

## Case Report

## Broken Heart During Treadmill Exercise Testing: An Unusual Cause of ST-Segment Elevation

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**Takotsubo cardiomyopathy, treadmill exercise testing, ST-segment elevation.**

Treadmill exercise testing is a commonly used diagnostic test for the assessment of chest discomfort and exercise-induced arrhythmias. The presence of ST-segment elevation during exercise is considered a marker of severe ischaemia, usually secondary to a critical lesion in a proximal coronary artery. We present a novel cause of ST-segment elevation during exercise testing: “broken heart syndrome”, also known as transient left ventricular apical ballooning syndrome, or takotsubo cardiomyopathy. Takotsubo cardiomyopathy is a rare, yet well-described, reversible cardiomyopathy triggered by profound psychological or physical stress. The exact aetiology of takotsubo cardiomyopathy is still unknown. However, the occurrence of takotsubo cardiomyopathy during exercise in this case report is in keeping with the sudden catecholamine surge secondary to treadmill exercise testing, which leads to abnormal ventricular contraction and contributes to wall motion abnormalities. Further studies are needed to elucidate the pathogenesis of the disease and consequently determine specific preventive therapies.

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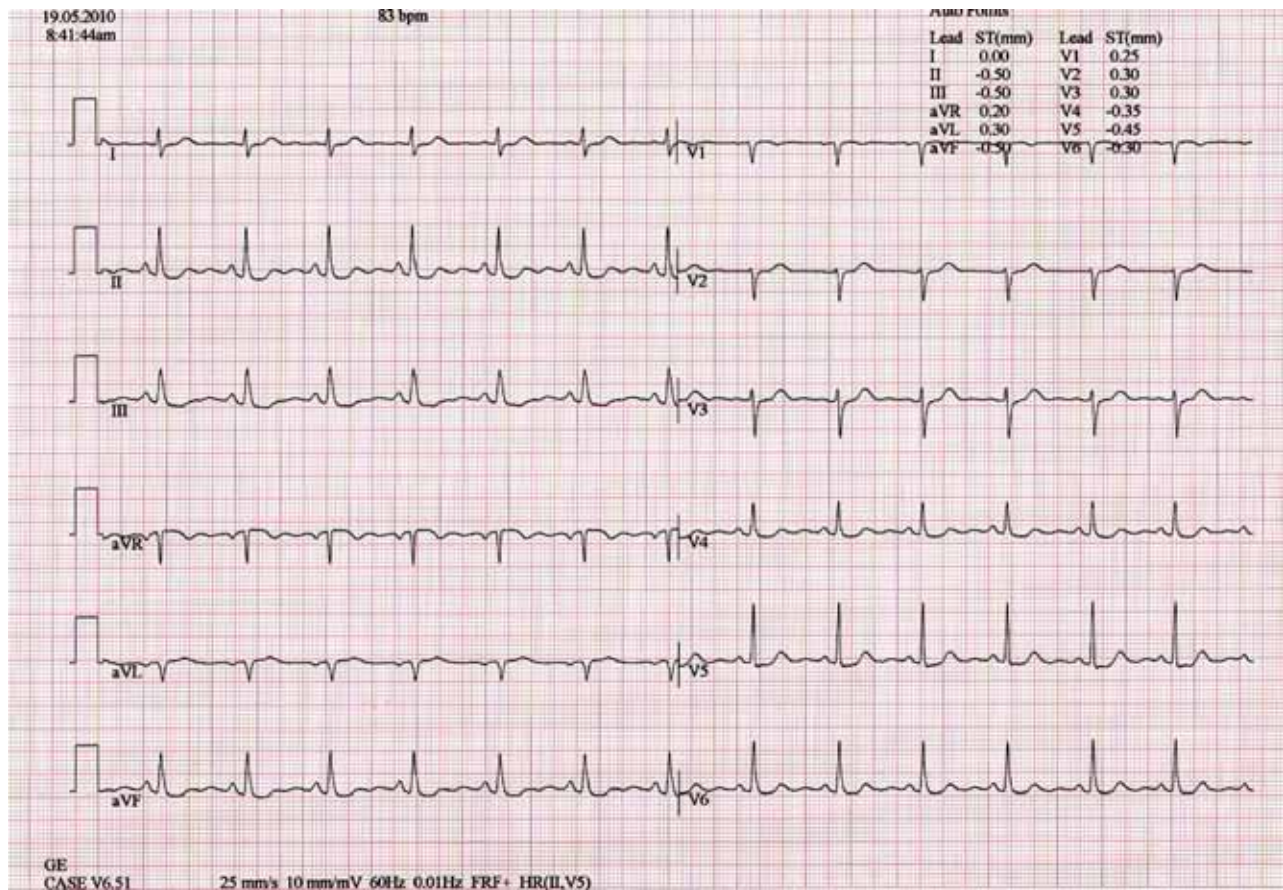
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**T**readmill exercise testing is a commonly used diagnostic test for the assessment of chest pain and exercise-induced arrhythmias. It is known to cause a hypersympathetic state, leading to catecholamine excess. The following case report describes a novel cause of ST-segment elevation during exercise testing: the “broken heart syndrome”.

