Apical Hypertrophic Cardiomyopathy Masked by Takotsubo Syndrome

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Abstract

We describe the case of a 66-year-old female presented to our emergency department (ER) with acute chest pain and diagnosed with Takotsubo syndrome that initially prevented from suspecting an apical hypertrophic cardiomyopathy at echocardiography.

Keywords: Apical hypertrophic cardiomyopathy, cardiac magnetic resonance imaging, echocardiography, Takotsubo cardiomyopathy

Introduction

Takotsubo syndrome (TTS) is characterized by transient left ventricular dysfunction and apical ballooning without obstructive coronary artery disease.[1] Hypertrophic cardiomyopathy (HCM) is a genetic disorder; apical HCM (ApHCM) is a variant characterized by hypertrophy of the ventricular apical myocardium.[2] We describe the case of a patient whose diagnostic suspicion of ApHCM was initially masked by her presentation with TTS.

Case Report

A 66-year-old female presented to our ER with acute chest pain associated with cold sweat and dizziness. Her medical history was significant only for a minor stroke related to patent foramen ovale, for which she was treated with aspirin 100 mg/day.

On admission, blood pressure was 113/78 mmHg; oxygen saturation was 97% on room air. A 12-lead electrocardiogram (ECG) documented sinus rhythm 109 bpm with ST elevation in the inferolateral leads, so the patient underwent emergency invasive coronary angiography with a presumptive diagnosis of an acute ST elevation myocardial infarction. No significant coronary artery stenosis were detected and left ventriculography showed mid-apical akinesia, a pattern consistent with TTS [Figure 1]. No obvious stressful trigger was identified. At laboratory studies, troponin T was 503 ng/dL and creatine kinase-MB 53 U/L.

A transthoracic echocardiogram [Figure 2] confirmed mid-apical akinesia with hypercontractility of the basal segments without obstruction of the left ventricular outflow tract and a moderate reduction of the left ventricular systolic function (left ventricular ejection fraction [LVEF] 35%).
days later, an echocardiogram [Video 1] documented apical akinesia with hypercontractility of the mid-basal segments, LVEF 50%, and increased wall thickness, which was attributed to edema (thickness of septum and posterior wall = 12 mm).

Her hospital course was uneventful and the patient was discharged on bisoprolol, ramipril, and torasemide. Upon discharge, a repeat ECG showed negative T waves in the precordial (V3–V6) and inferior leads [Figure 3].

An echocardiogram scheduled at 1 month after discharge [Figure 4 and Video 2] documented normal kinesis and LVEF 74%, compatible with recovery after TTS, but also thickened apical ventricular segments (thickness of lateral apex = 12 mm) and apical systolic cavity obliteration, confirmed with contrast echocardiography (Sonovue), suspected for ApHCM [Video 3].

Cardiac magnetic resonance (CMR) was therefore scheduled. 3T CMR (Magnetom Skyra, Siemens) [Figure 5] confirmed the echocardiogram findings and T2 mapping excluded the presence of edema in the thickened myocardial segments (values <45 ms). T1 mapping documented an increase of myocardial T1 values of mid-apical segments, with an increase of extracellular volume (ECV) values (36%, normal value <25%), but no late gadolinium enhancement was observed. These data were suggestive of a diagnosis of ApHCM.

A regular follow-up with echocardiography and 24-h ECG monitoring was, therefore, programmed.
**DISCUSSION**

Previous case reports in literature documented myocardial edema in the subacute phase of TTS,\(^3\) responsible for thickening of the involved segments and mimicking ApHCM.\(^4,5\)

In the above-described case, the increased thickness of the apical ventricular segments after the acute phase of TTS was not related to edema, suggesting that ApHCM was not evident until fully recovery of the apical ballooning, making it a challenging diagnosis.

To our knowledge, this is the first reported case in which ApHCM was masked at presentation by TTS with apical ballooning on echocardiography. CMR excluded the presence of edema as the cause of apical ventricular thickening but documented an increase in ECV, compatible with ApHCM.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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