

# Rare Giant T-Wave Inversions Associated With Myocardial Stunning: Report of 2 Cases

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**Abstract:** Prominent T-wave inversions are well recognized electrocardiographic signs that can occur in acute myocardial infarction (AMI). However, the giant negative T waves may be associated with myocardial stunning without AMI.

This case report describes 2 patients without AMI who developed rare giant T-wave inversions measuring up to 35 mm in depth and QT prolongation after admission to hospital. While 1 patient presented with acute pulmonary edema, the other patient presented with severe chest pain at rest and transient ST elevation.

The giant T-wave inversion with QT prolongation may be caused by myocardial stunning due to the triple vessel diseases and elevated wall stress, high-end diastolic pressure and decreased coronary arterial flow during pulmonary edema in the first patient. The giant T-wave inversion with QT prolongation in the second patient may be caused by myocardial stunning due to the left anterior descending artery spasm (transient ST elevation) leading to transient total occlusion of left anterior descending artery. Percutaneous coronary intervention was successfully undergone for both patients. The patients remained well.

The electrophysiologic mechanism responsible for giant T-wave inversion with QT prolongation is presently unknown. The two cases demonstrate that the rare giant negative T waves may be associated with myocardial stunning without AMI.

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**Abbreviations:** ECG = electrocardiogram, GNT = giant negative T waves, LQTS = long QT syndromes, AMI = acute myocardial infarction, CT = computed tomography, LVEF = left ventricular ejection fraction.

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Ethical approval was obtained from the ethics committee of the Second Affiliated Hospital of Wenzhou Medical University. Written informed consent was obtained from the patients for publication of this case report and any accompanying images.

The authors have no conflicts of interest to disclose.

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## INTRODUCTION

The T wave represents the period of ventricular repolarization on the surface electrocardiogram (ECG) that occurs during ventricular diastole.<sup>1</sup> Abnormalities of the T wave on the ECG often provide valuable clues to underlying pathology in cardiac illness as well as systemic syndromes. Giant negative T waves (GNTs) defined in 1979 by Yamaguchi et al<sup>2</sup> as negative T waves with greater than 10 mm (1 mV) amplitude have been associated with a variety of clinical conditions, including coronary artery disease, hypertrophic cardiomyopathy, left or right ventricular hypertrophy with strain, cerebrovascular accidents, and long QT syndromes (LQTS).<sup>3-6</sup> Acute myocardial infarction (AMI) is the most common condition associated with GNT in the ECG. We describe two patients who developed rare giant T-wave inversions measuring up to 35 mm (35 mV) in depth and QT prolongation without AMI after admission to hospital.

## CASE PRESENTATION

### Case 1

A 77-year-old Chinese man was admitted to our hospital because of shortness of breath accompanied by diaphoresis during sleep for 5 hours. His past medical history was remarkable for hypertension diagnosed 10 years ago. Previous 12 lead ECG done in the patient with no symptoms for a routine health maintenance examination 4 months ago showed sinus arrhythmia and left ventricular hypertrophy and asymmetrically inverted T waves (leads I, II, AVL, V4-V6) and normal QT and QTc intervals (Figure 1A). The patient did not obtain follow-up care, and has not been on antihypertensive medication. The family history was unremarkable for any acute or chronic medical problems. He had no drug allergies and no history of past surgeries, smoking, and drinking.