### Case presentation

A 69-year-old Caucasian lady, known to suffer from hypertension, dyslipidaemia and anxiety, underwent treadmill exercise testing with the standard Bruce protocol for evaluation of chest pain. She described a few months’ history of infrequent dull retrosternal discomfort, radiating to the back, with associated palpitations, occurring mainly at rest and resolving after few minutes. The physical examination and resting electrocardiogram were nor-

mal (Figure 1). During exercise testing, she had achieved 3 metabolic equivalents when she developed similar retrosternal discomfort, accompanied by development of Q waves in leads V<sub>1</sub> and V<sub>2</sub>, ST-segment elevation in leads V<sub>2</sub>, V<sub>3</sub>, I and aVL, and ST-segment depression in III, aVF, V<sub>4</sub>-V<sub>6</sub> (Figure 2) that persisted on recovery. There was a 10 mmHg rise in systolic and diastolic blood pressure during exercise testing (from 140/80 to 150/90 mmHg). The patient was started on intravenous nitrates and heparin, based on the presumed diagnosis of acute coronary syndrome. A coronary angiogram was performed within less than an hour. The ventriculogram revealed apical akinesia and anterior hypokinesia, and mild impairment of global systolic function (Figures 3 & 4); however, the coronary arteries were angiographically normal. This was accompanied by a rise in cardiac markers (troponin I 9.7 ng/ml, creatinine kinase 375 u/L).



**Figure 1.** Resting electrocardiogram showing normal sinus rhythm.

The patient did well on conservative treatment and the ECG changes resolved within less than 24 hours. An echocardiogram after 48 hours revealed anterior and apical hypokinesia (Figures 5 & 6); these changes resolved on repeat echocardiography performed a month later. Consequently, our patient was diagnosed as having had takotsubo cardiomyopathy as a result of treadmill exercise testing.

