T Wave Inversion with QT Interval Prolongation in Multiple ECG Leads Associated with Acute Pulmonary Embolism: A Case Report

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T wave inversion in precordial leads of electrocardiogram (ECG) are often seen in patients with acute coronary syndrome (ACS), but may be due to other etiology such as acute pulmonary embolism (PE). We report on a 74 year-old woman admitted with a diagnosis of unstable angina because of chest pain and ECG changes of T wave inversion and QT interval prolongation in multiple leads. Coronary angiography showed no evidence of coronary artery stenosis. Acute PE was diagnosed after perfusion lung scintigraphy was done. Within one month following anticoagulant therapy, the ECG revealed complete resolution of the T wave inversion and QT prolongation.

Key words: electrocardiogram, T wave inversion with QT prolongation, pulmonary embolism

Introduction

Acute pulmonary embolism (PE) is well known to cause numberous ECG changes. T wave inversion in the right precordial leads is the most common abnormality. This ECG changes mimicking acute coronary syndrome (ACS) are frequently misdiagnosed. Global T wave inversion with QT prolongation associated with acute PE is not common and was first reported by Liu in 1993. We report a 74-year-old woman with ECG showing T wave inversion and QT prolongation in multiple leads, who was diagnosed as unstable angina initially. The ECG findings reverted to normal within one month of anticoagulation treatment.

Case Report

A 74-year-old woman was brought to our Emergency Room because of chest pain and dyspnea for one week. She had had a 10-year history of hypertension with regular treatment with atenolol. There was no history of chest pain or smoking in the past. On physical examination, her blood pressure was 120/72mmHg, pulse 68 beats per minute, respirations 23 per minute and temperature 36°C. There were clear bilateral breathing sounds. The heart rhythm was regular and a grade 2/6 systolic murmur was heard at the left lower sternal border. A chest X-ray film showed mild cardiomegaly. The ECG showed sinus rhythm with T wave inversion and QT prolongation in III, aVF and V1-V4 leads (Fig. 1A). Her blood sugar was 105mg/dl, creatine kinase and CK-MB were 42 and 9IU/L, respectively, and troponin I was 0.19ng/ml (normal range <0.5ng/ml). Complete blood cell count and other blood biochemistry were within normal limits. Under the impression...
of unstable angina, aspirin and clopidogrel were given, and nitrate and heparin were administered parenterally. Supplemental oxygen was administered. She was then admitted to our medical intensive care unit. Cardiac catheterization was done 12 hours later and revealed no evidence of coronary artery stenosis. A follow-up ECG showed QT interval decreased and T wave inversion persisted. Echocardiography showed good left ventricular (LV) function, moderate tricuspid regurgitation, with an estimated right-side systolic pressure of 52mmHg. The D-dimer level was 664ng/mL. Perfusion lung scintigraphy revealed multiple segmental and subsegmental perfusion defects in both lung fields (Fig. 2A). Pulmonary embolism was impressed. The patient was discharged asymptomatic 7 days later after heparization and maintained on oral anti-coagulant.
One month later, a follow-up ECG revealed resolution of T wave inversion and the QT interval became normalized (Fig. 1B). Follow-up lung scan 2 years later revealed nearly complete resolution of perfusion defect (Fig. 2B).

**Discussion**

The ECG findings associated with PE are numerous, including rhythm abnormalities, nonspecific ST segment-T wave changes, T wave inversion in the right precordial leads, right or left axis deviation, S1Q3 or S1Q3T3 pattern, right bundle branch block, and acute cor pulmonale\(^3\). A recent study by Ferrari et al.\(^1\) showed inverted T waves in the right precordial leads to be the most frequent ECG sign of massive PE. They found that this ECG sign was also associated with the best sensitivity, specificity, and positive and negative predictive values for diagnosing PE. But the prevalence of T wave inversion with QT interval prolongation during acute phase of PE was not mentioned in that study. Global T-wave inversion with QT interval prolongation during acute phase of PE was first reported in 1993\(^2\). In a retrospective study\(^4\), this novel ECG phenomenon was found in 5 of 140 patients with acute PE. Several other diseases causing T-wave inversions with QT interval prolongation have been reported; the most common being acute myocardial infarction (particularly subendocardial infarction) and acute central nervous system disorders. In addition, cardiac metastasis, apical hypertrophic cardiomyopathy, pheochromocytoma, cocaine abuse, non-ischemic pulmonary edema and takotsubo cardiomyopathy may result in T-wave inversions with QT interval prolongation\(^5\-\(^6\).

