Female, 40-year-old, 1.67 m tall patient, weighing 71 kg, with cardiac frequency of 85 bpm, blood pressure $110 \times 70$ mmHg, reporting chest pain and dyspnea associated with an apparent ST segment elevation at anterior wall ECG. Enzymatic curve elevation was not observed. The patient’s social history revealed a serious family argument in the afternoon with the onset of pain early in the evening.

Echocardiogram demonstrated a left ventricular apical aneurysm, and coronary computed tomography angiogram was normal. The patient was referred to the Unit of Radiology and Diagnostic Imaging of Hospital Pró-Cardíaco for evaluation using cardiac magnetic resonance imaging.
because of (transient) left ventricular dys-
similar to that of acute myocardial infarct (3–5).
mental functional changes
different clinical presentations and seg-
Anatomy and enervation may explain the
cardiac vessels were normal (1,2).
Cardiac enzymes, segmental involvement
rupture syndrome, catheterization is required but is
drome is presented followed myocardial infarction.

**Diagnosis:** Stress-induced cardiomyopathy (transient left ventricular apical ballooning, broken-heart syndrome or Takotsubo syndrome).

**COMMENTS**

Firstly described by a Japanese group – Satoh et al. (3,4) early in the 1990’s decade – as a condition affecting preferentially middle-aged women who, after going through high-degree stress, presented clinical findings similar to those of acute myocardial infarct (precardial pain, changes in cardiac enzymes, segmental involvement of the left ventricle), while the coronary vessels were normal (1,2).

In stress-induced cardiomyopathy (Takotsubo syndrome), either a single or both ventricles may be compromised. Additionally, the condition may present as a reverse syndrome with mid-ventricular akinesia, and hypercontractility of the basal and apical segments (1–3).

The precise physiopathology of this syndrome remains unknown; however the condition is associated with exaggerated sympathetic stimulation suggesting catecholamine release as a common pathway for this condition (3,4).

Regional differences in the adrenergic anatomy and enervation may explain the different clinical presentations and segmental functional changes (3–5).

The clinical presentation may be very similar to that of acute myocardial infarct because of (transient) left ventricular dys-function, associated chest pain, electrocardiographic changes and mild enzyme elevation, mimicking an infarction (4,5).

In cases mimicking acute coronary syndrome, catheterization is required but is negative for coronary occlusion. Cardiac ventriculography can identify left ventricular ballooning. In clinical cases where the likelihood of acute coronary syndrome is low, coronary CT angiography may be performed along with cardiac enzyme evaluation, as in the present case (1,3,4).

Echocardiography, left ventriculography, cardiac CT angiography and magnetic resonance imaging demonstrate apical ballooning with hyperkinesia of the ventricular basal segment, resembling a fishing pot for trapping octopus utilized in Japan (Takotsubo) (Figure 4) (5).

Takotsubo syndrome may be triggered by quite variable factors, including stress. Multiple factors have already been reported as triggering factors: psychological conditions, exacerbation of systemic, pulmonary, neurogenic, gastrointestinal and renal diseases, besides other non-specific factors. Patients with pheochromocytoma or those submitted to echocardiogram with dobutamine may also develop this cardiomyopathy (6–8).

Stress related syndrome is most frequent in women aged between 60 and 75 years of age. The most common symptom is retrosternal pain; however some clinical presentations may mimic an acute coronary syndrome with dyspnea, increased cardiac enzymes (troponin) and electrocardiographic abnormalities. More severe cases may progress with cardiogenic shock and respiratory failure as a result of acute pulmonary edema (2,3,6).

Absence of significant coronary obstruction and reversibility of left ventricular dysfunction constitute relevant concepts to be taken into consideration for the diagnosis, with recovery of the ventricular function on average in up to 18 days, although it may take up to three months from the symptoms onset (1,3,4,7).

Although the actual prevalence of this condition is still to be defined, retrospective surveys suggest that 2% of cases assisted as acute coronary syndrome are diagnosed as Takotsubo syndrome (1–3).

**Cardiac magnetic resonance imaging**

Cardiac magnetic resonance imaging with the delayed enhancement technique can characterize the absence of a significant myocardial lesion, allowing the detection of a possible ischemic lesion/infarct and the exclusion of myocarditis as a differential diagnosis (9,10,11).

Magnetic resonance imaging can demonstrate changes in the ventricular contractility and is useful as follow-up study because of the absence of ionizing radiation or nephrotoxic contrast agent.
Final considerations

Cardiac magnetic resonance imaging should be performed in all patients with diagnostic suspicion for a correct characterization of the absence of myocyte lesion and follow-up of the disease.

In the present case, the clinical and imaging follow-up with resolution of the change in the segmental contraction confirms the diagnosis. The condition management is based on hemodynamic support measures.

REFERENCES