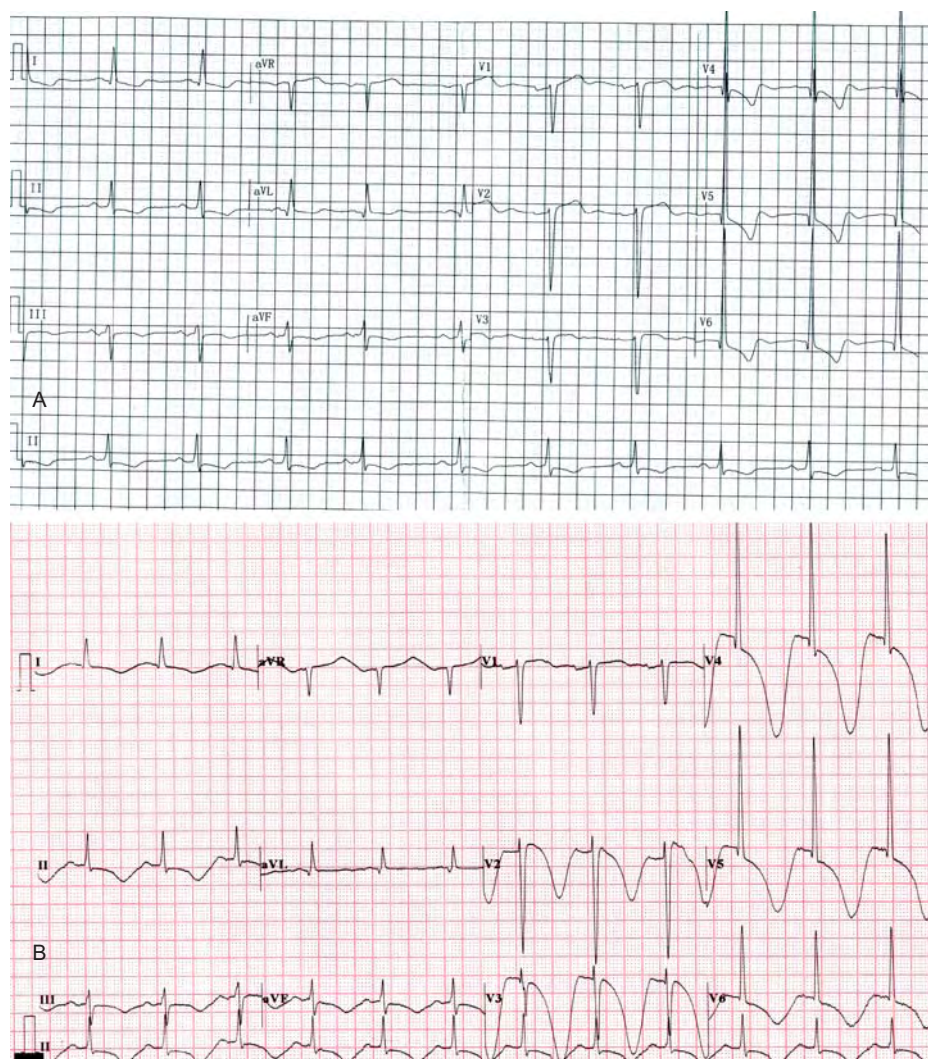
Upon arrival at the hospital, his blood pressure was 236/147 mmHg. His heart rate was regular at 110 beats/min and his respiratory rate was 26 breaths/min. The patient could not lie flat because of orthopnea. The jugular venous pressure was elevated, the apex beat was deviated slightly to the left side and there was no heart murmur, but there were bilaterally basal pulmonary rales. Findings of abdominal and neurological examinations were unremarkable, and lower extremities were without edema.

Initial investigations revealed that levels of complete blood cell count, serum electrolytes, glucose, and blood creatinine were normal. An arterial blood gas test showed a PH of 7.238 and an arterial oxygen pressure of 61.6 mmHg and a carbon dioxide pressure of 49 mmHg while the patient was breathing 5 L of oxygen by face mask. Amino terminal proBNP is 5663 pg/mL (normal: 0-525 pg/mL). Initial troponin I levels were 0.06 ng/mL (reference, <0.1) 5 hours after symptom onset. The admission ECG revealed that T waves were inverted in all leads except aVL, aVR and V1, and the

QT interval (611ms with a QTc of 629ms) was markedly prolonged (Figure 1B), which became prominent in next 10 minutes, with very huge negative T-wave inversion with QT interval prolongation in leads I, II, III, aVF, and V2 through V6, reaching a depth of 35 mm below the isoelectric line in lead V4 (Figure 1C). A transthoracic echocardiography demonstrated a slightly dilated left ventricle (left ventricular end-diastolic internal diameter of 59mm) with a left ventricular ejection fraction (LVEF) of 0.40, thickened interventricular septal wall (14mm), normal left ventricular posterior wall thickness (11mm), and severe hypokinesia of the left ventricular posteroinferior wall and posterior septum. A computed tomography (CT) of the brain showed no evidence of infarction or hemorrhage. A CT of the chest

showed pulmonary edema with small bilateral pleural effusion. A diagnosis of acute heart failure due to coronary artery disease and hypertensive heart disease was made. The patient was administered morphine and furosemide intravenously and nitroglycerin drip, and received aspirin and clopidogrel. The patient symptomatically improved and was hemodynamically stable 2 hours after the treatment, whereas the ECG had no remarkable change compared with the ECG done 10 minutes after the admission.

