

## CASE REPORT

### Isolated deep T-wave inversion on an electrocardiogram with normal wall motion

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## Introduction

Determining the cause of deep T-wave inversion is a diagnostic challenge [1]. Although differential diagnosis of deep T-wave inversion includes life-threatening conditions, such as myocardial infarction and stroke, even a thorough workup may not reveal its etiology [2]. Deep T-wave inversion is commonly associated with takotsubo cardiomyopathy or takotsubo-like myocardial dysfunction [3], but this cannot be diagnosed without wall motion abnormalities [4]. We experienced an otherwise healthy elderly woman in whom deep T-wave inversion of unknown origin was incidentally found and who developed typical takotsubo cardiomyopathy 10 years later. Our findings provide evidence that isolated deep T-wave inversion is an after-effect of takotsubo cardiomyopathy.

## Case Report

In 2003, 10 years before the present admission, the electrocardiogram (ECG) of a 73-year-old healthy woman showed deep T-wave inversion during an annual health check. This finding raised suspicion of myocardial infarction. She had

#### Key Clinical Message

The electrocardiogram (ECG) of a 73-year-old, asymptomatic woman showed deep T-wave inversion. The complete workup was negative. Ten years later, she developed takotsubo cardiomyopathy with abnormal ECG again. Isolated deep T-wave inversion might be an aftereffect of takotsubo cardiomyopathy that does not warrant an invasive workup.

#### Keywords

Cardiology, deep T-wave inversion, takotsubo cardiomyopathy.

been in good health without physical or emotional stress. She recalled no chest symptoms, and her ECG at a health check 6 months previously was negative. She had no history of hypertension, diabetes mellitus, or dyslipidemia. She never smoked, but drank a 350-mL can of beer daily. She appeared well on admission. Her vital signs were normal, except for a blood pressure of 178/80 mmHg. Her weight was 44 kg and her height was 146 cm. A general physical examination showed no abnormalities. Laboratory tests, including cardiac enzymes, were all within normal limits. The ECG showed negative T waves in leads II, III, aV<sub>F</sub>, and V<sub>2-6</sub> (Fig. 1A). Echocardiography showed no decline in wall motion and no morphological abnormalities. Coronary angiography did not show any coronary artery stenosis. Left ventriculography showed normal wall motion with an ejection fraction of 82%. She was discharged on day 2. On reexamination 9 days after discharge, the T-wave inversion was much shallower (Fig. 1B), and 3 months later, it had disappeared (Fig. 1C). We treated the hypertension with candesartan cilexetil, and 6 months later, her blood pressure was under control. In 2013, 10 years later, after hearing of the death of her grandchild, she became short of breath and experienced chest pain. On

admission 7 h after the onset of symptoms, she was still in acute distress, although she appeared alert and well oriented. Her vital signs were normal, with a blood pressure of 134/76 mmHg. Physical examination showed jugular venous distension and systolic murmur at the cardiac apex.

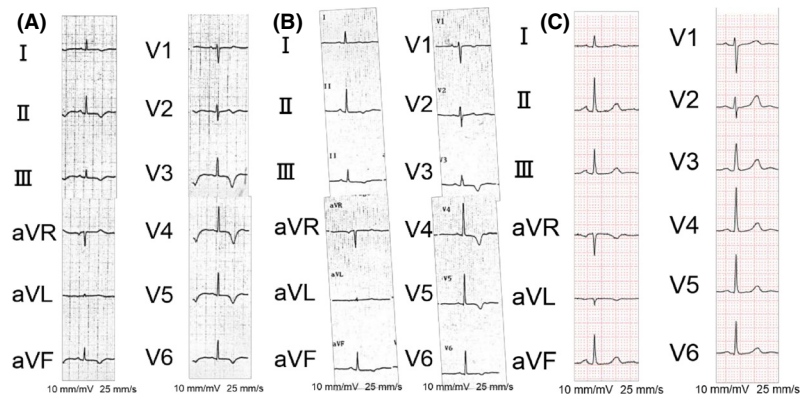
The creatine kinase level was 499 U/L (reference: 40–150 U/L), creatine kinase MB isoenzymes were 51.2 ng/mL (reference: 0.0–6.9 ng/mL), and the creatine kinase isoenzyme index was 10.7% (reference: 0–3.5%). Troponin T was positive. ECG on admission showed a normal sinus rhythm with ST-segment elevation in leads II, III, aV<sub>F</sub>, and V<sub>2–6</sub> (Fig. 2A). Chest radiography showed congested lungs, mild cardiomegaly, and bilateral pleural effusion. Transthoracic echocardiography indicated severe segmental left ventricular dysfunction involving the septum, anteroseptal territory, and apex, with grade two mitral regurgitation. The estimated left ventricular ejection fraction was 40%. Emergent coronary angiography

showed normal coronary arteries. Left ventriculography revealed akinesis of the apical wall and compensatory hyperkinesis of the basal walls (Fig. 3A and B).

