

# MANAGING TAKO-TSUBO WITH DELAYED SYSTOLIC ANTERIOR MOTION AND RIGHT VENTRICLE DYSFUNCTION AFTER MITRAL VALVE REPAIR

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## Abstract

Takotsubo syndrome is a rare and serious condition, often induced by major physical or psychological stress. Both its diagnosis and treatment could be challenging; herein we describe a complex case of Takotsubo syndrome following mitral valve repair surgery associated with occurrence of systolic anterior motion of the mitral valve, severe mitral regurgitation, consequent right heart failure and its peculiar clinical management.

## INTRODUCTION

Takotsubo Syndrome (TS), also known as acute left ventricle apical ballooning syndrome or stress-induced cardiomyopathy, is a rare condition, first described in Japan in the early 1990's by Sato et al[1], characterized by hypokinesis of the left ventricle and ballooning of the apex, with hypercontractile base and non-obstructive coronary artery disease, causing a transient but potentially lethal left ventricle systolic dysfunction. Despite clinical symptoms consistent with acute myocardial infarction, normal coronary arteries are usually detected upon cardiac catheterization. It is now recognized that in addition to emotional stress, also surgical procedures and administration of exogenous catecholamines play an important role as precipitant factors of the disease. Here we describe a challenging case of TS occurring after surgery for mitral valve repair complicated with systolic anterior motion of the mitral valve (SAM), severe mitral insufficiency and right ventricular dysfunction.

## Case presentation

A 63 years-old woman was admitted at our institution in March 2020, with diagnosis of severe myxomatous mitral regurgitation. Pre-operative echocardiography showed a prolapse of both mitral leaflets and annular dilatation, preserved systolic function and normal kinesis. Coronary angiography was normal.

A mitral valve repair through a minimally invasive approach at the right fourth intercostal space was performed. Valve repair was achieved by posterior leaflet resection and annuloplasty with an open band (Futureband, Medtronic Inc.). Custodiol cardioplegia was used. No aortic insufficiency and good delivery of cardioplegia was assured. Cardiopulmonary bypass time was 114 minutes, and cross-clamp time was 82 minutes. Conversion to a full sternotomy was required due to a sudden bleeding caused by ruptured left atriotomy. No re-clamping was needed. Post-op transesophageal echocardiogram (TEE) showed good repair with only trivial mitral regurgitation and good cardiac function.

On the first post-operative day (POD), the patient suffered generalized tonic-clonic seizures. Haemodynamic instability occurred and inotropic support was required (norepinephrine 0,08 mcg/Kg/min, epinephrine 0,04 mcg/Kg/min)

TEE showed a severely reduced left ventricle ejection fraction (30%). Basal segments appeared hyperkinetic while apical segments were hypo/akinetic with typical ballooning aspect, no SAM was reported. EKG showed diffuse ST elevation. Due to an increase of myocardionecrosis markers, coronary angiography was repeated, with no new findings. Then, Takotsubo Syndrome was suspected, and pharmacological hemodynamic support was continued, adding levosimendan at 0,1 mcg/Kg/min.

On the 6th POD severe hypotension re-occurred. TEE showed slight improvement in systolic function (LVEF 44%) with persistent apical akinesis but this time with persistent hyperkinesis of the base of the left ventricle causing a new onset of SAM of the anterior mitral leaflet with left ventricle outflow tract obstruction and severe mitral regurgitation [Figure 1]. Consequent pulmonary artery hypertension and right ventricle dysfunction complicated the clinical condition. No lung injury or pulmonary oedema were present.

Inhaled nitric oxide (NO) at 20 parts per million (ppm) and sildenafil IV were immediately added.

No extracorporeal membrane oxygenation (ECMO) was needed. Aggressive fluid administration was started despite the right ventricle dysfunction and IV Esmolol was titrated as soon as the hemodynamic condition allowed a progressive reduction of inotropes (norepinephrine 0,02 mcg/Kg/min, epinephrine 0,02 mcg/Kg/min). After slow iNO weaning the patient was maintained on Sildenafil infusion until the tenth POD and oral beta-blocker therapy with bisoprolol until discharge.

The patient gradually improved, and the 21st POD echocardiogram showed a recovered LVEF (55%), no SAM and only slight apical hypokinesia with mild mitral regurgitation [Figure 2]. After 5 days the patient was discharged for rehabilitation in good conditions.

## DISCUSSION

In 2007 Itoh et al.[2] described the first intraoperative case of TS following cardio-pulmonary bypass. This was hypothesized to be caused by direct myocardial damage during the operation or a stunning subsequent to a huge catecholamines relapse [3].

In our case, probably, we had three concomitant precipitating factors: the emergency conversion to full sternotomy, the post-weaning tonic-clonic seizure and the consequent hemodynamic instability and need for high dose vasopressors agents.

Takotsubo syndrome has been classified as a transient and benign disease.[4] However, recent studies indicated that some critical complications and sudden death may occur, especially when right ventricle failure is associated.[5]

Therefore it should be considered in the differential diagnosis of patients developing sudden cardiogenic shock after surgery.

In this case, given the anatomical characteristics of a repaired mitral valve with mixomatous leaflets we had to deal not only with a disfunctioning LV but also with a SAM causing severe mitral insufficiency, pulmonary hypertension and RV failure. The management of this peculiar combination is particularly challenging, especially in the acute phase. On one hand the treatment of SAM normally involves aggressive volume loading and beta-adrenoceptor blockade, but on the other hand right ventricle function can deteriorate once this is provided, especially with the coexistence of pulmonary hypertension. Even though inotrope administration is contraindicated in patients with SAM, we found that in the acute phase levosimendan, noradrenaline and low dosages of adrenalin were helpful to sustain hemodynamics. We observed that adding esmolol to reach a heart rate between 85 and 100 bpm provided the best balance for this delicate situation. Probably, simultaneous inotropic support with low adrenalin and noradrenaline dosages and inhaled NO contributed to improve right ventricle function despite the aggressive fluid administration.

In conclusion, even though it is complex to deal with two conditions which normally require treatments that collide (SAM and right ventricle dysfunction), we believe that recognizing takotsubo syndrome and finding a good balance between betablockers, inotropes, fluid administration and inhaled NO is crucial and can be life-saving for the patient.

## REFERENCES

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## FIGURE AND VIDEO LEGENDS

Figure 1 : apical four chamber transthoracic echo showing apical ballooning with systolic anterior motion of the anterior leaflet of the mitral valve, left ventricle outflow tract obstruction and consequent severe mitral regurgitation

Figure 2 : 21<sup>st</sup> post-operative day, after recovery only mild regurgitation and an apical cap persisted.