Serial ECG (Figure 1D–F) recordings were obtained. By hospital day 3, the ECG (Figure 1C) demonstrated improved T-wave inversion with prolonged albeit improved QT intervals (QTc, 470ms). The T-wave inversion lasted for 10 days with gradual attenuation and eventually returned to



**FIGURE 1.** Serial ECGs demonstrating the development and recovery of very giant T-wave inversions and extreme QT prolongation. A. The prior ECG done in the patient with no symptoms for a routine health maintenance examination 4 months ago showed sinus arrhythmia and left ventricular hypertrophy and asymmetrically inverted T waves (leads I, II, AVL, V4–V6) and normal QT and QTc intervals. B. The admission ECG revealed that T waves were inverted in all leads except aVL, aVR and V1, and the QT interval (611 ms with a QTc of 629ms) was markedly prolonged. C. ECG 10 minutes after admission revealed very huge negative T-wave inversion in leads I, II, III, aVF and V2 through V6, reaching a depth of 35 mm below the isoelectric line in lead V4. D. ECG on hospital day 3 showed the T-wave inversion with gradual attenuation. E. ECG on hospital day 4 showed the T-wave inversion became smaller. F. ECG on hospital day 10 the T-wave inversion eventually returned to near baseline values as prior ECG in the patient with no symptoms.

