MR imaging of Tako-Tsubo syndrome.

Poster No.: C-1837
Congress: ECR 2014
Type: Educational Exhibit
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Keywords: Hemodynamics / Flow dynamics, Edema, Acute, Imaging sequences, MR, Cardiovascular system, Cardiac, Anatomy
DOI: 10.1594/ecr2014/C-1837

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Learning objectives

The aim of this review is to describe the notion, characteristics, and findings in MRI of Takotsubo syndrome.

To illustrate the radiological key points that may help in distinguishing Takotsubo syndrome from other cardiac disorders, with emphasis on acute myocardial infarction and myocarditis.

Background

Patients with chest pain, ECG changes, and elevated myocardial enzymes are initially admitted in the emergency department with a diagnosis of acute coronary syndrome. Approximately 7-10% of them have an angiographic study showing no significant alterations in the coronary arteries. Takotsubo syndrome is characterized by angina-like chest pain, ECG changes, moderate elevation of enzymes, left ventricular (LV) systolic dysfunction with impaired contractility, typically affecting the apical and mid segments of the LV myocardium, and absence of myocardial necrosis in the delayed enhancement sequence of magnetic resonance imaging (MRI).

This disorder has been reported more frequently in women in their seventh to eighth decades of life, and is typically associated with a physical or emotional stressful precipitant. However, Takotsubo syndrome presents a favorable prognosis, with disappearance of all these changes and complete recovery of LV systolic function within a few weeks or months. The correct differential diagnosis is essential due to prognostic and therapeutic implications, and should be performed with other causes of acute coronary syndrome with normal coronary angiography, emphasizing acute myocardial infarction (AMI) with normal coronary arteries and myocarditis.

Epidemiology

Takotsubo syndrome was first described in Japan by Sato et al. in the early 90s and was considered an independent entity in 2001. This disorder has received other nomenclatures: broken heart syndrome, transient apical dyskinesia, "apical ballooning

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stress cardiomyopathy, etc. Takotsubo refers to typical morphology acquired in ventriculography of the LV when apical akinesia and hypercontractility of the basal segments are manifested. The resulting image resembles Takotsubo, a Japanese vase used to trap octopus. Fig. 1 on page 6

The Takotsubo syndrome is thought to be responsible for 1% to 2% of all admissions for acute coronary syndrome, although the lack of consensus on diagnostic criteria makes it probably a misdiagnosed disease, so the real incidence is currently unknown. In our institution 15 cases of stress cardiomyopathy were identified since 2011.

High incidence in postmenopausal women 60-75 years old has been reported in literature of this subject even though the age range can be variable. Not many cases have been described in men or young people. Nevertheless there is no large database to suggest its prevalence in the general population. In our institution, most patients were female aged between 55 and 85 except one case of a 70-years-old male.

The Takotsubo syndrome is usually precipitated by emotional stress. The most common precipitant is death of a close family member but other reported precipitants include court appearance, fear of a procedure, arguments and surprise parties/reunions, etc. Some of these factors are included in the table of Fig. 2 on page 6

Pathogenesis

The pathophysiology of Takotsubo syndrome is unknown but is likely multifactorial, involving vascular, endocrine, and central nervous systems.

There is not at present an established etiopathogenic mechanism to justify this entity. The most accepted theory in the published series is the toxicity of catecholamines and neuropeptides stress. The development factor in these patients is a physical or emotional stressful situation, which contributes to stimulation of the sympathetic system with subsequent release of catecholamines, encouraging the epicardial coronary spasm, microvascular spasm with decreased coronary flow reserve and dysfunction endothelial. Direct damage to myocardial tissue due to catecholamine toxicity has also been proposed.
The high prevalence of this syndrome in elderly women and climatery raises the possibility of hormonal influence. Recent studies concerning estrogens as protector role have been documented.

Nonetheless, there are few scientific studies published to support these hypotheses and more evidence in literature is required.

In our institution 4 patients who had a previous episode of stressful emotional situation were reported (recent death of a parent, date coincides with the anniversary of the death of a spouse, personal discussion and work stress), as well as prior cocaine consumption in another patient and presence of bilateral pheochromocytoma discovered as incidental finding in other case.

