Myocardial perfusion scintigraphy during chest pain: An atypical presentation of takotsubo cardiomyopathy?

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Although Takotsubo cardiomyopathy (TCM) knowledge is increasing, the exact pathophysiology remains unclear. TCM represents 1%–2% of all troponin positive acute coronary syndromes, affects predominantly postmenopausal women, and is commonly preceded by exposure to severe physical or emotional stress. Transient wall motion abnormalities mimicking ST-elevation myocardial infarction is expected as well as increase of troponin levels and echocardiography alterations. This case report is about a patient that as far as we know is the first case that shows the use of myocardial perfusion imaging in the acute phase of TCM. In general, the TCM Mayo Clinic diagnostic criteria have been very helpful in the clinical setting. In this specific case, however, the presence of reduced myocardial perfusion in the acute phase combined with increased troponin levels seemed to be in contradiction with the exclusion of obstructive coronary artery disease.

Key Words: Takotsubo • cardiomyopathy • myocardial perfusion

BACKGROUND

Although Takotsubo cardiomyopathy (TCM) knowledge is increasing, the exact pathophysiology remains unclear. TCM represents 1%–2% of all troponin positive acute coronary syndromes, affects predominantly postmenopausal women and is commonly preceded by exposure to severe physical or emotional stress. Transient wall motion abnormalities mimicking ST-elevation myocardial infarction (STEMI) can be expected as well as increase of troponin levels and (echocardiographic) wall motion abnormalities. Although the TCM Mayo Clinic diagnostic criteria have helped in establishing diagnosis (i.e., including among others exclusion of coronary artery disease or acute plaque rupture), TCM remains a challenge to diagnose.[1]

CASE PRESENTATION

A 48-year-old female patient presented at the emergency department of a tertiary hospital with typical angina after severe emotional stress (i.e., the death of a relative very important to her). Prior to this event, she had never experienced any complaints of thoracic discomfort. Pain persisted for approximately 60 minutes. She had a positive family history for coronary artery disease and used fluoxetine for a minor depression. Physical exam was normal. The electrocardiogram showed no changes and the echocardiogram showed akinesia of the mid-ventricular segments.

To exclude myocardial ischemia, myocardial perfusion scintigraphy was requested according to a chest

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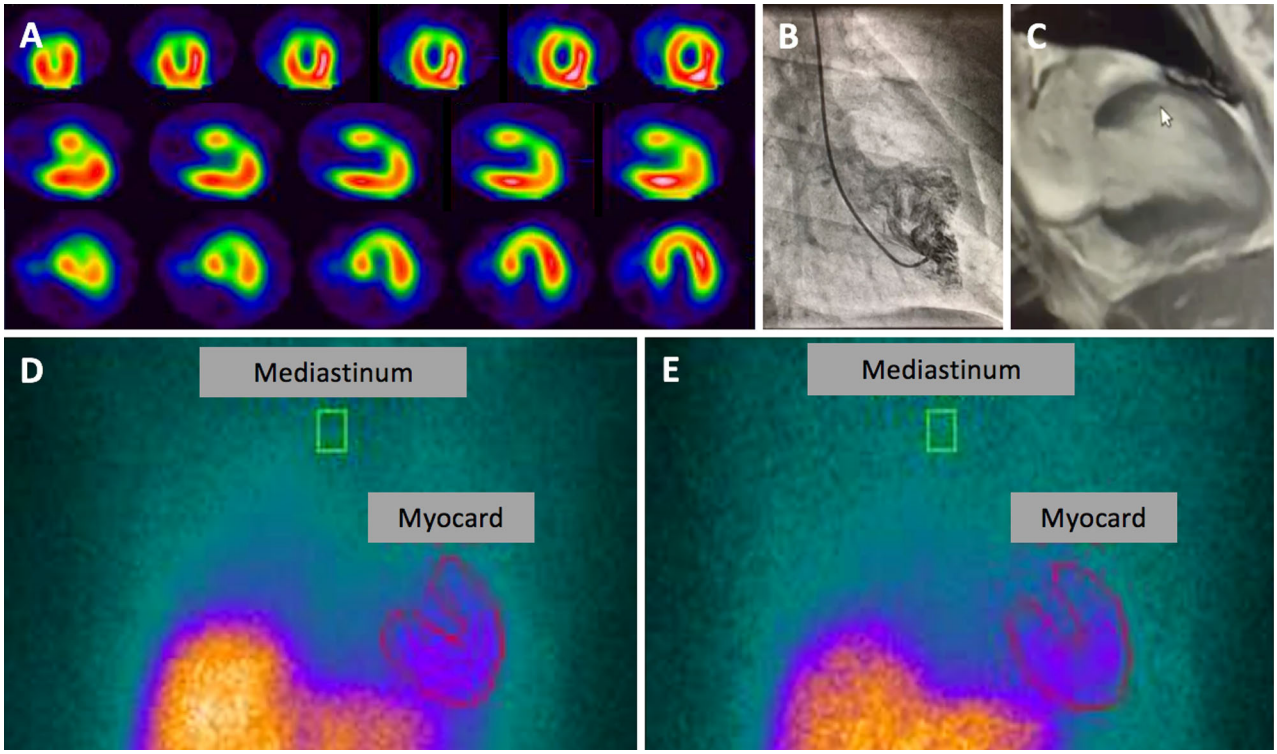