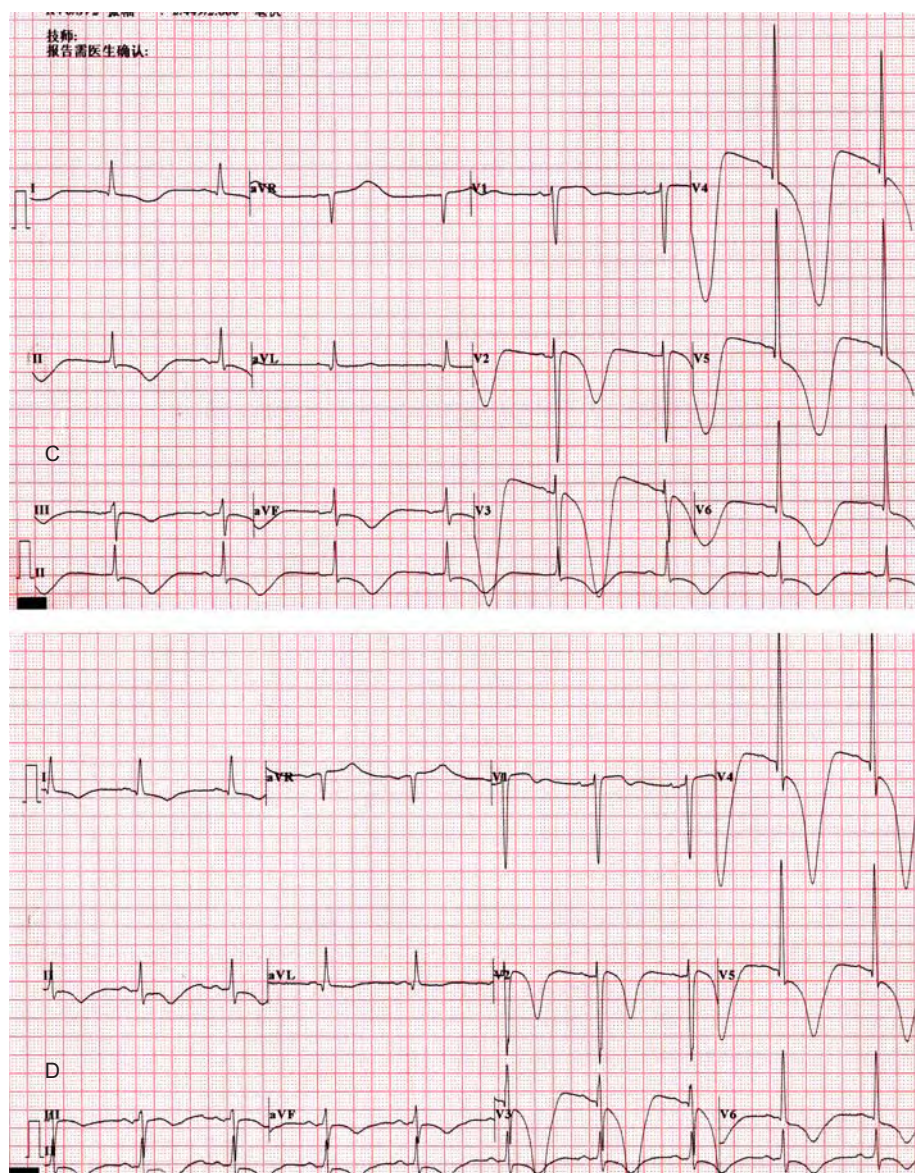


FIGURE 1. Continued

near-baseline values (Figure 1F). By hospital day 11, the transthoracic echocardiography demonstrated an improved LVEF of 0.52. Troponin I levels drawn were not consistent with myocardial infarction. The patient's peak troponin level was 0.23 ng/mL (reference, <0.1). Creatine kinase-MB was not increased during this course. By hospital day 6, the patient was taken to cardiac catheterization that revealed a normal left main coronary artery, a long tubular stenosis of approximately 70% to 80% in the mid-to-distal anterior descending artery, a 60% stenosis in the second diagonal branch of coronary artery, a 95% stenosis at the proximal obtuse marginal branch (a branch of the circumflex artery), a 60% stenosis in the distal right coronary artery, and a 99% stenosis of the posterior descending coronary artery with TIMI 2 flow. The hypokinetic area in the echocardiography corresponded to the anatomical distribution of coronary arteries. The patient successfully underwent percutaneous

coronary intervention in the distal right coronary artery and left anterior descending coronary artery. He was discharged on medication for heart failure and coronary artery disease and hypertension. The patient remained well. The ECG was basically similar to prior ECG in the patient with no symptoms; the huge negative T waves disappeared and the transthoracic echocardiography demonstrated an improved LVEF of 0.59 after 1 month.

### Case 2

A 68-year-old man with a preserved exercise capacity was admitted to our hospital because of a 10-days history of short-lasting chest pain that occurred exclusively at night. He had a history of smoking for 40 years. Physical examination on admission was unremarkable, except that the heart rate was very slow (44 beats/min). A 12-lead ECG recorded on

