



Case Report

Takotsubo Cardiomyopathy after Elective Aortic and 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 (CAD). Reports of Takotsubo syndrome after cardiac surgery are exceptional. We describe a case of Takotsubo cardiomyopathy in a 57-year-old man after elective aortic and mitral valve replacement following recent convalescence from infective endocarditis. Takotsubo syndrome should be considered in the differential diagnosis of patients presenting acute myocardial infarction, cardiogenic shock or any low cardiac output syndrome after cardiac surgery.

Keywords

aortic valve replacement, mitral valve replacement, postoperative acute myocardial infarction, Takotsubo cardiomyopathy

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 (CAD). This syndrome is also known as 'broken heart' syndrome, am-pulla cardiomyopathy, transient left ventricular apical ballooning syndrome, apical ballooning syndrome, transient left ventricular dysfunction syndrome, and stress induced cardiomyopathy because stress itself has been implicated in its pathophysiology. This syndrome was first described in 1990 and 1991 in Japan by Satoh et al. and Dote et al.¹⁻⁴ It was initially characterized by unique pattern of transient (hours to weeks) wall motion abnormality - transient left ventricular apical ballooning, occurring in the absence of significant epicardial coronary artery disease, presenting as an acute coronary syndrome, most frequently in postmenopausal elderly women and is often triggered by

stressful situations. Left ventriculography reveals a peculiar shape of the left ventricle resembling a 'takotsubo', which, in fact, is a type of bottle with a round bottom and a narrow neck, used in Japan for trapping octopus.⁴ More recently, series of cases have been reported in Caucasian population in Europe and North America, but reports of Takotsubo syndrome after cardiac surgery are extremely rare. We describe a case of Takotsubo cardiomyopathy in a patient after elective aortic and mitral valve replacement following recent convalescence from infective endocarditis.

CASE REPORT

A 57-year-old man, with history of recent convalescence from infective endocarditis rheumatic fever in childhood, congestive heart failure (New York Heart Association functional class III) and concomitant chronic obstructive pulmonary disease (COPD), and HbsAg positive test confirmation, was admitted for elective aortic and mitral valve replacement.

Preoperative transthoracic and transesophageal echocardiography demonstrated normal left ventricular ejection fraction: (EF - 52% according to Teicholz M-mode; EF - 50% according to Simpson's method). The native aortic valve was tricuspid with vegetations, with severe aortic regurgitation (grade 4) and transvalvular systolic peak gradient - 15 mm Hg. The echocardiography revealed mild left ventricular hypertrophy; interventricular wall thickness - 14 mm, posterior left ventricular wall thickness - 13 mm and the following measurements, regarding dimensions and volumes: left ventricular end-diastolic dimension - LVEDD-66 mm; left ventricular end-systolic dimension - LVESD-47 mm; left ventricular end-diastolic volume- LVEDV-213 ml; and left ventricular end-systolic volume - LVESV-106 mm. The dimensions of the left atrium were 60/72 mm. The transthoracic and transesophageal echocardiography demonstrated and revealed the native mitral valve with Carpentier's Functional Classification Type II moderate to severe mitral regurgitation (grade 3-4), due to leaflet prolapse and vegetations upon the anterior mitral leaflet (AML). The tricuspid valve had intact valvular apparatus but dilated annulus, resulting in moderate tricuspid valve regurgitation (grade 2). The pulmonary artery pressure was elevated - 53 mm Hg. The right atrial dimension was 62/56 mm. and the right dimension - 42 mm. The latter echocardiographic findings revealed normokinetic left ventricular segments, no pericardial effusion and adhesions but bilateral pleural effusions: left pleural cavity - 600 ml; right pleural cavity - 1000 ml. Preoperative coronary angiography and left ventriculography demonstrated right dominant coronary circulation and atherosclerosis-free epicardial coronary arteries. Ventriculography demonstrated grade 4 severe mitral valve insufficiency. Aortography - grade 4 severe aortic valve insufficiency and no evidence of aortic dissection. Perioperative 12-lead ECG showed sinus rhythm with frequency of 80 beats per minute and an incomplete right bundle branch block. Abdominal echography showed hepatomegaly, ascites and chronic parenchymal process in both kidneys. In conclusion, the preoperative echocardiography confirmed severe aortic and mitral valve regurgitation, normal left ventricular function, and the preoperative diagnostic coronary angiography revealed no coronary lesions. The operation was performed through a median sternotomy and standard total cardiopulmonary bypass and blood cardioplegia-induced cardiac arrest. The native mitral valve was replaced by a mechanical bileaflet Sorin Bicarbon 33 mm heart valve prosthesis (Sorin Group Milan, Italy). The aortic valve was replaced by another mechanical bileaflet Sorin Bicarbon 27 mm heart valve prosthesis. Cardiopulmonary bypass time was 96 minutes, aortic cross-clamp time was 63 minutes and reperfusion time was 22 minutes. The extracorporeal circulation weaning was uneventful. The patient was transferred to the intensive care unit where hemodynamic parameters suddenly deteriorated in the first postoperative hours. The low cardiac output syndrome failed to respond to infusion therapy and progressively higher doses of catecholamines-dopamine and dobutrex were needed to obtain normal hemodynamics. An electrocardiogram showed sinus rhythm of 90 beats per minute with ST-segment

