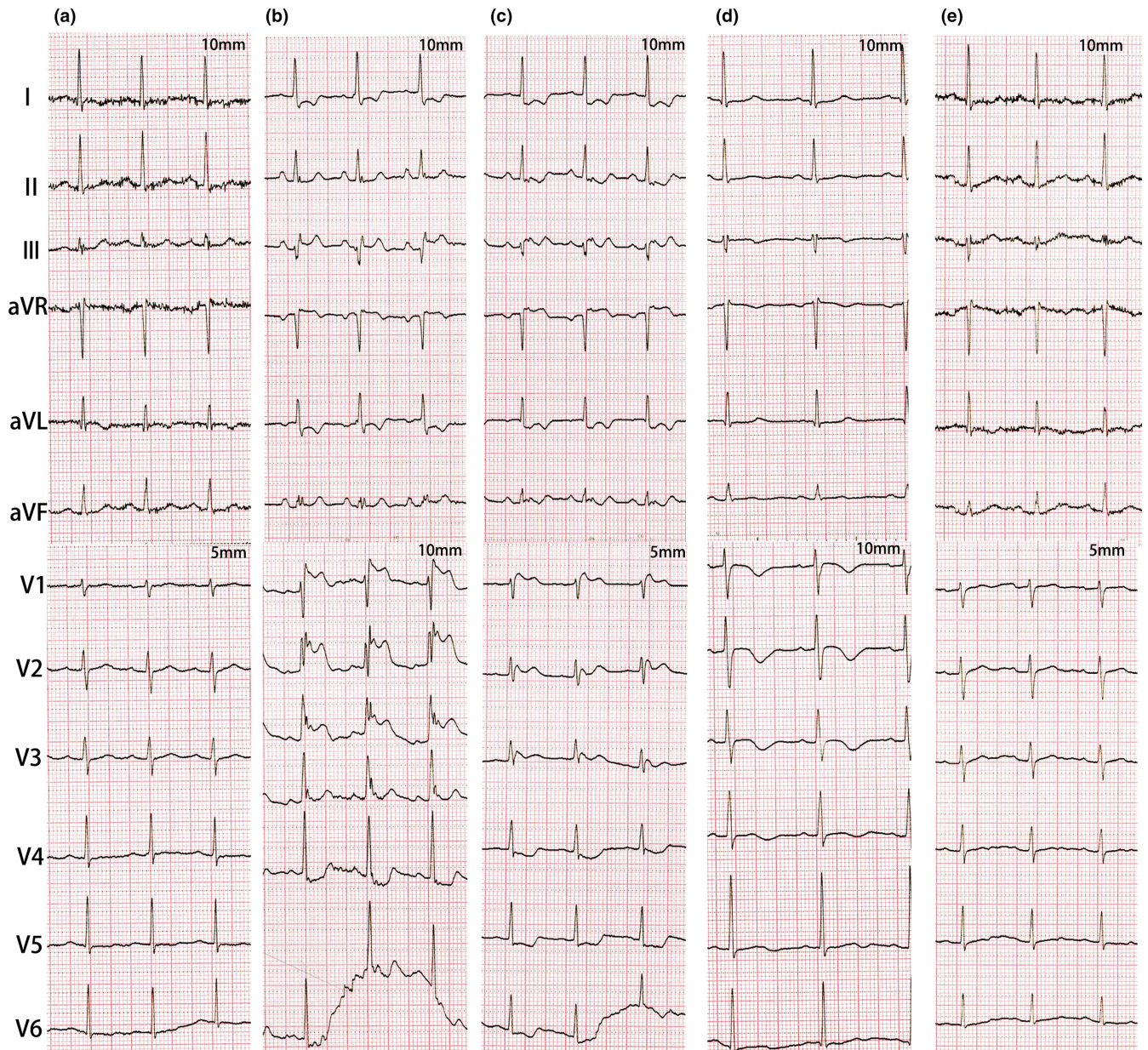
A 58-year-old woman with history of hypertension was admitted to our hospital due to dyspnea induced by activity, which persisted for approximately 3–5 min each time for 1 month, and syncope for 17 days. Four days earlier, she experienced gradually worsening dyspnea that progressed to occur at rest in addition to episodes of fatigue and palpitation. There was no recent surgery, trauma, or prolonged travel, and she had no family history of APE. Physical examination was unremarkable. On admission, her pulse rate was regular (94/min), blood pressure 174/104 mmHg, and respiratory rate 20 breaths/min. The initial ECG showed sinus rhythm with negative T wave (NTW) in leads I and aVL, and low T wave in leads  $V_4$  to  $V_6$  (Figure 1a). Complete blood count, blood chemistry, and cardiac troponin I were within normal limits except the D-dimer of 0.72 mg/L (<0.5 mg/L). An echocardiogram showed no obvious abnormalities. Informal report of computed tomography (CT) pulmonary angiography revealed no obvious acute pulmonary embolism, while on the second day at admission, formal report indicated partial embolism of small arteries in the middle and lower lobe of the right lung (Figure 2).

