

Original Article

Acute pulmonary embolism with electrocardiographic changes mimicking acute coronary syndrome: a case report and literature review

Fei Wang, Jian-Qiang Kang, Shu-Yan Chen, Gen-Fa Wang

Department of Geriatrics, Xinhua Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200092, China

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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 dyspnoea and chest discomfort 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. More recently, precordial T wave inversion has been described to be a clue to suggest PE. However, this ECG change is more likely to present in ACS. We herein reported a case of a 78-year-old man presenting with progressive shortness of breath on exertion secondary to submassive pulmonary embolism which was initially misdiagnosed as ACS due to diffuse T wave inversion in both precordial leads V1-6 and inferior Leads II, III and aVF. Here, we discussed the diagnosis of this case and reviewed the medical literature with an emphasis on the limitations of ECG for the differentiation between PE and ACS.

Keywords: Acute pulmonary embolism, acute coronary syndrome, T wave inversion

Introduction

Acute pulmonary embolism (PE) is potentially a life threatening emergency that needs prompt treatment. Symptoms and clinical signs of PE are often nonspecific and may overlap with acute coronary syndrome (ACS), creating difficulties in the differential diagnosis [1]. The 12-lead electrocardiogram (ECG) is often the initial available diagnostic examination. Although variable electrocardiographic findings have been reported to be associated with acute PE, the ECG alone is of limited value for the confirmation or exclusion of the disease [2, 3]. Here we report a case of PE that was initially misdiagnosed as ACS due to ECG manifestation of diffuse T wave inversion in both precordial leads V1-6 and inferior Leads II, III and aVF.

Case report

A 78 year old man with a history of hypertension presented to the emergency department due to progressive shortness of breath on exertion for seven days. He denied chest pain, pal-

itation, cough and hemoptysis. His usual medication included angiotensin receptor blocker and calcium channel blocker. His blood pressure was 124/70 mm Hg; pulse regular at 82 beats/minutes; and respiratory rate, 20 breaths/minutes. His physical examination was unremarkable except for minor bilateral rales in the lower lung fields. Laboratory assessment revealed markedly elevated pro-brain natriuretic peptide (BNP) which was 11479 pg/ml (normal < 526 pg/ml). The serum level of cardiac isoenzymes, including troponin T, was within the normal reference ranges. A 12-lead ECG showed sinus rhythm with diffuse T wave inversion in both precordial leads V1-6 and inferior Leads II, III and aVF (**Figure 1**). From the above findings, a provisional diagnosis of non ST segment elevation ACS complicated by heart failure, possibly due to critical stenosis/occlusion of proximal left anterior descending coronary artery that wrapped around the cardiac apex, was suspected and the patient was hospitalized with the intention of coronary angiography.

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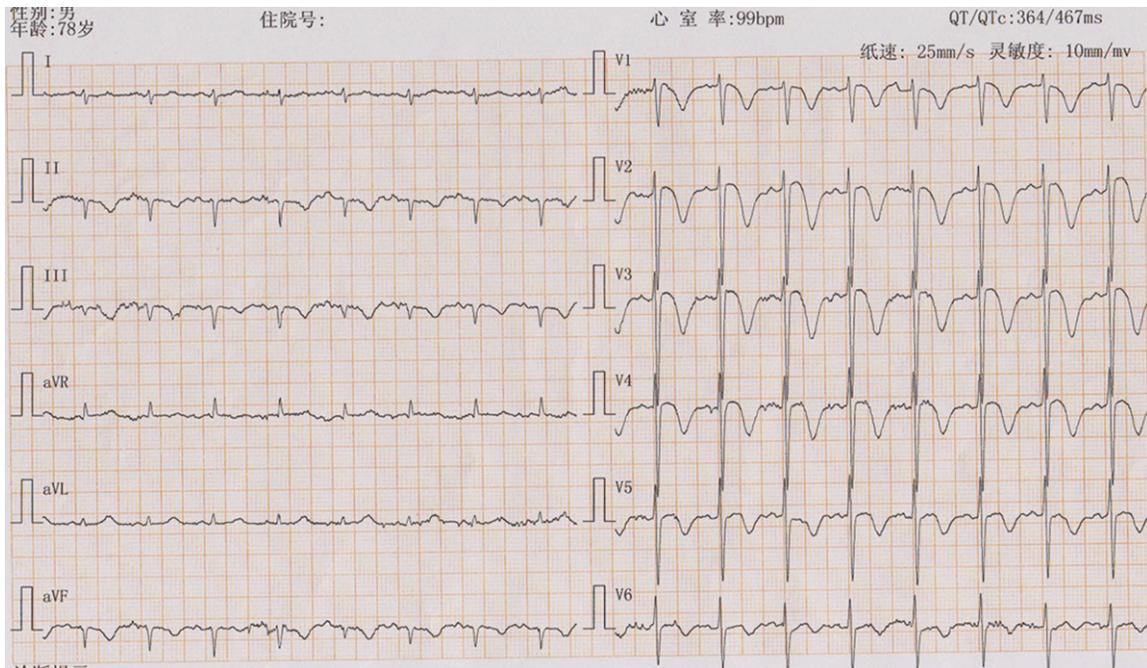


