

Acute Coronary Syndrome versus Takotsubo Cardiomyopathy: A Diagnostic Dilemma

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Abstract

We describe a patient presenting with ischemic ECG changes and troponin elevation after a tonic-clonic seizure, with LV dysfunction on echocardiography characteristic of takotsubo cardiomyopathy. Coronary angiography was initially delayed, but later showed obstructive CAD in the LAD and RCA. Cardiac MRI helped to clarify the observed regional wall motion abnormalities as likely due to ischemia rather than takotsubo cardiomyopathy. This case highlights the diagnostic uncertainty in cases where takotsubo cardiomyopathy coexists with obstructive CAD, and the growing role of cardiac MR in cases of diagnostic uncertainty.

Keywords: Takotsubo; Cardiomyopathy; Acute myocardial infarction; Cardiac magnetic resonance imaging

Introduction

Takotsubo cardiomyopathy (TTC) is characterized by transient left ventricular dysfunction and is classically associated with apical ballooning seen on echocardiography [1,2]. It can be associated with emotional or physical stressors. It is often difficult to differentiate takotsubo cardiomyopathy from acute coronary syndrome based on symptoms, ECG findings and biomarkers alone. Cardiac MRI is not routinely used in the diagnosis of TTC but has been described as a modality that can help to differentiate TTC from acute myocardial infarction (MI) [3]. We report a case of a patient presenting with LV dysfunction characteristic of takotsubo cardiomyopathy after a tonic-clonic seizure. Coronary angiography revealed obstructive CAD in the LAD and RCA and findings on cardiac MRI attributed the wall motion abnormalities to acute ischemia rather than takotsubo cardiomyopathy.

Case Report

A 60 year old female with a history of gastric bypass surgery and smoking presented to the hospital with nausea, vomiting and diarrhea. In the preceding days she had been taking large doses of ibuprofen and naproxen/esomeprazole for hip pain. In the Emergency Department, she developed a generalized tonic-clonic seizure requiring intubation for airway protection. CT/CTA imaging of the brain was normal. She was extubated six hours later when her level of consciousness improved. A Cardiology consultation was requested for anterior ST elevations with an elevated troponin level. Her level of consciousness was altered but she denied having any chest pain or dyspnea. Laboratory investigations revealed a low magnesium of 0.11 mmol/L. The initial CK was elevated at 556 U/L. The high sensitivity troponin I was initially 6163 ng/L, and peaked at 11843 ng/L over a period of 9 hours. Her ECG showed ST elevations initially in V3, then progressed to ST elevations in anteroseptal leads and subsequently to ST elevations in anteroseptal and inferior leads (Figure 1). She was accepted in transfer to the PCI hospital for consideration of an urgent coronary angiogram. The next morning, an urgent transthoracic echocardiogram (TTE) was obtained. This showed a dilated akinetic apex and moderate LV systolic dysfunction that was compatible with the appearance of takotsubo cardiomyopathy (Figure 2).