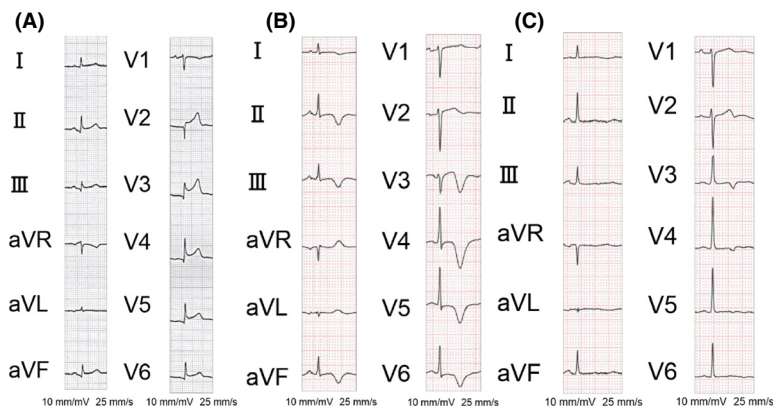
Acute myocardial infarction was ruled out by coronary angiography. Blood tests showed an elevation in norepinephrine and dopamine levels. However, 24-h urine tests showed no elevation in metanephrine or normetanephrine levels, making a diagnosis of pheochromocytoma less probable. Brain computed tomography showed no traumatic changes or hemorrhage. Blood tests, echocardiography, and left ventriculography results excluded myocarditis and hypertrophic cardiomyopathy. Finally, we diagnosed the patient with takotsubo cardiomyopathy.

Oxygen, furosemide, candesartan cilexetil, and heparin were administered on day 1.

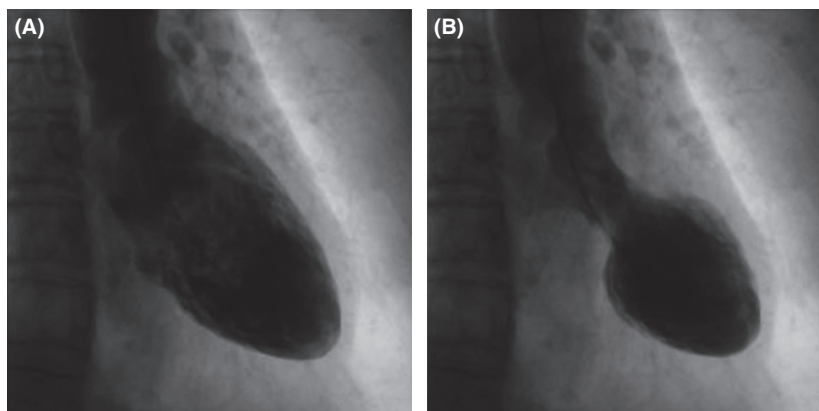
A transthoracic echocardiogram on day 10 showed mild improvement of global wall motion. On day 11, she had become asymptomatic, although the ECG showed



**Figure 1.** Electrocardiograms (ECGs) during and after the first admission to hospital. (A) ECG on day 1 shows negative T waves in leads II, III, aV<sub>F</sub>, and V<sub>2–6</sub>. Note the deeply inverted T waves (7 mm) in leads V<sub>3–4</sub>. (B) ECG on day 11. T-wave inversion has become much shallower. (C) ECG 3 months after discharge. T-wave inversion has disappeared.



**Figure 2.** ECGs during and after the second admission to hospital. (A) ECG on day 1 shows a normal sinus rhythm with ST-segment elevation in leads II, III, aV<sub>F</sub>, and V<sub>2–6</sub>. (B) ECG on day 11 shows deeply inverted T waves in leads II, III, aV<sub>F</sub>, and V<sub>3–6</sub>. (C) ECG 3 months after the onset shows normalization of ST-T changes.



**Figure 3.** Left ventriculography (right anterior oblique view) during the second admission to hospital. Diastolic (A) and systolic (B) phases show akinesis of the apical wall and compensatory hyperkinesis of the basal walls.

deeply inverted T waves in leads II, III, aV<sub>F</sub>, and V<sub>3-6</sub> (Fig. 2B), similar to those observed at the first admission (Fig. 1A). Her general condition had stabilized, and she was discharged on day 14. The ECG obtained 3 months after the onset of symptoms was nearly normal (Fig. 2C).

## Discussion

In 1998, well before takotsubo cardiomyopathy was generally recognized, Hansoti and Dharani [2] reported 10 previously healthy patients (nine women) with idiopathic isolated global T-wave inversion, similar to that observed in the present case. Aside from negative echocardiography and left ventriculography, the clinical features of female predominance, 40–60 years of age, acute chest pain or discomfort, normal creatine kinase MB isoenzymes, a negative coronary angiogram, and a good prognosis are compatible with the diagnosis of takotsubo cardiomyopathy. Therefore, the isolated deep T-wave inversion observed during the first episode in our patient was most likely an after-effect of takotsubo cardiomyopathy. This conclusion is based on the fact that no other cause was identified, and the ECG findings at the second admission to hospital were similar to those associated with typical recurrent takotsubo cardiomyopathy [5]. In typical cases of takotsubo cardiomyopathy, ST-segment elevation on the ECG and abnormal wall motion on echocardiography are observed at the onset. These events are followed by T-wave inversion, which persists after normalization of echocardiographic images [6], as observed during the first episode in our patient. Most patients with takotsubo cardiomyopathy complain of chest pain and dyspnea, although some are asymptomatic [7]. In our patient, asymptomatic takotsubo cardiomyopathy may have been present before the periodic health examination. In conclusion, when clinicians encounter isolated deep T-wave

inversion, they should suspect that it is an after-effect of takotsubo cardiomyopathy because the generally favorable prognosis does not warrant an invasive workup, such as coronary angiography.

## Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

## Conflict of Interest

The authors declare no competing interest.

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