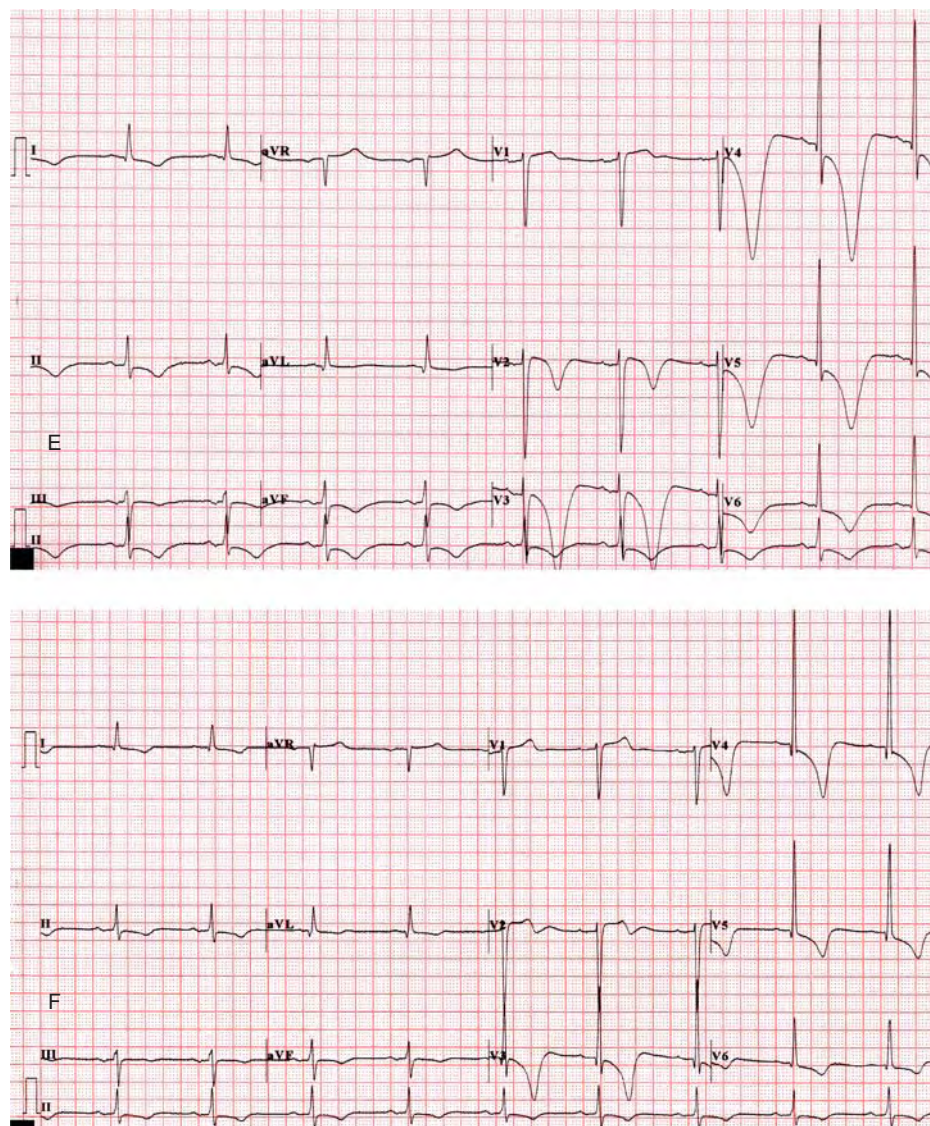


FIGURE 1. Continued

admission in the patient with no symptoms showed sinus bradycardia and left ventricular high voltage ( $Rv5 + Sv1 = 4.9\text{mv}$ ) and normal T waves and normal QTc intervals (Figure 2A). Initial troponin I, brain natriuretic peptide, transthoracic echocardiography, and chest x-ray were normal. The patient was administered aspirin and clopidogrel prepared for a coronary artery angiography.

Later that evening, he was woken because of severe chest pain accompanied by diaphoresis during sleep. The ECG monitor showed marked ST segment elevation followed by polymorphic ventricular tachycardia, which quickly reverted to sinus rhythm. The 12-lead ECG taken just after the episode of pain showed a sinus rate of 48 beats/min with ST segment elevations in leads V1–V5, most prominently in leads V3–V4 (Figure 2B). The chest pain resolved with 1 sublingual nitroglycerine tablet within 1 minute. The episode of ST-segment elevation lasted for about 5 minutes. A diagnosis of variant angina was made. Subsequent serial ECGs were obtained (Figure 2C–F). The ECG (Figure 2C) done 6 hours

after symptom onset revealed markedly inverted T waves in leads I, aVL, and V4–V6, biphasic T waves in leads V1–V3, and a prolonged QT interval (590ms with a QTc of 506ms) (Figure 2C), which became prominent in the next 3 hours, with very GNT inversion with QT interval prolongation in leads V3–V5, reaching a depth of 35mm below the isoelectric line in lead V4 (Figure 2D). By hospital day 3, the ECG (Figure 2E) demonstrated improved T-wave inversion with normal QT intervals. The T-wave inversion lasted for 8 days with gradual attenuation and eventually returned to near baseline values (Figure 2F). Troponin I levels drawn were not consistent with myocardial infarction. The patient's peak troponin level was 0.21 ng/mL (reference,  $<0.1$ ). Creatine kinase-MB was not increased during this course. By hospital day 3, the patient was taken to cardiac catheterization, which revealed a normal left main coronary artery, a tubular atherosclerotic stenosis of approximately 85% to 90% in the proximal-to-mid left anterior descending artery, a 30% stenosis in the distal right coronary artery, and a normal circumflex

artery (Figure 2G). The fixed stenosis of left anterior descending artery demonstrated by coronary angiography corresponded to the ST elevations in leads V1–V5 in the patient with chest pain. Percutaneous coronary intervention was successfully undergone in the left anterior descending artery (Figure 2H). He was discharged on medication for coronary artery disease and variant angina. During the 10-month follow-up, the patient remained clinically free of symptoms.

