Case report - Cardiac general

Takotsubo cardiomyopathy after elective mitral valve replacement

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Abstract

Takotsubo cardiomyopathy is a syndrome characterized by transient acute left ventricular dysfunction, electrocardiographic changes that can mimic acute myocardial infarction and minimal release of myocardial enzymes in the absence of obstructive coronary artery disease. Reports of Takotsubo syndrome after cardiac surgery are exceptional. We describe a case of Takotsubo cardiomyopathy in a 68-year-old woman after elective mitral valve replacement. Takotsubo syndrome should be considered in the differential diagnosis of patients presenting cardiogenic shock after cardiac surgery.

Keywords: Takotsubo cardiomyopathy; Transient left ventricular apical ballooning syndrome; Stress-induced cardiomyopathy; Mitral valve replacement; Cardiac surgery; Cardiogenic shock

1. Introduction

Takotsubo cardiomyopathy is a cardiac syndrome characterized by transient left ventricular dysfunction, electrocardiographic changes that can mimic acute myocardial infarction and minimal release of myocardial enzymes in the absence of obstructive coronary artery disease [1]. This syndrome is also known as the transient left ventricular apical ballooning syndrome, ampulla-shaped cardiomyopathy, ‘broken heart syndrome’ or stress-induced cardiomyopathy, because stress has been implicated in its pathophysiology [2, 3]. This syndrome was first described in 1991 in Japan, and patients were usually postmenopausal women after experiencing an inciting stressful event [4]. More recently, case series have been reported in Caucasian populations in Europe and North America [1, 5], but reports of Takotsubo syndrome after cardiac surgery are exceptional [2].

We describe a case of Takotsubo cardiomyopathy in a patient after elective mitral valve replacement.

2. Case report

A 68-year-old woman was admitted for elective mitral valve replacement for severe mitral valve disease with severe pulmonary hypertension and congestive heart failure (New York Heart Association functional class III). Her past medical history included rheumatic heart disease, permanent atrial fibrillation, cerebrovascular accident with no residual deficit, and anxiety disorder. Preoperative echocardiography confirmed severe mitral regurgitation, severe mitral stenosis and normal LV function. Preoperative diagnostic coronary angiography revealed no coronary lesions. Through a median sternotomy on standard cardiopulmonary bypass and cardiac arrest, the mitral valve was approached through a transseptal incision extended into the roof of the left atrium. The native mitral valve was replaced with a 31-mm On-X prosthesis (On-X Life Technologies Inc, Austin, TX, USA), preserving posterior mitral leaflet and annulo-ventricular continuity. Cardiopulmonary bypass time was 83 min and aortic cross-clamp time was 68 min. After uneventful weaning off cardiopulmonary bypass, intraoperative transesophageal echocardiography revealed adequate mitral prosthetic function, normal global and regional left ventricular contractions, and normal right ventricular function. The patient was transferred to the intensive care unit and hemodynamic parameters suddenly deteriorated in the first minutes of stay in the unit. Low blood pressure failed to respond to intravascular fluid and progressive higher doses of noradrenaline and adrenaline were needed. An electrocardiogram showed atrial fibrillation, with a ventricular rate of 120, with negative T-waves in the precordial leads. Urgent echocardiogram showed severe mid-ventricular dysfunction and apical akinesis, with hyperdynamic basal segments contraction (Fig. 1). The LV ejection fraction was estimated to be 15–20%. Intra-aortic balloon pump was added to inotropic support to improve management of cardiogenic shock. The increase of cardiac enzyme levels did not confirm perioperative myocardial infarction, and hemodynamic conditions allowed to abruptly stop inotropic support on postoperative day 3. Daily transthoracic echocardiography showed gradual improvement of LV function with LV ejection fraction returning to 55% on postoperative day 6. This fact, the pattern of LV wall motion abnormalities

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observed, and the absence of angiographic coronary arterial disease, allowed to establish the diagnosis of Takotsubo syndrome. On postoperative day 7, the patient was transferred to a standard hospitalization area and beta-blockers treatment was initiated. On postoperative day 11, transthoracic echocardiography showed normal LV function with LV ejection fraction of 65% and normal pulmonary arterial pressure. She was discharged home on postoperative day 12. Follow-up echocardiography at one, three and six months demonstrated normal LV function with a LV ejection fraction of 65–75% (Fig. 2).

3. Comments

Takotsubo cardiomyopathy has been observed most commonly in postmenopausal women after an episode of acute emotional or physiological stress, general surgery, hypoglycemia, and hyperthyroidism [3, 5]. Proposed Mayo Clinic Criteria [1] for the diagnosis of the Takotsubo cardiomyopathy are: (1) transient akinesis or dyskinesis of the left ventricular apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution; (2) absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (3) new electrocardiographic abnormalities (either ST-segment elevation or T-wave inversion); and (4) absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, and hypertrophic cardiomyopathy.