Clinical findings

Clinically, Takotsubo syndrome is similar to an acute coronary syndrome. These patients are seen in emergency department for chest pain in approximately 50-60% of cases, and dysnea may be associated. Cardiac arrest as an initial symptom of this syndrome is exceptional.

Electrocardiogram (ECG) changes on presentation are variable (Fig. 3 on page 7), and can mimic acute myocardial infarction with ST elevation involving most commonly anterior segments and typical deep T-wave inversion. Prolongation of the QT interval and transient anteroseptal Q waves can be also visualized.

Generally cardiac enzymes are elevated at presentation, but the levels are markedly lower than would be expected according to the extent of wall motion abnormalities of LV and ECG findings.

Echocardiogram reveals the initial and reversible abnormalities in myocardial contractility of the LV, visualizing hypokinesia, dyskinesia or akinesia of the apical segments as more frequent findings. Classically, the apical or mid-ventricular segments (or both) of the LV are hipokinetic or akinetic, and the basal segments often exhibit increased contractility. Fig. 4 on page 8
Moreover, this test also allows diagnose uncommon mechanical complications described in Takotsubo syndrome like thrombus formation favored by apical akinesis. Subsequently, angiography performed in these patients shows typically the absence of significant lesions in the coronary arteries.

Several diagnostic classifications have been suggested to Takotsubo syndrome but consensus in the literature to establish them has not been found. Generally, the most accepted are the diagnostic criterias proposed by the Mayo Clinic (Fig. 5 on page 8), which are summarized in:

- Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments, with or without apical involvement. Regional wall motion abnormalities extend beyond a single coronary vascular bed. A preceding physical or emotional stressor is often present, but not necessarily.

- No obstructive coronary disease or acute plaque rupture (determined angiographically)

- New electrocardiographic abnormalities (ST-segment elevation, T-wave inversion, or both) or modest elevation in cardiac troponin level.

- Absence of pheochromocytoma or myocarditis.

**Differential diagnosis:**

The differential diagnosis of Takotsubo syndrome should be performed with other causes of acute coronary syndrome with absence of coronary artery obstruction demonstrated angiographically, focussing in AMI with coronary artery recanalization and myocarditis. Diagnosis of Takotsubo syndrome requires to rule out other causes of reversibleLV contractile dysfunction. The value of the MRI is based on the ability to detect the presence or absence of myocardial necrosis through delayed enhancement sequence. Takotsubo syndrome shows no data of myocardial necrosis unlike AMI and myocarditis. The importance of accurately determining the final diagnosis in these cases is based on the therapeutic and prognostic implications of each entity.

**Prognosis and therapeutic approach**
Although this cardiomyopathy has a benign clinical course in the mid-long term, with complete recovery of ventricular function and complete resolution of other alterations in a few weeks, a small percentage of cases with several complications have been reported, most of them arising from the ventricular dysfunction and heart failure. In our institution two cases of cardiogenic shock were documented, although the subsequent clinical course was favorable. No patient death were reported.

Therapeutic approach of Takotsubo syndrome has not been approved. Nonetheless, the usual treatment of acute coronary syndromes and complications is recommended. The use of beta-blocking drugs are also advised to counteract the deleterious effects of catecholamines and improve contractility.

**Images for this section:**

Fig. 1: Ventriculography in diastole and systole. Systole image shows akinesia of mid and apical segments with hypercontractility of basal segments, forming the typical image of Takotsubo, a Japanese vase used to trap octopus (red line).
**Fig. 2:** Precipitating factors for Takotsubo syndrome.

**Fig. 3:** ECG in suspected Takotsubo syndrome. INITIAL ECG: Sinus rhythm. Left anterior hemiblock. ST segment elevation of 1 mm in V4 and V5 (superior image red arrow). ECG 48 HOURS LATER: Typical electrocardiographic evolution of Takotsubo syndrome: giant negative T waves of 5mm in precordial leads (inferior image red arrow). Generalized disturbance of repolarization. Normal QT.
Fig. 4: Echocardiogram of a patient with Takotsubo syndrome in diastole and systole, showing contractile dysfunction of the LV mid and apical segments during systole with hyperkinesis of the basal segments (red arrows), forming the typical image of Takotsubo.