## Discussion

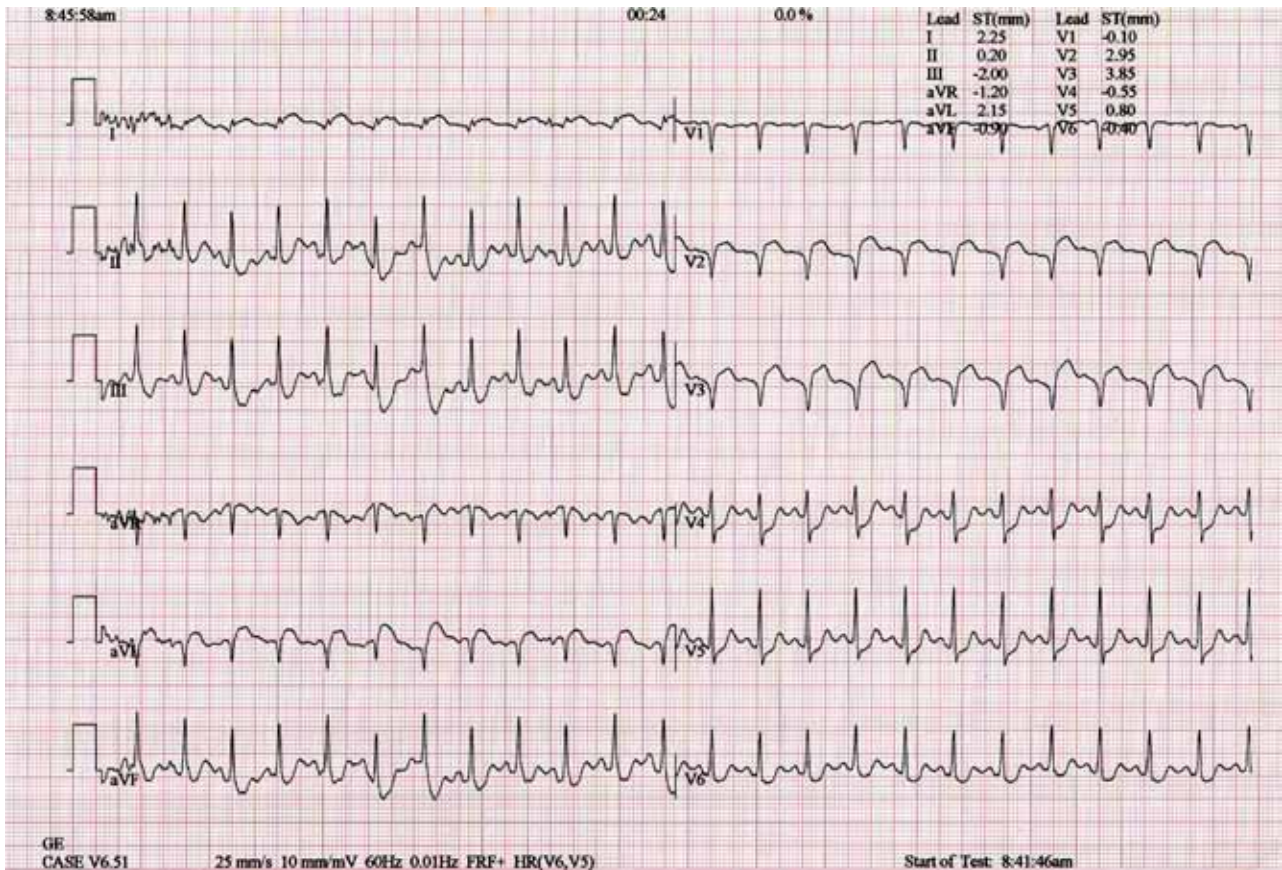
Takotsubo cardiomyopathy, also known as transient left ventricular apical ballooning syndrome, stress-induced cardiomyopathy and “broken heart syndrome”, is a rare, yet well-described, reversible cardiomyopathy triggered by profound psychological or physical stress. It is typically characterized by acute onset of ischaemic-like chest pain or dyspnoea; transient apical and mid-ventricular regional wall motion abnormalities, with the regional wall motion abnormalities extending beyond a single epicardial vascular distribution; minor elevation of cardiac biomarkers; dy-

namic electrocardiographic changes; and the absence of epicardial coronary artery disease.<sup>1</sup> The above-mentioned criteria were fulfilled by our patient.

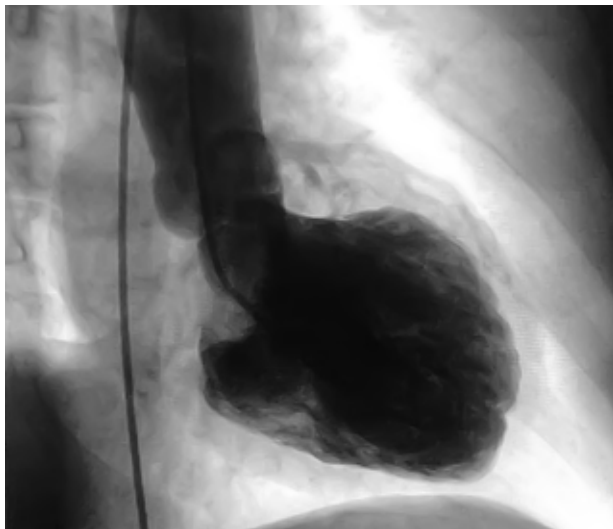
To the authors’ knowledge, this is the first reported cause of ST-segment elevation during exercise testing secondary to takotsubo cardiomyopathy. ST elevation during exercise is considered a marker of severe ischaemia, usually due to critical proximal coronary artery stenosis.<sup>2</sup> Occasionally, it may be due to coronary artery spasm in subjects with coronary artery disease (CAD) who do not have significant fixed stenosis.<sup>3</sup> Bruce et al<sup>4</sup> have demonstrated that areas of dyskinesia or aneurysm in subjects with CAD can also produce ST-segment elevation during exercise in the absence of severe ischaemia. There is only one published case report describing a positive exercise test secondary to takotsubo cardiomyopathy.<sup>5</sup> However, in that report, apical dyskinesia was associated with ST-segment depression rather than elevation.