## DISCUSSION

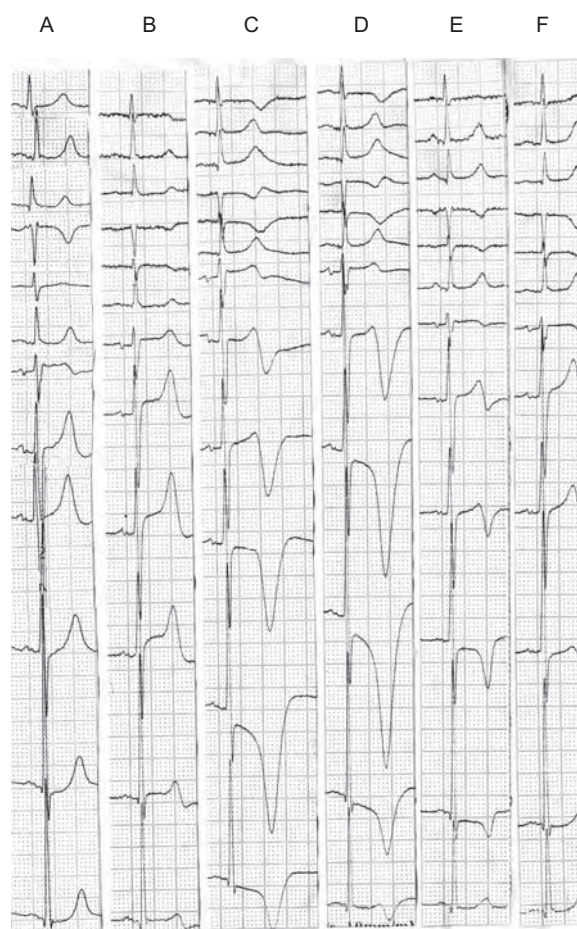
The giant T-wave inversion appears as a manifestation of ventricular repolarization abnormalities, and are associated with various clinical conditions such as myocardial infarction, pericarditis, hypertrophic cardiomyopathy, central nervous system diseases, electrolyte imbalance (potassium or calcium deficiency), LQTS, or drug effects.<sup>3–7</sup> In the present 2 cases, AMI was unlikely because of the absence of QRS changes, and a lack of remarkable change in troponin I levels. Pericarditis, hypertrophic cardiomyopathy, cerebral vascular accident, and electrolyte imbalance were ruled out because of noninflammatory findings and absence of asymmetrical ventricular hypertrophy on echocardiography and normal findings on head CT and electrolytes. LQTS can be congenital or acquired that can occur with drugs, acquired cardiac conditions, and electrolyte imbalance.<sup>8</sup> In our patients, congenital, drug-induced or electrolyte imbalance induced LQTS were unlikely because of the prolonged QT intervals and GNT normalized concomitantly with improvement of myocardial dysfunction and ischemia; the 2 patients had taken no drugs known to cause T wave and QT interval changes, and electrolyte levels were also normal. The appearance of giant T-wave inversions and prolonged QT intervals in the 2 patients was probably associated with reversibly ischemic myocardial stunning without AMI because troponin I was only slightly elevated and cardiac catheterization revealed significant stenoses of coronary arteries.

Prominent T-wave inversions and QT prolongation are well recognized electrocardiographic signs that can occur in AMI. However, our cases demonstrate rare GNTs associated with myocardial stunning without AMI. Myocardial stunning is a well-defined phenomenon of postischemic myocardial dysfunction, which is a result of an acute ischemic insult.<sup>9</sup> The giant T-wave inversion with QT prolongation may be caused by myocardial stunning due to the triple vessel diseases and elevated wall stress, high-end diastolic pressure and decreased coronary arterial flow during pulmonary edema in the first patient. The key drugs for acute management of acute pulmonary edema are oxygen, diuretics, and vasodilators.<sup>10</sup> Opiates and inotropes are used more selectively, and mechanical support of the circulation is required only rarely.<sup>10</sup> The patient (Case 1) improved symptomatically and was hemodynamically stable 2 hours after administering nitroglycerin drip and intravenously administering morphine and furosemide. The giant T-wave inversion with QT prolongation in the second patient may be caused by myocardial stunning due to the left anterior descending artery spasm (The transient ST elevation in leads V1–V5 was found; Figure 2) leading to transient total occlusion of left anterior descending artery. Reperfusion after brief myocardial ischemia does not induce necrosis and results in prolonged but reversible contractile dysfunction.

Besides myocardial ischemia, the more recently described stress also could induce myocardial stunning via myocardial adrenoceptors.<sup>11</sup> T-wave changes in the 2 patients

with acute pulmonary edema and coronary artery spasm, respectively, were also caused by an acute rise in the cardiac sympathetic tone with a local excess of norepinephrine. The surges of norepinephrine and sympathetic outflow in the patients instigate not only the electrical abnormalities but also can lead to physical myocardial damage that maintains the T-waves inversion for several days.