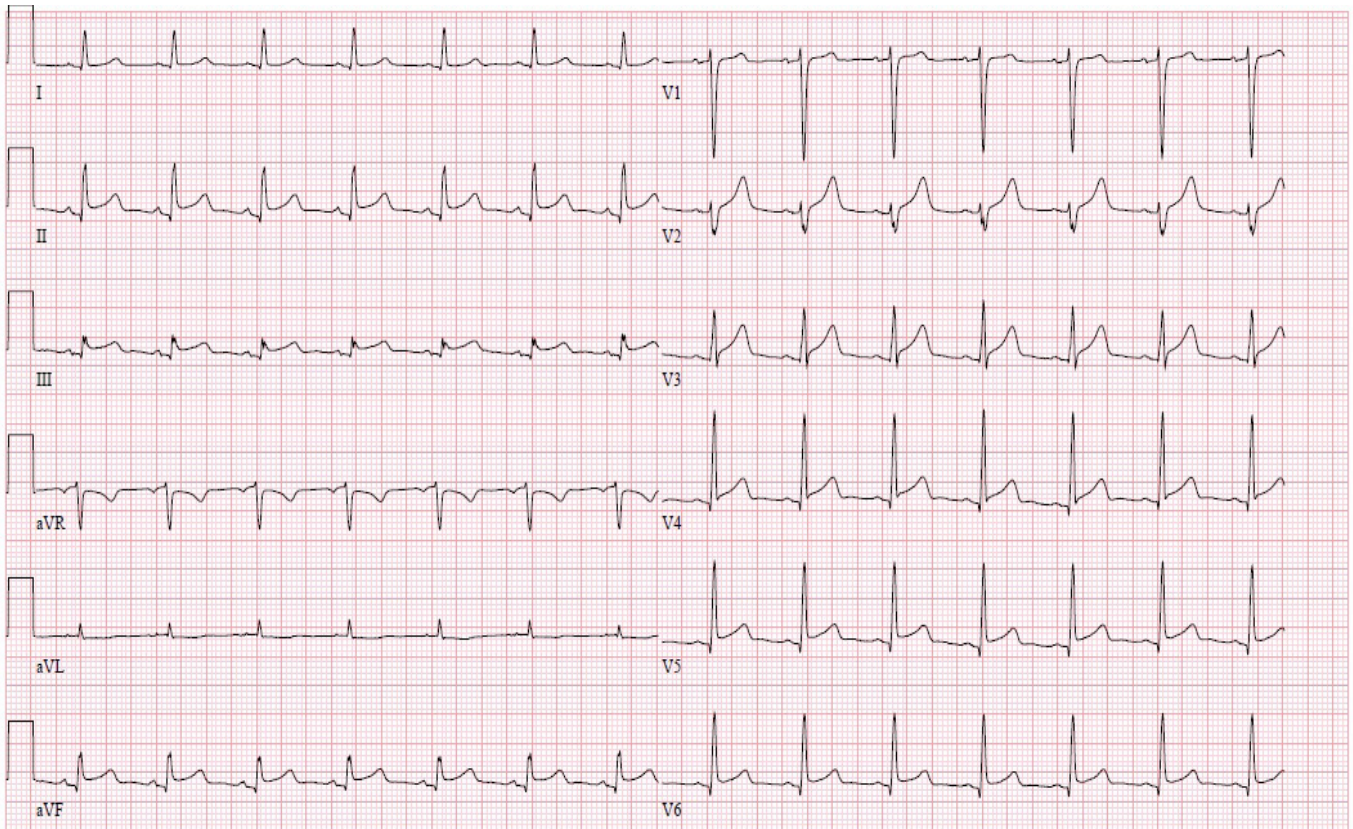


Figure 1. Electrocardiogram. Diffuse ST elevations seen in anteroseptal, lateral and inferior leads, and QTc prolongation.