Figure 1. (**A**) SPECT perfusion images obtained when the tracer was injected during pain. There are anterior and septal perfusion defects. (**B**) Left ventriculography in the right anterior-oblique projection showing anterior wall motion abnormality, (**C**) Cardiac MRI shows no delayed gadolinium enhancement, (**D**) early planar I-123 MIBG images in the anterior projection (the mediastinum and myocardium regions are outlined), (**E**) late I-123 images.

pain protocol. Images obtained when the tracer was injected during the pain, showed reduced to absent perfusion in the anterior and antero-septal segments (Figure 1A). The gated images showed reduced anterior wall thickening. The initial troponin level was negative.

Coronary angiography was performed and revealed 50% stenosis of the proximal left anterior descending artery (LAD) and the first diagonal branch. Fractional flow reserve (FFR) was 0.84 in both the LAD and the diagonal branch. Left ventriculography showed a moderate increase of end-systolic volume and antero-lateral akinesia (Figure 1B).

The second troponin, after 6 hours from the first and 30 minutes after coronary angiography, was positive (0.6 ng·mL^{-1}, normal range < 0.16 ng·mL^{-1}). A repeat electrocardiogram showed T-wave inversion.

Based on these findings, the diagnosis of atypical TCM was most likely. 48 hours after presentation echocardiography showed improved contraction pattern with minor hypokinesis of the antero-septal wall only. Cardiac magnetic resonance imaging (MRI) showed a small area of edema in the anterior wall without signs of late gadolinium enhancement (Figure 1C).

Two months after this episode, the patient was stable. ^{123}I-*m*IBG imaging was performed [early (Figure 1D) and late (Figure 1E)]. The early Heart/Mediastinum (H/M) ratio was 2.07 and the late H/M ratio was 1.74 and myocardial ^{123}I-*m*IBG washout was increased (47%). Since this episode, the patient has done well on medical therapy.

DISCUSSION

As far as we know this case is the second that described the use of myocardial perfusion imaging (MPI) in the acute phase of TCM. An earlier report showed a normal perfusion during the acute phase.[2] In contrast we found rather severe perfusion abnormalities. In the earlier report the TCM was precipitated by physical stress, while our case was precipitated by emotional stress.

In this specific case, the troponin elevation was minimal compared to the extent of the perfusion abnormality and the CAD was not hemodynamically significant based on physiological assessment using FFR. But, it is increasingly recognized that 5-10% of patients with myocardial infarction (MI) could have no

severe CAD[3,4] These patients are generally younger and more often women in comparison with patients with MI and severe obstructive CAD.[5,6]

The underlying pathophysiological mechanisms of MI in the absence of severe CAD are poorly understood, although several different mechanisms have been proposed, including plaque disruption, spasm, thromboembolism, dissection, microvascular dysfunction, ischemic myocardial injury attributable to supply/demand mismatch, and clinically non-detected myocarditis or TCM.[3,7]

The difference between the report by Dorfman *et al.* (2) and the case presented here (i.e., normal vs. abnormal perfusion in the acute phase) implies the wide spectrum of clinical presentation of TCM. Our finding of prolonged or persisting sympathetic dysfunction has been described previously and is most likely not associated with increased risk of future events.[8]

CONCLUSION

The perfusion abnormalities and denervation provide an interesting association, but the cause and effect relationships remain elusive.

Disclosures

Maria Marta Maggiotto Sabra, Fernanda Salomão Costa, Jader Cunha de Azevedo, Claudio Tinoco Mesquita, and Hein J. Verberne declare that they have no conflicts of interest to disclose.

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