It has been demonstrated that the myocardium is composed of 3 electrical layers: the endocardium, the epicardium, and the M-cell layer located within the midmyocardium.<sup>12</sup> Each of these layers has distinct electrical



**FIGURE 2.** Serial 12-lead ECGs demonstrating the development and recovery of very giant T-wave inversions and coronary angiography. A. The admission ECG was done in the patient with no symptoms. B. The ECG done in the patient with chest pain revealed ST segment elevations in leads V1–V5, most prominently in leads V3–V4. C. The ECG done 6 hours after symptom onset revealed markedly inverted T waves in leads I, aVL, and V4–V6, biphasic T waves in leads V1–V3. D. The ECG done 9 hours after symptom onset revealed very giant negative T-wave inversion in leads V3–V5, reaching a depth of 35 mm below the isoelectric line in lead V4. E. ECG on hospital day 3 showed the T-wave inversion with gradual attenuation. F. ECG on hospital day 8 the T-wave inversion eventually returned to near baseline values as the admission ECG. G. Coronary angiography showing a severe stenosis of approximately 85% to 90% in the proximal to mid left anterior descending artery (right anterior oblique projection). H. Repeat left coronary angiography following successful stenting.

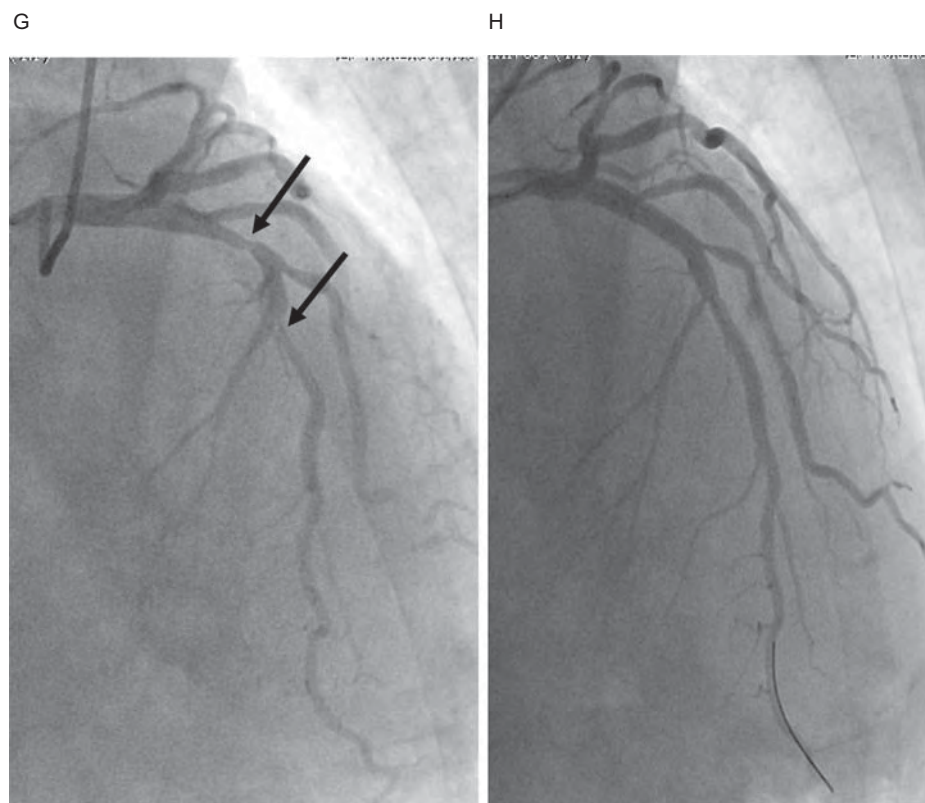


FIGURE 2. Continued

properties and a different action potential. The M cell exhibits a significantly longer action potential duration than the epicardial and endocardial cell types and coincides with the end of the T wave. It has been hypothesized that when myocardial infarction destroys endocardium, the long action potential of the M-cell layer can dominate the ECG, producing a very long QT interval.<sup>13</sup> Therefore, it is tempting to speculate that the ECG findings of markedly inverted T waves with a prolonged QT interval in our patients are thought to reflect ischemic stunning of the subendocardium.

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