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Research Article

JOURNAL OF SURGERY AND RESEARCH

ISSN: 2640-1002

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.

Key clinical message: Takotsubo syndrome after cardiac surgery is insidious to recognize. Management can be challenging, especially when in combination with left ventricle outflow tract obstruction, anterior systolic motion, mitral regurgitation and right heart failure.

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 (figure 1). Its prevalence has been reported to be approximately 2% (up to 10% if only women are considered) of all patients presenting with clinical manifestation of acute coronary syndrome [2]. 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 with diagnosis of severe myxomatous mitral regurgitation. No other co-morbidities were present and BMI was 26.7 kg/m^2. 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,

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Citation: Luigi Garufi, Francesco Giosuè Irace, Andrea Salica, Ruggero de Paulis. Managing Tako-Tsubo with Delayed Systolic Anterior Motion and Right Ventricle Dysfunction after Mitral Valve Repair. Journal of Surgery and Research. 6 (2023): 140-142.

Received: March 06, 2023
Accepted: March 14, 2023
Published: April 17, 2023

Garufi L et al., J Surg Res 2023
DOI:10.26502/jsr.10020286

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), during the weaning from mechanical ventilation the patient suffered generalized tonic-clonic seizures and extubation was not reached. No history of epilepsy was found and after propofol administration no other neurological issue occurred. CT Brain Scan without contrast was performed in order to exclude neurological events. Suddenly, haemodynamic instability occured 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 (Video 1) (figure 2). Consequent pulmonary artery hypertension (detected with the swan-ganz catheter) and right ventricle dysfunction complicated the clinical condition. The Echo measurement of the fractional area change (FAC) showed a reduction to 31%. The LVOT measured gradient was 44mmHg. No lung injury or pulmonary oedema were present. No CT scan was performed to exclude pulmonary embolism as suspicion was low since the patient was on full anticoagulation because of an episode of atrial fibrillation occurred on the 4th POD. 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 (Video 2) FAC was 41%. After 5 days the patient was discharged for rehabilitation in good conditions.

Discussion

In 2007 Itoh et al.[3] 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 [4]. 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 and its prevalence after cardiac surgery is difficult to estimate [5]. However, recent studies indicated that some critical complications and sudden death may occur, especially when right ventricle failure is associated [6]. 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 myxomatous leaflets we had to deal not only with a dysfunctioning 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.

Conflict of interest

The authors declare that there are no conflicts of interest.

Ethics statement

IRB approval was obtained.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author.

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DOI:10.26502/jsr.10020286

Consent statement

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy

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