Giant Inverted T waves in the emergency department: case report and review of differential diagnoses

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Abstract

Inverted T waves are frequently seen in electrocardiograms (ECGs) and may represent a myriad of pathologies or nonspecific change. However, deep (giant) inverted T waves are only seen in a few clinical conditions. Presence of giant T waves should generally prompt investigations for apical (Yamaguchi) variant of hypertrophic cardiomyopathy, raised intracranial pressure, severe myocardial ischemia, posttachycardia syndrome, and others. This report describes an unusual case of moderate but not massive pulmonary embolism presenting with an ECG finding of giant inverted T waves. A review of the common conditions associated with such an ECG is also presented.

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Keywords:

Pulmonary embolism; T-wave inversion; ECG findings; Hypertrophic cardiomyopathy; Myocardial Ischemia

A 55-year-old man presented to the emergency department with mild aching chest discomfort and exertional dyspnea for 1 week. History was unremarkable except for marijuana, intravenous cocaine (arms and legs), and tobacco use. He was afebrile, alert, and in no apparent distress. His pulse rate was 67 beats/min, blood pressure was 154/92 mm Hg, respiratory rate was 16/min, and oxygen saturation was 97% on room air. There was no jugular venous distention. He had normal lung, cardiac, abdominal, and neurologic examinations. Serum electrolytes, creatinine, complete blood count, and chest x-ray were normal. Urine drug screen was positive for cocaine. Creatine phosphokinase was 261 U/L (reference range, 25-200 U/L), whereas troponin I was less than 0.5 ng/mL (normal). His electrocardiogram (ECG) at presentation showed deep inverted T waves in anterior precordial and inferior leads with no significant ST changes (ECG shown in Fig. 1, top panel). He was admitted with a diagnosis of unstable angina and was treated with aspirin, nitrates, and enoxaparin. An echocardiogram showed normal left ventricular systolic function. Right-sided chambers were normal. Pulmonary artery systolic pressure was moderately elevated at 46 mm Hg. A coronary angiogram was normal.

What is the diagnosis?

Discussion

The differential diagnoses of T-wave inversions are diverse and ischemia, inflammation, electrolyte abnormalities, cocaine use, trauma, and others.1 T-wave inversion can be normal in leads aVR, III, and V1. It may be seen in young adults (juvenile T-wave pattern). T-wave inversion is also seen in anxiety states, hyperventilation syndrome, and even with digoxin use.2

However, deep symmetrically inverted T waves (>5 mm), also called giant inverted T waves, are classically described in only a few conditions (Table 1).2

1. Apical hypertrophy (Yamaguchi syndrome): this variant of hypertrophic cardiomyopathy is characterized by myocardial hypertrophy localized to the apex of the left ventricle. Electrocardiogram shows giant inverted T waves in the midprecordial leads.3 Echocardiography/magnetic resonance imaging or computed tomography scan can confirm the diagnosis.

2. Raised intracranial pressure: prominent T waves are also seen in cerebrovascular accidents especially

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subarachnoid hemorrhage. T waves are diffuse with a widely splayed appearance with a prolonged QT interval. The proposed pathogenesis is cardiomyocytolysis from excessive sympathetic stimulation. Similar changes are reported after truncal vagotomy, bilateral carotid endarterectomy. 

3. Myocardial ischemia: deep T-wave inversions are described in subendocardial infarction and occur due to regional delay in ventricular repolarization. Deep inverted T waves in multiple precordial leads V1 through V4 are typically seen after transmural ischemia caused by high-grade stenosis of the proximal left anterior descending artery. Patients become asymptomatic by the time of presentation, and the ECG changes offer a clue to the critical diagnosis. T-wave inversions also occur after acute myocardial infarction and persist for days to weeks.

4. Posttachycardia and postpacemaker syndrome: T-wave inversions after supraventricular tachycardia or ventricular tachycardia were attributed to demand ischemia and may persist for days to weeks. However, cardiac memory involving the potassium ion channels is now considered the mechanism. T-wave inversions occur in bundle branch blocks and Wolff-Parkinson-White syndromes from secondary repolarization changes to the abnormal depolarization.

In the present case, the echocardiogram excluded apical hypertrophy. A normal coronary angiogram made ischemia much less likely, although vasospasm could not be entirely excluded. Also, intracranial pathology or tachycardia induced T-wave changes was unlikely. Our working diagnosis was transient ischemia from cocaine-induced vasospasm.

Giant T-wave inversions have rarely been associated with pulmonary embolism (PE). Pulmonary embolism was not our initial consideration as the patient had not been dyspneic at rest and was not tachycardic or hypoxic. Furthermore, deep inverted T waves are generally associated with large PE, which presents in a more dramatic fashion.

Due to the presence of moderate pulmonary hypertension and no other obvious etiology, a computed tomography angiogram was performed, which showed large bilateral thrombi that were partially occlusive in both main pulmonary arteries. Duplex scan showed left popliteal vein thrombus. A workup for hypercoagulable states was negative. Etiology of the thrombus was likely related to drug use. The patient was started on coumadin, and a week later, the ECG demonstrated resolving T-wave inversions.

Fig. 1. ECG on admission (top panel) and at 1 week (bottom panel).

Table 1
Conditions usually associated with deep symmetrically inverted T waves (>5 mm)

<table>
<thead>
<tr>
<th>Condition</th>
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<tr>
<td>Left ventricular apical hypertrophy</td>
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<td>Raised intracranial pressure</td>
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<tr>
<td>Myocardial ischemia</td>
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<td>Posttachycardia and postpacemaker T-wave pattern</td>
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<tr>
<td>Bundle branch blocks and Wolff-Parkinson-White syndrome</td>
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<td>Idiopathic global T-wave inversion syndrome</td>
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(ECG shown in Fig. 1, bottom panel). The ECG also demonstrates development of right ventricular hypertrophy as calculated by the Butler-Leggett formula.

**Giant inverted T waves in PE**

T-wave inversions in PE were first described in 1938. Ferrari et al.\(^9\) described ECG changes in 80 patients with massive PE. Anterior T-wave inversions were the most common (68%) and best correlated with severity of massive PE. Proposed mechanisms include right ventricular strain and decreased perfusion to the anterior myocardium from hypotension resulting from PE.\(^{10,11}\) More recently, Sarin et al.\(^{12}\) reported 2 cases of hemodynamically stable patients with moderate sized PE and inverted T waves (though they were not deep). This case illustrates the importance of including even moderate PE without hemodynamic instability in the differential diagnoses for deep T-wave inversions.

**Conclusions**

Giant inverted T waves are usually found in apical hypertrophy, posttachycardia syndrome, myocardial ischemia, and intracerebral hemorrhage. However, they may also occur with PE and should be considered in the differential diagnoses.

**References**