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PRONOUNCED T WAVE INVERSION IN AN 18 YEAR OLD PATIENT WITH ACUTE PULMONARY EMBOLISM.

Zack Paul M, Klamerus Mark A, Carlson Katherine. Ingham Cardiovascular Group, Michigan State University, USA

INTRODUCTION

The ECG findings of diffuse T wave inversion and QTc prolongation have been reported as a rare occurrence in the setting of acute pulmonary embolism [1-3]. We report a case of a young patient with acute pulmonary embolism and a presenting ECG demonstrating pronounced T wave inversion of greater magnitude than seen on previously published electrocardiograms.

CASE REPORT

An 18 year old white female presented to the emergency department with sudden onset of severe dyspnea. She was previously in good health, and an active participant in her high school track team. The patient had no history of tobacco exposure. She had a history of oral contraceptive use beginning four months prior to admission. On physical examination, the temperature was 98.8 degrees F, the blood pressure was 117/70 mm Hg, the heart rate was 68, the respiratory rate 20 and the oxygen saturation 97% on room air. The lungs had scattered rhonchi. On cardiac examination, there was a regular rhythm with a soft systolic murmur at the left sternal border. The ECG at the time of admission (Figure 1) demonstrated pronounced T wave inversion in multiple leads with as much as 12 mm of T wave inversion in the precordial leads and a prolonged QTc of 452 ms.



The initial laboratory evaluation demonstrated normal complete blood count, electrolytes and renal function. There was elevation of the D-dimer level. Standard chest radiography was normal but a subsequent chest CT scan demonstrated extensive bilateral pulmonary emboli. She was treated with full anticoagulation using intravenous Heparin and oral Warfarin with prompt improvement in symptoms of dyspnea. The hospital evaluation included normal serial creatine kinase (CK-MB) and troponin-I levels. Bilateral lower extremity venous ultrasound studies demonstrated no evidence of deep venous thrombus. A trans-thoracic echocardiogram demonstrated normal left ventricular size and contractility, enlarged right ventricular size with right ventricular volume overload pattern and elevated estimated right heart pressures with a pulmonary artery systolic pressure of 55 mm Hg. She was discharged home on the fourth hospital day with continuation of Warfarin and discontinuation of oral contraceptives. An outpatient hematology evaluation found no identifiable coagulation disorder. At a six week follow up visit, she was asymptomatic. The ECG (Figure 2) demonstrated resolution of T wave inversion with a normal QTc of 394 ms.



FIGURE 2

A follow up echocardiogram demonstrated normal right ventricular size and function, normal right ventricular contractility and improvement in estimated right heart pressures, with a pulmonary artery systolic pressure of 35 mm Hg. A standard treadmill stress test demonstrated an exertional level of 17 METS and 95% of maximum predicted heart rate without symptoms of chest pain, arrhythmias or ECG evidence of myocardial ischemia.

DISCUSSION

Diffuse T wave inversion with QTc prolongation is known to occur as an unusual finding in patients with acute pulmonary embolism [1-3]. Our patient is the youngest reported case to date and the ECG demonstrates a greater magnitude of T wave inversion than previously reported.

The mechanism of T wave inversion in the setting of acute pulmonary embolism is not known and has been proposed to be secondary to global or right ventricular myocardial ischemia, a catecholamine mediated phenomenon or attributable to the release of humoral factors including histamine and serotonin [2]. The QTc prolongation seen in the setting of pulmonary embolism has been suggested to be secondary to a mechanical-electrical feedback mechanism [4].

The clinical significance of these ECG findings in patients with acute pulmonary embolism is uncertain. Although QTc prolongation has been associated with polymorphic ventricular tachycardia [5], such arrhythmias have not been described in the few patients reported with this ECG pattern and acute pulmonary embolism. In patients with T-wave inversion and QTc prolongation but without acute pulmonary embolism, a limited QT dispersion has been found on electrophysiological evaluation [5] which may indicate a more benign prognosis of this ECG pattern with respect to life threatening ventricular arrhythmias.

It is of note that at the time of presentation our patient had a lower heart rate than would typically be observed with acute pulmonary embolism. Sinus nodal dysfunction, including prolonged sinus node recovery time and sinoatrial conduction time has been found in young patients with long QT syndrome [6], and a similar phenomenon may have contributed to the relatively decreased heart rate seen in our patient. Following normalization of her ECG, our patient was found to have a normal heart rate response to maximal treadmill exercise.

Further investigation is needed to determine the prevalence of this ECG pattern in the setting of acute pulmonary embolism and to better clarify the electrophysiological mechanism and associated clinical implications.

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