Figure 1. ECG at admission showing sinus rhythm with diffuse T wave inversion in all precordial leads and inferior Leads.



Figure 2. Pulmonary CT angiography performed on the next day after admission showing bilateral inferior pulmonary artery thrombosis (arrows).

After admission, the ECG and cardiac isoenzymes were repeated and showed no evolutionary changes. No significant abnormalities were found on chest X-ray. Telemetry revealed sinus rhythm with occasionally atrial premature beats

and paroxysmal atrial tachycardia. His D-dimer assay was 1.17 mg/L (normal < 0.5 mg/L). Arterial blood gas analysis showed PaO₂ of 8.04 kpa (normal = 11-13 kpa), PaCO₂ of 4.24 kpa (normal = 4.5-6 kpa), pH of 7.428 and oxy-

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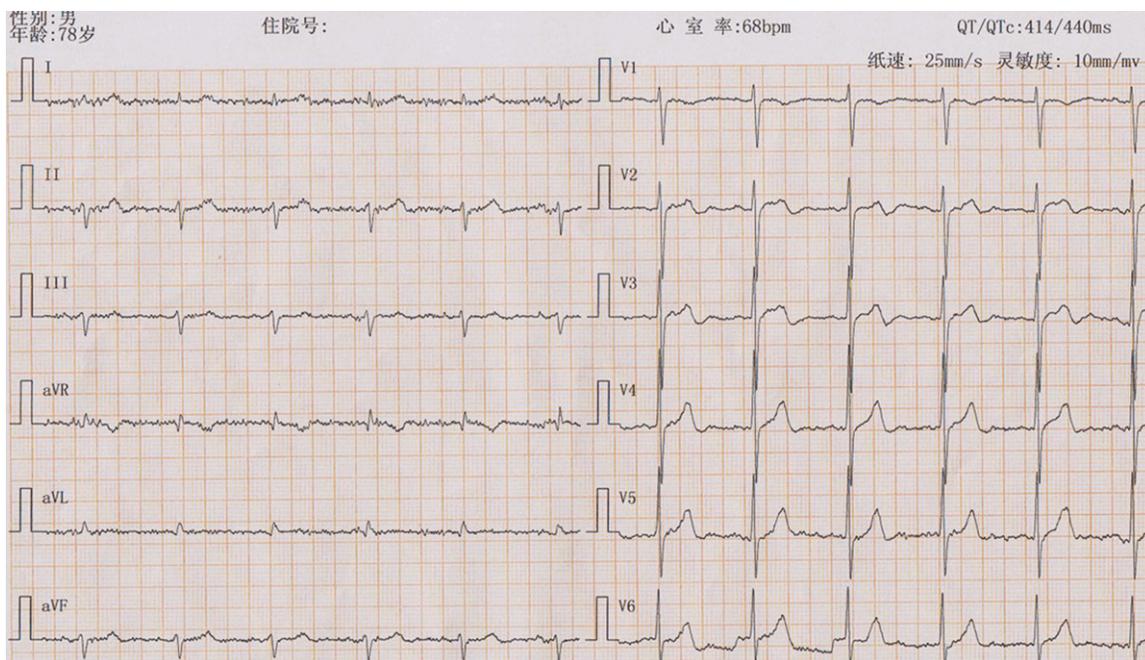


Figure 3. ECG recorded 2 months after the confirmed pulmonary artery embolism showing normalization of diffuse T wave inversion.

gen saturation of 91%. Echocardiography was performed, revealing normal left ventricular ejection fraction and no segmental contraction abnormalities. The right atrium was enlarged and there was mild to moderate tricuspid regurgitation with an estimated right ventricle systolic pressure to be around 75 mm Hg. Based on these findings, acute pulmonary embolism was suspected and computed tomographic (CT) pulmonary angiography was done immediately, which showed bilateral inferior pulmonary artery thrombosis (**Figure 2**), confirming the echocardiographic suggestion of PE. The patient was treated with low molecular weight heparin. After an uneventful recovery, he was discharged on oral anticoagulation therapy with warfarin. The ECG recorded 2 months after the index event showing normalization of diffuse T wave inversion (**Figure 3**). The patient was symptom free and doing well at six months of follow up.