**Proposed Mayo Clinic Criteria for Diagnosis of Takotsubo Cardiomyopathy**

1. Transient hypokinesia, akinesia, or dyskinesia of the left ventricular mid segments, with or without apical involvement. Regional wall motion abnormalities extend beyond a single coronary vascular territory. A preceding physical or emotional stressor is often present.

2. Absence of obstructive coronary disease or acute plaque rupture (determined angiographically).

3. Electrocardiographic abnormalities (ST-segment elevation, T-wave inversion, both) or modest elevation in cardiac troponin level.

4. No pheochromocytoma or myocarditis.
**Fig. 5:** Proposed Mayo Clinic Criteria for Diagnosis of Takotsubo syndrome.
Findings and procedure details

1. Cardiac-MRI

In our institution, we use a specific MRI protocol for suspected acute coronary syndrome, including Takotsubo syndrome, based on CINE-MRI SSFP sequence, T2 IR FSE sequence and Delayed enhancement sequence. We proceed to summarize some important aspects and contributions:

- **CINE-MRI SSFP (steady state free precession) or white blood sequence:** Gradient echo sequences allows functional studies cardiac due to its high temporal resolution, making it possible to analyze images in CINE-RM mode. Images are obtained in the horizontal and vertical long axis (two and four cameras) and the short axis. The main feature of this sequence is that the blood signal is hyperintense in contrast to hyperintense myocardium signal, which provides a good definition of the ventricular margins. It allows assessment of the segmentary and global contractility, ventricular volumes and ejection fraction of LV. Fig. 6 on page 18

- **2IR FSE T2 or black blood sequence:** Fast spin echo sequence with double inversion recovery T2-weighted with fat suppression. Images are acquired in the short axis and in long axis. In this sequence the existence of intramyocardial edema is valued. It is important to identify whether edema has a vascular location. Fig. 7 on page 18

- **Delayed enhancement sequence after injection of gadolinium:** It is used to assess the presence of myocardial necrosis. Images are acquired in long axis and the short axis. Generally Gadolinium is a contrast with extracellular space distribution, from the intravascular space to the interstitial space. When myocardial damage exists, the contrast crosses the cell membrane and gets into cardiomyocytes, which determines a slowing of the washing phase of the gadolinium. Enhancement patterns can be subendocardial infarction, transmural or subepicardial, depending on the affected myocardial region.

2. MRI findings
CINE-MRI SSFP sequence:

It reveals abnormalities in the LV wall motion affecting the mid and apical segments, which may be akinetic or dyskinetic. In addition to this, an excessive contractility of basal segments has been described in this disorder. All these findings lead to the typical image that gives name to this entity; the morphology acquired by the LV is very similar to the Japanese vase used in hunting octopuses called TakoTsubo.

**Fig. 8 on page 19**

Alterations in myocardial contractility cause LV systolic dysfunction that can reduce ventricular ejection fraction, causing derived symptoms from heart failure. Occasionally, cardiac output may be severely compromised even to cause cardiogenic shock, but this occurs in a reduced number of patients. Variables that could contribute to serious complications resulting from heart failure in Takotsubo syndrome have been studied, such as age, presence of diabetes or patients with ventricular arrhythmias.

CINE-MRI SSFP sequence also allows to evaluate some complications described in Takotsubo syndrome like the dynamic obstruction in the LV outflow even with associated systolic anterior motion of the anterior mitral leaflet, similar to findings in hypertrophic cardiomyopathy. Detect this complication has great importance because treatment with some drugs may raise end-systolic volume at the expense of decreased contractility.

Another uncommon but serious complication is thrombus intracavitary formation in the LV. Although ventriculography or echocardiography are useful for identifying thrombus, small clots may be underdiagnosed with these techniques. MRI can contribute in these cases identifying the thrombus by means of CINE-MRI and Delayed enhancement sequences, which has great impact on the patient because the introduction of anticoagulant therapy reduces mortality.