The patient was treated with aspirin, clopidogrel, isosorbide mononitrate, metoprolol, atorvastatin, and irbesartan hydrochlorothiazide initially. The night after admission, she experienced sudden syncope after urinating, which persisted for approximately 2 min. At the time of the onset, she was unconscious and hypotensive (76/50 mmHg), heart rate 95 bpm, and tachypneic with a respiratory rate 25 breaths/min. Oxygen saturation was 90% while breathing 33% oxygen. On 12-lead ECG, there was sinus rhythm with incomplete right bundle branch block, STE up to 3 mm in precordial leads  $V_1$ - $V_4$ , ST-segment depression in leads  $V_5$ - $V_6$ , and NTW in leads I and aVL (Figure 1b). Emergency transthoracic echocardiogram revealed RV dilatation, moderate pulmonary hypertension, and moderate tricuspid regurgitation (Figure 3a-c). Lower extremity venous Doppler showed bilateral intermuscular vein thrombosis. Hence, APE was highly suspected due to symptoms of syncope and dyspnea, the new presence of ECG manifestations, and RV dysfunction. However, given the unstable hemodynamic status, CT pulmonary angiography was not repeated. The patient received infusion of 50 mg alteplase over 2 hours and was hemodynamically stable after thrombolytic therapy.

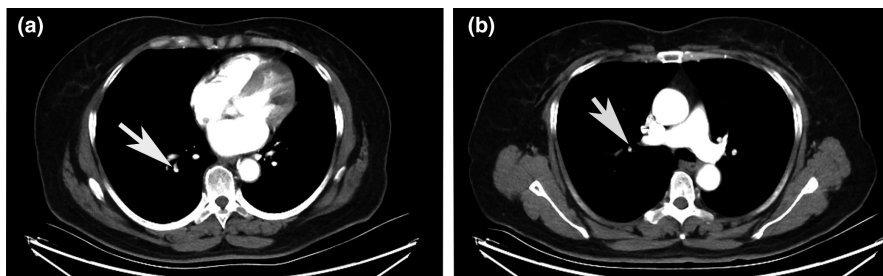
Bo Zheng and Fei Bian authors contributed equally to this work.