**T-wave inversions with QT interval prolongation in multiple ECG leads are often seen in patient with ACS, but also occur in those with acute PE. To distinguish the two conditions using standard 12 lead ECG is a challenge for cardiologists and emergency department physicians. In a recent study, Kosuge et al.\(^9\) evaluated the value of ECG for discriminating between patients with acute PE and ACS who had negative T waves in the predordial leads (V1-V4) on the admission ECG. Their conclusions have pointed toward the presence of negative T-wave in both leads III and V1 allows acute PE to be differentiated simply but accurately from ACS in patients with negative T waves in the precordial leads.

The pathophysiology of this ECG changes associated with PE remain unknown. Several mechanisms including coronary insufficiency, humoral factors including histamine and serotonin induced myocardial ischemia, and catecholamine mediated phenomenon have been proposed. It
has been suggested that coronary insufficiency following pulmonary embolism may be caused by anoxemia, shock, RV strain, and possibly reflex coronary vasoconstriction\(^\text{(10)}\). In massive PE, decreases in mean arterial pressure associated with increases in right ventricular (RV) end-diastolic pressure impair the myocardial perfusion and oxygen supply. Elevated right-sided pressures can further impair coronary perfusion and LV distensibility by increasing coronary venous pressure. Both increased oxygen demands resulted from elevated wall stress and decreased oxygen supply have been shown to precipitate RV ischemia, which is thought to be the cause of RV failure\(^\text{(11)}\). RV infarction has been described during the acute phase of massive PE, with angiographically normal epicardial coronary arteries\(^\text{(12)}\), it is conceptually appearing to attribute the ECG findings to myocardial ischemia. However, in a series of 12 patients with massive PE and an ECG appearance suggestive of myocardial ischemia, a very early myocardial scintigraphy with MIBG did not demonstrate any perfusion defect suggestive of ischemia\(^\text{(13)}\). These ECG changes in acute PE can not be explained solely on the mechanism of coronary insufficiency. The release of humoral factors including serotonin and histamine in acute PE that could lead to pulmonary arterial vasoconstriction and increased pulmonary artery pressure with resultant right ventricular dysfunction and myocardial ischemia\(^\text{(11,14)}\). Therefore, it is possible that the T wave inversion with QT interval prolongation during the acute phase of PE is due to the effects of these humoral factors. Another explanation for these ECG changes in acute PE could be via a catecholamine mediated phenomenon as it occurred in patients with pheochromocytoma and cocaine use. Recently, a unique syndrome of transient left ventricular apical ballooning, also known as takotsubo cardiomyopathy, characterized by transient LV systolic dysfunction and ECG changes mimicking acute myocardial infarction despite the absence of obstructive significant coronary artery stenosis is also associated with deep inverted T wave with QT interval prolongation\(^\text{(8)}\). Wittstein et al. have reported very high levels of catecholamines in this syndrome but whether catecholamins are released in significant amounts in acute PE remained to be investigated.

In conclusion, acute PE may result in T-wave inversion with QT interval prolongation. This ECG changes and the clinical symptoms such as chest pain or dyspnea are often difficult to differentiate from ACS. Early recognition and appropriate management may lead to normalization of the ECG changes and prevent hemodynamic deterioration or death.

References

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與肺栓塞有關的心電圖多導程T波倒置及QT間期延長—病例報告

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雖然急性冠心症的心電圖胸前導程常出現T波倒置，但是這種心電圖變化也可見於其他原因如急性肺栓塞。我們報告一位74歲女性病例，因為胸痛以及在心電圖多個導程出現T波倒置及QT間期延長的異常變化，被診斷為不穩定心絞痛。但血管攝影顯示無冠狀動脈狹窄情形，而在肺灌注攝影檢查後診斷為急性肺栓塞，經抗凝治療後心電圖完全恢復正常。

關鍵詞：心電圖，T波倒置及QT間期延長，肺栓塞