**Fig. 9 on page 20**

- 2 IR FSE T2 or black blood sequence:

Edema is a typical finding in Takotsubo shown as areas of increased signal with a diffuse or transmural distribution in both the apical and mid planes of the LV, which are not related to vascular territories, unlike AMI. Most often these areas affected correspond to dysfunction in the LV contraction displayed in CINE-MRI sequences. **Fig. 10 on page 20**
- Delayed enhancement sequence after injection of gadolinium:

Absence of delayed enhancement is the most characteristic finding in Takotsubo syndrome, reflecting the lack of myocardial necrosis. In fact, this sequence represents the major contribution of MRI in the differential diagnosis of AMI and myocarditis. Fig. 11 on page 21

Nevertheless, although the absence of delayed enhancement is a finding indicative of Takotsubo syndrome, it is not considered a diagnostic criteria. Several reports have been documented the presence of a tenuous delayed enhancement which certain authors attribute to areas of inflammation or edema.

In our institution delayed contrast sequences showed no enhancement in most Takotsubo syndromes with two exceptions; a faint and focal enhancement was detected in two patients which was interpreted as areas of secondary inflammation. Fig. 12 on page 21

Examples of MRI findings in Takotsubo syndrome. Fig. 13 on page 22 Fig. 14 on page 23 Fig. 15 on page 24
**Fig. 13:** Cardiac-MRI sequences in 68-years-old female with recent episode of occupational stress presented chest pain and slight increase of ST segment in admission ECG, akinesia in all faces of LV mid segments and apex with basal hypercontractility segments demonstrated in ecocardiography without alterations in coronary arteries angiographically. A y B. Diastolic and systolic images in CINE sequence shows hipokinesia of LV apical segments. C. T2 sequence manifest hyperintense areas in all faces of the LV mid and apical segments suggesting intramyocardial edema. D. Delayed enhancement sequence shows absence of hyperintense areas demonstrating absence of intramyocardial necrosis. Complete recovery of LV wall motion within days. Transient apical diskinesia (Takotsubo syndrome) was diagnosed.

**References:** Hospital universitario La princesa - Madrid/ES

**Fig. 14:** Cardiac-MRI sequences in 77-years-old female suspected of Takotsubo syndrome. A y B. Diastolic and systolic images in CINE sequence show severe hipokinesia of LV apical and mid segments (blue arrows) with hyperkinesis of basal segments (yellow arrow). LVEF:36%. C. T2 sequence demonstrates edema in mid and apical segments of LV apex, anterior and inferior faces(red arrow) D. Delayed enhancement sequence shows absence of intramyocardial necrosis.

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Fig. 15: 27-years-old female with Takotsubo syndrome and pheochromocytoma as incidental finding (red arrows). Several international authors have proposed diagnostic criteria for Takotsubo syndrome in the literature, but they are not clearly established. It is common to all of them that the presence of a pheochromocytoma is exclusionary for the diagnosis of Takotsubo cardiomyopathy. However, the possibility of supraphysiologic elevations of catecholamines as etiopathogenic factor of this syndrome arises, so it seems logical that they could share the same pathophysiological mechanism with cardiomyopathy caused by pheochromocytoma. Thus, in this case, we would point out that it could be considered as a transient stress cardiomyopathy secondary to pheochromocytoma.

References: Hospital universitario La princesa - Madrid/ES

3. Atypical forms

Clasically, in Takotsubo syndrome, the apical or mid-ventricular segments (or both) of the left ventricle have wall motion abnormalities. Nevertheless, another patterns of the LV systolic dysfunction have been reported. Takotsubo syndrome with mid-ventricular aquinesis with LV apical segments non affected has been described, representing a relatively frequent finding. Aquinesis of basal segments with no changes in contractility of LV mid and apical segments has as well been suggested. We have notified in our institution a Takotsubo syndrome with only wall motion abnormalities reflected in LV mid segments. Fig. 16 on page 25
4. Evolution

The most specific finding of Takotsubo syndrome is the complete reversal in short-term of all changes, and involves the mandatory condition that provides the diagnosis of this entity.