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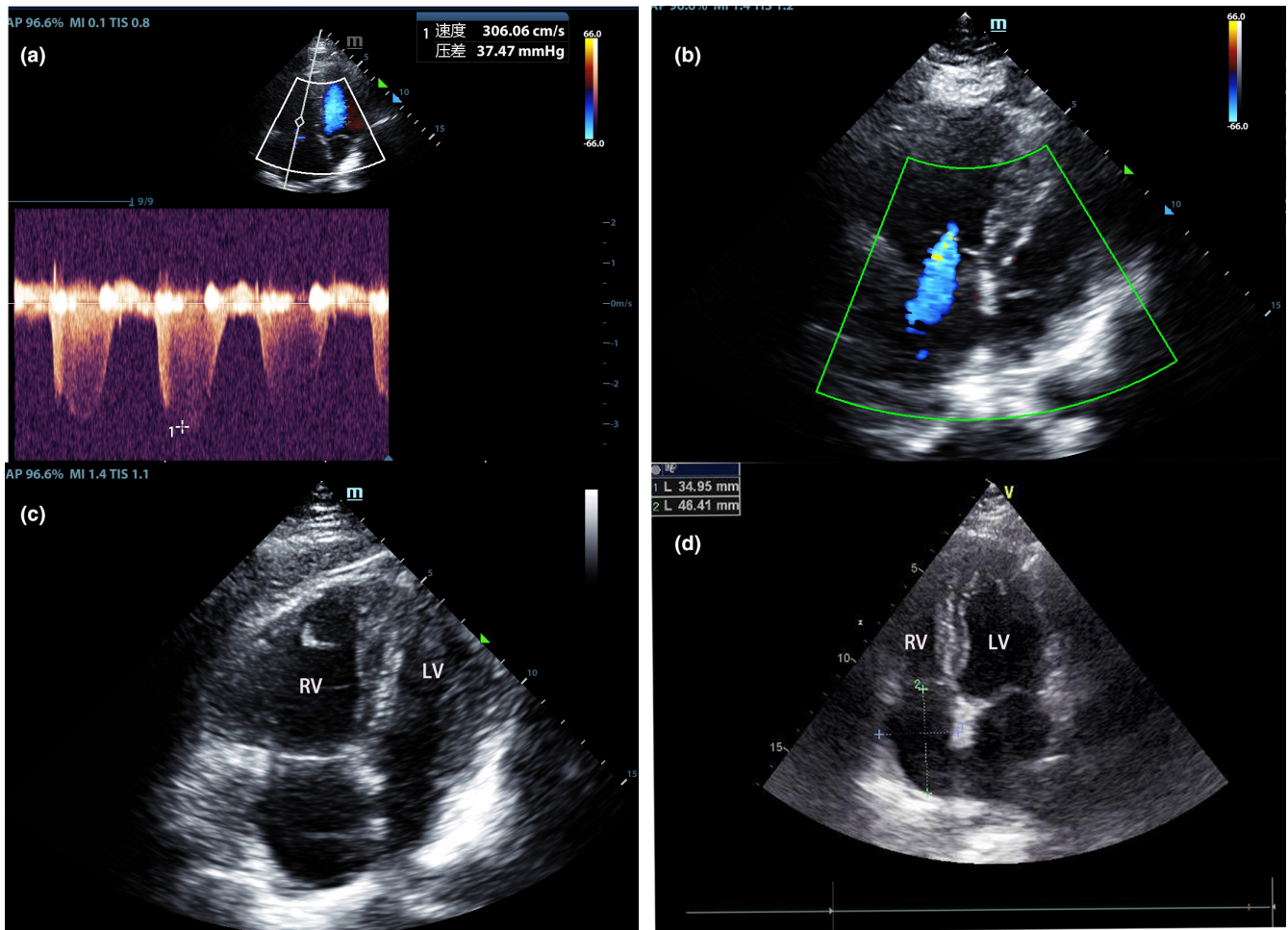
**FIGURE 1** (a) Initial ECG showed NTW in leads I and aVL and low T wave in leads  $V_4$ - $V_6$ . (b) When she experienced sudden syncope, ECG was performed, showing incomplete right bundle branch block, STE up to 3 mm in precordial leads  $V_1$ - $V_4$ , ST-segment depression in leads  $V_5$ - $V_6$ , and NTW in leads I and aVL. (c) When the BP recovered, ECG showed STE in leads  $V_1$ - $V_4$  declined. (d) Two days after admission, ECG showed NTW in leads  $V_1$ - $V_4$ . (e) The ECG 8 days later showed disappearance of NTW in leads  $V_1$ - $V_4$



**FIGURE 2** (a, b) Computed tomographic pulmonary angiogram at horizontal section showed partial embolism of small arteries in the middle and lower lobe of the right lung

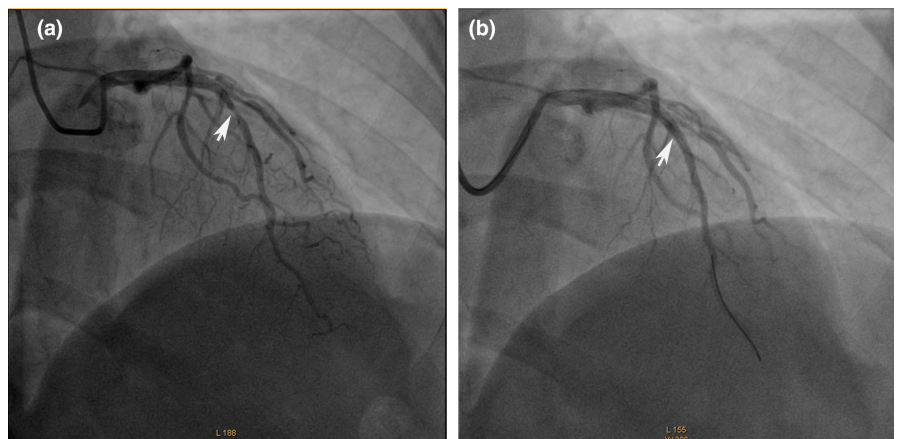
A repeated ECG during the period of thrombolytic therapy, when the BP recovered to 105/75 mmHg, showed STE in leads  $V_1$ - $V_4$  declined gradually (Figure 1c). Two days after admission, the ECG showed NTW in leads  $V_1$ - $V_4$  (Figure 1d) and the

ECG 8 days later showed disappearance of NTW in leads  $V_1$ - $V_4$  (Figure 1e). Repeated troponin I level was 1.21 (normal value 0-0.09 ng/ml). Repeated transthoracic echocardiogram showed normal RV and LV morphology and function (Figure 3d). The