Discussion

Symptoms such as dyspnea and chest discomfort are often shared in patients with PE and ACS, raising some problems of differential diagnosis between the two [1]. To make the situation more complicated, some laboratory find-

ings including elevated concentrations of cardiac troponin and proBNP can also be present in both conditions [4, 5]. Although the highly sensitive D-dimer is useful for the safe excluding of PE in patients with low to intermediate clinical probability of PE as confirmed by current European guidelines on PE [6], mild elevation can often be seen in ACS.

The 12-lead ECG is a prompt, inexpensive, and most available diagnostic tool for initial evaluation. With regard to ECG changes associated with acute PE, the classic S1Q3T3 pattern, first described by McGinn and White in 1935 [7], constitutes the most widely known ECG abnormality that has a high specificity but low sensitivity for the diagnosis of PE. According to the literature, other variable ECG findings associated with acute PE included sinus tachycardia, rightward axis shift, P-pulmonale pattern, displacement of the transitional zone to the left, complete or incomplete right bundle branch block, T wave inversion in leads V1-4 and ST segment elevation or depression [2, 8, 9]. However, none of these findings are specific for PE, with some patterns even mimicking ACS. There are occasionally case reports that misdiagnose massive or submassive PE as anterior/inferior ST segment elevation myocardial infarction.

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tion as well as non ST segment elevation ACS based on ECG findings [10-13]. More recently, simultaneous T-wave inversions in anterior and inferior leads has been described to be an important clue to suggest PE [14, 15]. Since T-wave inversions usually cause many physicians to narrow the focus of their diagnostic workup to ACS, detailed consideration of ECG changes may potentially help to differentiate PE from ACS. In a study comparing precordial T wave inversion in patients with PE and ACS caused by LAD disease, PE was strongly associated with the presence of inverted T in both leads III and V1-2 and maximum magnitude T inversion in leads V1-2. The combination of these 2 findings identified PE with 98% sensitivity and 92% specificity [16]. However, T wave inversion in all precordial leads V1-6, as in the present case, was a rarely ECG finding in patients with PE.

The cause of ECG change in the present case was uncertain, but may involve the pathophysiological consequence of PE. It has been shown that acute PE can result in abrupt increase in RV pressure overload or strain, excessive neurohumoral activation, impaired coronary blood flow, and hypoxia, leading to ischemia and dilatation of the right ventricle and possibly T wave inversion [17, 18]. Since lead III faces the inferior region of the RV, and leads V1-2 represents the anterior region of the RV, it is likely that, with increasing severity and dilatation of the RV towards the left, inverted T move towards the left, that is, from leads III to aVF to II in limb leads and from leads V1 to V6 in precordial leads.

In the case described here, the patient presented with progressive shortness of breath on exertion for seven days. The serum level of proBNP was markedly elevated. Physical examination revealed minor bilateral rales in the lower lung fields. There were no risk factors for PE and he was remarkably fit for his age. According to prediction scores such as Wells or Geneva, the patient had a low to intermediate clinical probability for PE [6]. In view of these findings, PE was not considered in the initial diagnostic workup. Notably, T wave inversion in all precordial leads V1-6 and inferior leads II, III and aVF led to the diagnosis of non-ST-elevation ACS. Fortunately, the patient was correctly diagnosed without prolonged delay and avoided unnecessary interventional coronary angi-

ography due to timely performed echocardiography, which suggested the presence of RV pressure overload possibly caused by PE that are finally confirmed by CT.

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. Because echocardiography has great value for the identification of RV overload and other potential abnormalities, it should be performed as rapidly as possible to make the correct diagnosis and enable appropriate therapeutic intervention.

Disclosure of conflict of interest

None.

Address correspondence to: Gen-Fa Wang, Department of Geriatrics, Xinhua Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200092, China. E-mail: genfa_wang@163.com

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