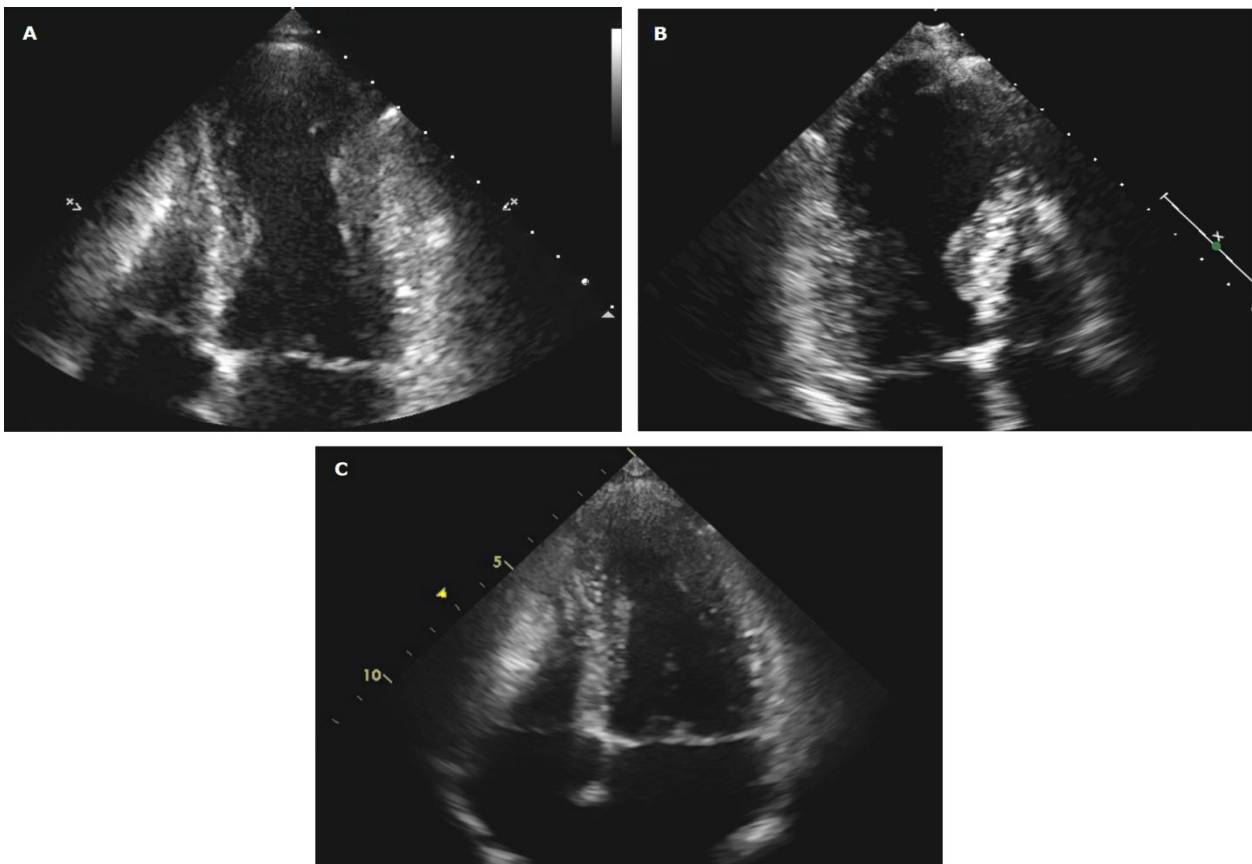


Figure 2. Transthoracic echocardiogram. A and B. Apical four chamber views demonstrating dilated akinetic apex and LV dysfunction typically seen in takotsubo cardiomyopathy. C. TTE repeated four months later with resolution of LV dysfunction.

Given the atypical presentation and the echocardiographic findings, coronary angiography was cancelled and the patient was managed conservatively for presumed TTC. A repeat TTE the next day showed slight improvement of LV function. Unfractionated heparin, aspirin and ticagrelor were all stopped after the presumed diagnosis of takotsubo cardiomyopathy was made on echocardiography. On the 4th day of admission, the patient developed retrosternal chest pain with numbness radiating down the left arm, which improved with nitroglycerin. This was associated with new ST elevations in the inferior leads. Given her ongoing pain, she was transferred to the PCI hospital and had an urgent coronary angiogram. This revealed a 70% stenosis of the proximal LAD, 50% stenosis of the mid LAD, and 100% stenosis of the right PDA. The lesion in the LAD appeared significant, but stable. The occlusion at the distal tip of the PDA may have been caused by thrombus or SCAD. Because the culprit lesion was unclear, PCI was not performed.

A cardiac MRI was performed and identified significant wall motion abnormalities in the apical segments and in the apex as well as significant late gadolinium enhancement involving approximately 75% of the myocardium in the apex. These findings were most in keeping with an ischemic LAD insult. There was also evidence of hibernating or stunning myocardium in the LAD territory.

The patient was scheduled for outpatient PCI, however this was delayed due to multiple complications including an upper GI bleed, drug-induced liver injury and recurrent hypomagnesemia. Four months later she received a stent and balloon angioplasty to the 70% mid-LAD lesion and balloon angioplasty to an 80% lateral 1st diagonal lesion. Repeat TTE prior to PCI showed mild apical hypokinesis and a normal LV ejection fraction (Figure 2).

Discussion

Takotsubo cardiomyopathy has been a diagnosis of exclusion including the absence of acute coronary obstruction or angiographic evidence of acute plaque rupture, although that does not preclude the presence of coronary artery disease per se [1,2]. Symptoms, ECG changes and the degree of rise in cardiac enzymes cannot reliably differentiate between acute coronary obstruction and TTC [2]. Cardiac MRI is increasingly recognized as an important imaging modality that can be used for the diagnosis and follow-up of TTC [3]. Cardiac MRI can accurately assess the myocardium and further characterize regional wall motion abnormalities, myocardial edema and late gadolinium enhancement (LGE) [3]. Absent or low intensity seen in LGE sequences is a unique finding that can differentiate TTC from acute MI or myocarditis [3].

In this case, there was diagnostic uncertainty regarding the presence of takotsubo cardiomyopathy. Evidence that supported the diagnosis of TTC included a history of physical stress, the characteristic finding of apical akinesis on echocardiography, and the near normalization of LV systolic function over time without angiographic interventions. However, the presence of obstructive disease in the LAD and RCA territories associated with corresponding ECG changes could account for the wall motion abnormalities seen.

The patient's re-presentation with chest pain and ECG changes after antiplatelet and antithrombotic medications were stopped is also more suggestive of obstructive CAD. The clinical presentation and results of routine investigations with ECG, echocardiogram and coronary angiography were ultimately unable to reliably differentiate between takotsubo cardiomyopathy and acute coronary syndrome. The diagnosis was made based on the findings on cardiac MRI that were consistent with LAD ischemia.

Previous case reports have described cases of TTC co-existing with ACS and ACS triggering TTC [4]. In these cases, it can be impossible to differentiate between the two diagnoses, as theoretically the wall motion abnormalities associated with ACS could also improve with time. Other possible triggers include spontaneous coronary artery dissection (SCAD) [5] and in this case epilepsy may have been considered a stress factor. There was no evidence of SCAD and although we cannot exclude the latter entirely, we were able to use cardiac MR as another imaging modality to further characterize the myocardium, which favoured acute coronary ischemia. This case contributes to the growing body of case reports that supports the use of cardiac MR to improve the diagnostic accuracy where there is diagnostic uncertainty [6].

Clarification of the early diagnostic criteria for takotsubo cardiomyopathy along with guidelines for treatment in the early acute phase are necessary to address cases where obstructive CAD coexists with or mimics TTC.

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