**FIGURE 3** (a) and (b) Color Doppler examination showed moderate tricuspid regurgitation and 37 mmHg of regurgitation pressure gradient. (c) The apical four-chamber view revealed RV dilatation. (d) The apical four-chamber view after treatment showed normal RV and LV morphology and function

**FIGURE 4** Coronary angiography. Coronary angiogram showed that 70–80% occlusion of the middle LAD artery (a, white arrow), followed by insertion of a drug-eluting stent (b, white arrow)



patient's symptoms are completely relieved within 48 h. Because of the similarity of the presenting symptoms and the electrocardiogram (ECG) manifestations, some doctors believed that the diagnosis of pulmonary embolism was controversial. A few days later, a coronary arteriogram confirmed 70–80% occlusion of the middle LAD artery (Figure 4a). A drug-eluting stent was implanted in the LAD (Figure 4b). The patient was discharged without any signs of complications.

## 2 | DISCUSSION

APE presents with a wide variety of ECG features (Omar, 2016). These include sinus tachycardia, S1Q3T3 pattern, rightward shift in QRS axis, complete or incomplete right bundle branch block, atrial arrhythmias, P-pulmonale, and precordial T-wave inversion (Özer et al., 2011; Stein et al., 1975). A few case reports describe ECG presentation of pulmonary embolism as ST-segment elevation in

precordial leads, mimicking anteroseptal myocardial infarction (Lin et al., 2009; Lu et al., 2018), but underlying mechanism is not very clear and several explanations have been provided as follows.

First is the development of RV transmural ischemia and infarction. When a massive pulmonary embolism occurs, acute elevation of afterload caused increased right ventricular systolic and end-diastolic pressure, leading to increased RV oxygen demand and to a significant reduction in pulmonary perfusion (Omar, 2016; Zhong-Qun et al., 2014). Leftward shift of the interventricular septum due to acute right heart failure will increase the left ventricle end-diastolic pressure and decrease the cardiac output; subsequently, hypotension and coronary flow can cause severe RV transmural ischemia, leading to STE in the precordial leads  $V_1-V_3/V_4$  (Zhong-Qun et al., 2013, 2014). Paradoxical coronary embolism may be the second explanation for ST-segment elevation in leads  $V_1-V_3/V_4$  (Cheng, 2005). Goslar and Podbregar (2010) reported a case of APE presenting with STE in the precordial leads  $V_1-V_4$ , where there was paradoxical embolism into the conus artery. When the right heart pressure increases, embolus pass through an atrial septal defect or patent foramen ovale, causing paradoxical embolism. In the present case, we describe a case of APE presenting with STE in  $V_1-V_4$ , along with severe stenosis of the left anterior descending (LAD) artery which is different from previous report. Therefore, one should be aware of the possibility of presentation of concomitant APE and STEMI induced by coronary atherosclerosis.

Pulmonary CTA is the most commonly used method for the diagnosis of pulmonary embolism. Although not a direct test for PE, ultrasound shows echocardiographic changes, such as RV dilation or systolic dysfunction, which plays an important role in diagnosis, differential diagnosis, and risk stratification (Omar, 2016; Shy et al., 2015). In our case, small-scale pulmonary embolism was neglected at the beginning. When syncope occurred, bedside ultrasound indicated the presence of RV dilatation, moderate pulmonary hypertension, and moderate regurgitation. We suspected that the embolism occurred again, and it was a massive embolism which has received thrombolytic therapy. Herein, we highlight the use of bedside ultrasound to aid in the diagnosis of pulmonary embolism, especially in an emergency.

In conclusion, APE may present with STE in leads  $V_1-V_3/V_4$ , which is hard to differentiate from STEMI. We need to make judgments based on the patient's medical history, clinical characteristics, electrocardiogram, cardiac color Doppler ultrasound, and D-dimer. Serious consequences can occur if any of these two diagnoses is missed. At the same time, one should be aware of the possibility of presentation of concomitant APE and STEMI; thrombolytic therapy may be a good treatment option.

## ACKNOWLEDGEMENT

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## CONFLICT OF INTEREST

The authors declare no potential conflicts of interest.

## AUTHOR CONTRIBUTIONS

BZ and FB performed the collection of data. JW wrote the manuscript and performed the literature search. JL and HX guaranteed the integrity of the entire study. All authors have read and approved the manuscript reviewed in the literature.

## ETHICS APPROVAL

This case report has been conducted according to the standards of the Declaration of Helsinki.

## DATA AVAILABILITY STATEMENT

All data are available and can be provided if requested.

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