The exact aetiology of takotsubo cardiomyopathy is still unknown, but several theories have been proposed, including multivessel coronary artery spasm,

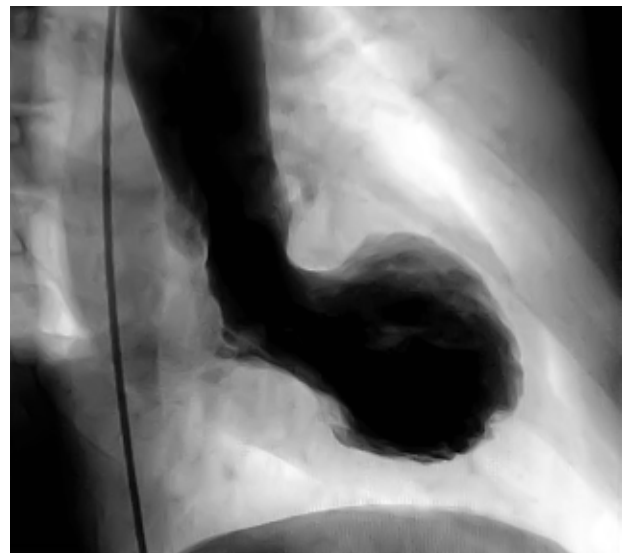




**Figure 2.** Electrocardiogram after 4 minutes exercise showing Q waves in leads V<sub>1</sub> and V<sub>2</sub>, ST-segment elevation in leads V<sub>2</sub>, V<sub>3</sub>, I and aVL, and ST-segment depression in III, aVF, V<sub>4</sub>-V<sub>6</sub>.



**Figure 3.** Ventriculogram at end-diastole.



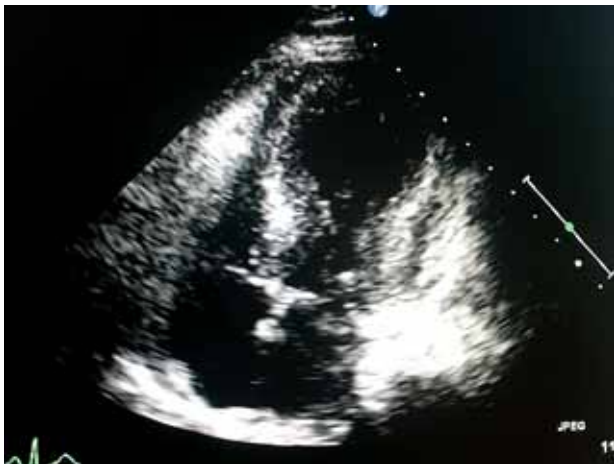
**Figure 4.** Ventriculogram at end-systole showing typical apical ballooning characteristic of takotsubo cardiomyopathy.

impaired cardiac microvascular function, impaired myocardial fatty acid metabolism, acute coronary syndrome with reperfusion injury, and endogenous

catecholamine-induced myocardial stunning and microinfarction.<sup>6</sup> Dhoble et al<sup>7</sup> suggest that a relatively transient hypertensive response during exercise stress



**Figure 5.** Echocardiogram at end-diastole.



**Figure 6.** Echocardiogram at end-systole showing apical hypokinesia.

may give rise to altered myocardial contractility, resulting in false positive results. They reported two patients experiencing apical and basal akinesia during exercise stress echocardiography; both showed a pathological hypertensive response during exercise testing but no significant ECG changes were noted. However, in our case no significant hypertensive response was noted, in spite of significant ECG changes. Our findings are thus more in keeping with the sudden catecholamine surge secondary to treadmill exercise testing, which leads to abnormal ventricular contraction and contributes to wall motion abnormalities. The role of catecholamines in the pathophysiology of takotsubo cardiomyopathy is supported

by structural and ultrastructural myocardial changes that are suggestive of direct catecholamine toxicity, together with increased local and systemic catecholamine levels in subjects with takotsubo cardiomyopathy, as reviewed by Koulouris et al.<sup>8</sup>

In conclusion, we report a novel cause of ST-segment elevation during exercise testing. Our case represented a vulnerable individual, known to suffer from anxiety, undergoing an exercise test, which exposed the patient to both physical and psychological stress. Nonetheless, the aetiology of takotsubo cardiomyopathy remains puzzling and further studies are needed to elucidate the pathogenesis of the disease and to determine specific preventive therapies. Our case also suggests that takotsubo cardiomyopathy should be considered in the differential diagnosis of ST-segment elevation following exercise stress testing.

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