ECG: Electrocardiographic changes described in Takotsubo syndrome are highly variable. Approximately one third of these patients have ST segment elevation involving mainly the anterior LV. Other nonspecific findings as deep T-wave inversion and nonspecific ST-T wave changes have been observed. There are some electrocardiographic alterations that are common in these patients, as the presence of symmetric T-wave inversion and QT prolongation, findings that disappear in weeks or months. Fig. 17 on page 25

Echocardiogram: Wall motion abnormalities of the LV in Takotsubo syndrome disappear completely in a few days or weeks. It is supposed to be the test indicated in the monitoring of these patients, since it allows to assess the global and segmental contractility of the LV wall and evaluate recovery of systolic function.

Cardiac MRI: Typically no monitoring of these patients with this imaging test is performed, although expect to find in a few days or weeks a complete recovery of contractility in the affected segments and systolic function in CINE-MRI. A vanishment of the edema in the affected segments in 2IR FSE T2 sequence is also seen, although the disappearance of these changes evolves more slowly.

Complete reversal of all abnormalities in Takotsubo syndrome occurs in a variable period of time, which in the literature describe from the earliest days until first quarter after acute episode. In our institution variability in the time to resolution of symptoms is evident too, identifying patients with Takotsubo syndrome that improve completely the LV wall motion within two weeks even days and others in which the recovery is documented in 2 months, although the disappearance of the signal changes caused by intramyocardial edema is slower compared to the recovery of LV contractility. Fig. 18 on page 26

Little data about the incidence of recurrences of Takotsubo syndrome have been reported, especially over the long-term follow-up, but is infrequently. Determine actual data and risk factors of recurrences is complicated because not all patients are followed,
and those who have received follow-up have been followed only during the first years. We have not notified any recurrence in patients with syndrome Takotsubo in our institution. In conclusion, the rate of recurrence of Takotsubo syndrome could be low, although there are few published studies regarding this matter.

5. Differential diagnosis

The main differential diagnoses in Takotsubo syndrome are other causes of acute coronary syndrome with normal coronary arteries on angiography, focussing in AMI and myocarditis. We will summarize below some of its distinctive features in MRI:

- **Acute myocardial infarction (AMI):** Subendocardial region is more sensitive to myocardial ischemia. Ischemic necrosis progresses from the subendocardium to the epicardium, showing subendocardial or transmural pattern in delayed enhancement sequence. These areas correspond to a coronary vascular territory. Furthermore, they have hypocontractility and edema in CINE-MRI and 2IR FSE T2 sequences respectively. Myocardial locations with edema and no hyperenhancement on delayed enhancement sequence are considered areas of stunned myocardium.

- **Myocarditis:** Its main features are the presence of MRI changes in contractility in CINE-MRI sequence and edema on 2IR FSE T2 sequence that does not follow a coronary vascular distribution. In late enhancement sequences patchy or nodular enhancements with a predilection for the lateral face and a subepicardial and mesocardial pattern are observed. If myocarditis is also accompanied by pericarditis (Myopericarditis) hyperenhancement of the pericardium in the delayed enhancement can be detected.

Fig. 19 on page 27
Fig. 19: Cardiac-MRI sequences performed in two different patients with AMI and myocarditis. AMI with normal coronariography and myocarditis: Systolic CINE 2 chambers image shows akinesia of apex and anteroapical segment of LV (yellow arrow). Preserved systolic function. In T2 short axis image, hyperintense areas are evident in the apex, corresponding to edema. Areas of transmural enhancement in apex and apical segments of septum in delayed enhancement sequence of 4 chambers (red arrow) , corresponding with myocardial necrosis, without evidence of viability. Myocarditis: Images of CINE 2 chambers sequence did not show abnormalities in LV wall motion. Edema in anterior, septal and antero-lateral face of mid segments were marked( green arrows). In 4 chambers of Delayed enhancement image, nodular foci of intramyocardial enhancement ( pink arrow) and linear enhancement in the pericardium at the apex ( orange arrow) were observed, findings suggestive of Myopericarditis .