Although the pattern of LV wall-motion abnormalities and clinical course are characteristics, other differential diagnosis such as poor myocardial protection and coronary air embolism should be considered in this case. Suboptimal myocardial protection is highly unlikely with normal LV function after weaning off cardiopulmonary bypass. Coronary air embolism is also improbable in this patient, because this entity affects more frequently to the right ventricle and its persistence for several hours would determine myocardial infarction consistent with some particular coronary artery territory.

The exact cause of Takotsubo syndrome is unknown [5]. The most accepted hypothesis shows it may represent a catecholamine-mediated myocardial stunning that results from a combination of myocardial ischemia related to diffuse microvascular dysfunction and, in some cases, multivessel epicardial spasm and metabolic injury [6, 7]. The explanation for the strong female predominance with the syndrome is also unclear. However, the explanation may be related to postmenopausal alterations of endothelial function in response to reduced estrogen levels and microcirculatory vasomotor reactivity in response to catecholamine-mediated stimuli [3, 6].

The prognosis of patients experiencing this syndrome is generally favourable; despite the frequently dramatic clinical presentation, almost all patients recover fully and the
LV systolic function, heavily compromised at presentation, improves rapidly in a period of days to weeks [1, 5–7].

Reports of Takotsubo syndrome after open cardiac surgery are few. It is possible that some cases of transient LV sphericalization after mitral valve replacement surgery described previously to the recognition of this phenomenon were cases of Takotsubo syndrome [2].

It is important to consider Takotsubo syndrome in the differential diagnosis of patients presenting cardiogenic shock after cardiac surgery. Dyskinetic apical and mid-ventricular segments with concomitant hyperdynamic basal segments may result in hemodynamically significant dynamic left ventricular intracavitary obstruction [1, 7, 8]. Hemodynamic impairments in this situation require significantly different management from that needed for hypotension due to pure pump failure. In this hemodynamic situation, it is recommended the administration of beta-blockers to increase diastolic ventricular filling time and left ventricular end-diastolic volume, administration of phenylephrine to increase afterload with subsequent reduction of the intraventricular gradient, and administration of fluid resuscitation if pulmonary congestion is not present.

References


eComment: Re: Takotsubo cardiomyopathy after elective mitral valve replacement

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Stress cardiomyopathy is a novel clinical syndrome affecting predominantly elderly female patients. The authors describe one of the first cases of Takotsubo cardiomyopathy after elective mitral valve replacement [1]. Clinical and diagnostic aspects accumulated and described in this article are important for understanding the pathophysiology of this disorder.

Our data show that this pathology concerns 1% of individuals with suspected acute myocardial infarction [2]. Among the patients with Takotsubo cardiomyopathy the left ventricular dysfunction is likely to recover rapidly, but the management of implications (contrary to atherosclerotic myocardial infarction/ischemia) are likely different. Although different complications (17%, 109/625), such as dysrhythmias (39%, 32/82), heart failure (50%, 41/82), and even cardiogenic shock (23%, 18/40 with acute heart failure) occurred, the prognosis appeared to be much better [2]. Moreover, some patients required intravenous dopamine or dobutamine infusion and mechanical supports, including a percutaneous cardiopulmonary support system. Although almost all patients with this syndrome were reported to have a favorable prognosis (mean period of recovery 11 ± 1 days [2], EF on admission 41.3 ± 1.9% vs. EF (recovery) 63.6 ± 1.0% (P = 0.000) [2]), careful clinical observation for critical complications or recurrences is recommended, especially after cardiovascular interventions, as described in the presented case [1]. In our clinic, we have observed two clinical cases of Takotsubo cardiomyopathy after excessive emotional stress: one in an elderly woman (52 years) and one in a young man (23 years) [2].

We agree that it is important to consider Takotsubo syndrome in the differential diagnosis of patients presenting cardiogenic shock after cardiac surgery and that the administration of beta-blockers in this hemodynamic situation after selective mitral valve replacement is recommended as well as the administration of fluid resuscitation if pulmonary congestion is not presented. The mechanism of this modality remains unclear, although exaggerated sympathetic simulation has been considered to play a major role [3, 4]. Several mechanisms were suggested as the leading factors: coronary vasospasm (epicardial and microvascular ischemia), catecholamine-mediated toxicity, excessive sympathetic activation.

A number of unanswered questions remain for this reversible form of cardiomyopathy. Why do middle aged/elderly women appear particularly susceptible to this disorder (mean age 66.2 ± 1.2 [2])? How does profound stress trigger its sudden onset? Why is the left ventricular apex selectively vulnerable to regional ballooning?

Obviously, we urgently need more information on the pathophysiology and optimal treatment of this syndrome. Research of this disorder, especially after open heart surgery, is actual and should be carried out in centers for cardio-vascular surgery to create optimal diagnostic criteria and treatment.

References