References: Hospital universitario La princesa - Madrid/ES

In conclusion, the contribution of MRI in the differential diagnosis of Takotsubo syndrome with myocardial infarction and myocarditis is based on (Table 1 on page 28):

In Takotsubo syndrome absence of late enhancement (taking into account remarks referred above), hypocontractility and edema without vascular distribution are characteristic findings, compared to subendocardial or transmural pattern of hyperenhancement , alterations in contractility and edema in areas that are well matched with coronary vascular territories seen in AMI. Edema and changes in contractility not related to a coronary vascular location are also seen in myocarditis, but unlike
Takotsubo syndrome, they show hyperenhancement on delayed enhancement sequence with subepicardial and mesocardial pattern.

**Images for this section:**

![Fig. 6: Left ventricular segmentation.](image-url)

**Fig. 6:** Left ventricular segmentation.
**Fig. 7:** Coronary anatomy and myocardial perfusion territories.

**Fig. 8:** 87-years-old female presented chest pain, changes in ECG in anterior LV segments, moderate elevation of myocardial enzymes and normal angiographic study. CINE-MRI sequence performed in long axis showed mild dilatation of LV and akinesia.
of mid and apical segments of LV with basal segments hypercontractility (blue arrows). Global systolic function was depressed with left ventricular ejection fraction (LVEF): 46%, compensated through basal segments hypercontractility. Bilateral pleural and pericardial effusion is also noted.

Fig. 9: Delayed enhancement sequence with long axis of 4 chambers shows a small apical intracavitary thrombus (red arrow) in a patient with suspect of Takotsubo syndrome.
**Fig. 10:** 2IR FSE T2 sequence, short axis, shows signal hyperintensity in septal and anterior faces of LV myocardium compatible with edema.

**Fig. 11:** Delayed enhancement sequence shows absence of myocardial enhancement related with no necrosis in long axis of 2 chambers (A) and 4 chambers (B) in patient presented in figure 8.
**Fig. 12:** Cardiac- MRI sequences of Takotsubo syndrome. A y B. Diastolic and systolic images in long axis of 2 chambers in CINE sequence. Hypokinesia in mid segment of LV anterior face (blue arrow). Note asymmetric basal septal hypertrophy. C. Long axis of 2 chambers and D. short axis in T2 sequence show edema in mid segment of LV anterior face and septum (green arrows). E. Long axis of 2 chambers and F. short axis of Delayed enhancement sequence show a slight enhancement in mid segment of LV anterior face (yellow arrows). This enhancement is much lower than expected in infarct or myocarditis, suggested by several authors edema/ inflammatory changes. Patient recovered later global myocardial contractility.
Fig. 13: Cardiac-MRI sequences in 68-years-old female with recent episode of occupational stress presented chest pain and slight increase of ST segment in admission ECG, akinesia in all faces of LV mid segments and apex with basal hypercontractility segments demonstrated in ecocardiography without alterations in coronary arteries angiographically. A y B. Diastolic and systolic images in CINE sequence shows hipokinesia of LV apical segments. C. T2 sequence manifest hyperintense areas in all faces of the LV mid and apical segments suggesting intramyocardial edema. D. Delayed enhancement sequence shows absence of hyperintense areas demonstrating absence of intramyocardial necrosis. Complete recovery of LV wall motion within days. Transient apical diskinesia (Takotsubo syndrome) was diagnosed.
Fig. 14: Cardiac-MRI sequences in 77-years-old female suspected of Takotsubo syndrome. A y B. Diastolic and systolic images in CINE sequence show severe hipokinesia of LV apical and mid segments (blue arrows) with hyperkinesis of basal segments (yellow arrow). LVEF:36%. C. T2 sequence demonstrates edema in mid and apical segments of LV apex, anterior and inferior faces (red arrow) D. Delayed enhancement sequence shows absence of intramyocardial necrosis.
Fig. 15: 27-years-old female with Takotsubo syndrome and pheochromocytoma as incidental finding (red arrows). Several international authors have proposed diagnostic criteria for Takotsubo syndrome in the literature, but they are not clearly established. It is common to all of them that the presence of a pheochromocytoma is exclusionary for the diagnosis of Takotsubo cardiomyopathy. However, the possibility of supraphysiological elevations of catecholamines as etiopathogenic factor of this syndrome arises, so it seems logical that they could share the same pathophysiological mechanism with cardiomyopathy caused by pheochromocytoma. Thus, in this case, we would point out that it could be considered as a transient stress cardiomyopathy secondary to pheochromocytoma.

Fig. 16: 83-years-old female (with previous stressful situation due to anniversary of the death of the spouse) presented chest pain, ECG changes, cardiac enzymes elevation and normal coronary arteries (angiographically demonstrated). 3 days later Cardiac-MRI was performed. CINE sequence (long axis of 2 chambers) shows akinesia of LV mid segments, with preserved contractility of apical and basal segments. T2 sequence demonstrates edema in mid segment of LV anterior face (yellow arrows) in both long axis of 2 chambers (A) and short axis (B). Enhancement in late contrast sequence was not present.
**Fig. 17:** Patient with Takotsubo syndrome. ECG shows presence of symmetric T-wave inversion (red arrow).
Fig. 18: Cardiac-MRI. A 64-years-old female presented akinesia of anterior and inferior faces of LV mid and apical planes with basal hypercontractility of LV in ventriculography. Coronary angiography was normal. Cardiac-MRI was performed 5 days later. Note complete recovery of LV segmental contractility during systolic images of CINE sequence in long axis of 2 chambers. T2 sequence shows edema of the anterior and septal faces of the LV apical segments (red arrows). Alterations were not demonstrated in delayed enhancement sequence.
Fig. 19: Cardiac-MRI sequences performed in two different patients with AMI and myocarditis. AMI with normal coronariography and myocarditis: Systolic CINE 2 chambers image shows akinesia of apex and anteroapical segment of LV (yellow arrow). Preserved systolic function. In T2 short axis image, hyperintense areas are evident in the apex, corresponding to edema. Areas of transmural enhancement in apex and apical segments of septum in delayed enhancement sequence of 4 chambers (red arrow), corresponding with myocardial necrosis, without evidence of viability. Myocarditis: Images of CINE 2 chambers sequence did not show abnormalities in LV wall motion. Edema in anterior, septal and antero-lateral face of mid segments were marked (green arrows). In 4 chambers of Delayed enhancement image, nodular foci of intramyocardial enhancement (pink arrow) and linear enhancement in the pericardium at the apex (orange arrow) were observed, findings suggestive of Myopericarditis.

<table>
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Table 1: Table with MRI imaging characteristics in differential diagnosis of Takotsubo syndrome, AMI and myocarditis.
Conclusion

Through our experience with Takotsubo syndrome and literature reviewed, we expose the following conclusions:

Takotsubo syndrome or transient apical cardiomyopathy is more prevalent in postmenopausal women population, and is characterized by chest pain, variable ECG changes, moderate enzyme elevation of myocardial damage and normal angiographic study. Cardiac- MRI findings are akinesia/dyskinesia of mid and apical segments of the LV myocardium, edema unrelated to coronary vascular distribution and absence of intramyocardial necrosis. Typically the disease is preceded by an episode of emotional stress. Takotsubo syndrome has a favorable prognosis, since these changes are transient and vanish within weeks - months, although serious complications can be associated. Regarding to our knowledge of the MR findings in Takotsubo syndrome, recovery of LV myocardial contractility is corroborated in a short period of time, estimated in days. Nevertheless, changes in the signal due to myocardial edema entail a slower recovery, dissapearing in the range of weeks-months in most of patients seen in our institution.

The differential diagnosis of Takotsubo syndrome includes other causes of acute coronary syndrome with no significant lesions in the coronary arteries, mainly AMI with normal coronary arteries and myocarditis. Accurate diagnosis is crucial for the prognostic and therapeutic implications involved. In this regard, cardiac - MRI through the sequence of delayed enhancement is a very useful tool to determinate the absence of myocardial necrosis that typically characterizes the Takotsubo syndrome, although this finding alone can not be considered diagnostic.

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