elevations in lead I, a VL and precordial V2-V6 leads. Urgent echocardiography showed severe mid-ventricular dysfunction and apical akinesia with hyperdynamic basal segments contraction. The left ventricular ejection fraction decreased and was measured 28-30%. The increase of cardiac serum marker - cardiac-specific troponin T- 2.42 mg/L, normal range (0-0.1 mg/L, SI Units), confirmed the probable diagnosis of perioperative myocardial infarction. The patient was urgently transferred to a catheterization laboratory for angiography which demonstrated no obvious reason for the development of an acute myocardial infarction. Contrast ventriculography, which could demonstrate wall motion abnormalities and deterioration of the ejection fraction, was not performed because of the obvious theoretical and practical difficulties in positioning the catheter through the mechanical aortic valve prosthesis into the left ventricle, and all the concomitant risks for damaging the valve prosthesis, the catheter itself, and any mechanical trauma to the heart and the aorta that could jeopardize the life of the patient. Having demonstrated no coronary artery disease that could be responsible for the development of the acute myocardial infarction, no additional intervention was needed and the patient was transferred back from the Cath lab to the cardiac intensive care unit. Intravenous direct anticoagulation was started and low doses of catecholamines and vasoactive drugs supported the hemodynamic state of the patient during the first 24 hours postoperatively. The diagnosis of Takotsubo syndrome was established after excluding the possibility that such a low cardiac output syndrome, mimicking an acute myocardial infarction, can be a result of an absent coronary artery disease. On postoperative day 2, the decrease in the serum troponin and the levels of cardiac biomarkers - creatine-phosphokinase and its CK-MB isoenzyme, which were in normal ranges after the surgical intervention, led to the total rejection of the diagnosis acute myocardial infarction. Daily transthoracic echocardiography showed gradual improvement of the left ventricular function with ejection fraction returning to 47% on postoperative day 10. The patient was discharged on postoperative day 14. Follow-up echocardiography one week after discharge revealed 48% ejection fraction and normal functioning of the mechanical valve prostheses. This routine examination was the final point in determining diagnosis of postoperative Takotsubo syndrome.

DISCUSSION

Takotsubo cardiomyopathy has been observed most commonly in postmenopausal period, an episode of acute emotional or physiological stress, general surgery, hypoglycemia and hyperthyroidism. Goustova, Bockeria et al., from Bakoulev Scientific Center for Cardiovascular Surgery Moscow, Russia, reported meta-analysis of apical ballooning syndrome. The data demonstrated that this pathology concerns 1% of the individuals with suspected acute myocardial infarction. Among patients with Takotsubo cardiomyopathy the left ventricular dysfunction is likely

to recover rapidly, contrary to atherosclerotic myocardial ischemia or infarction, which progression is quite different. Although various complications may occur, such as dysrhythmias, heart failure, even cardiogenic shock, the prognosis of the disease appears to be good. Moreover, some patients require dopamine or dobutamine (catecholamine) infusion and mechanical support, including a percutaneous cardiopulmonary support system. Although almost all patients, researched by Goustova, Bockeria et al., with this novel clinical syndrome, were reported to have a favourable prognosis (mean period of 11 ± 1 days, ejection fraction (on admission) $41.3 \pm 1.9\%$ vs. ejection fraction (on recovery) $63.6 \pm 1.0\%$ ($p=0.000$), careful clinical observation for critical complications or recurrences, is recommended, especially after cardiovascular interventions.² The proposed by Mayo Clinic Criteria for establishing the diagnosis of the Takotsubo cardiomyopathy are: transient akinesia or dyskinesia of the left ventricular apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution; absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; new electrocardiographic abnormalities, (either ST-segment elevation or T-wave inversion); and absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, myocarditis and hypertrophic cardiomyopathy. Although the pattern of left ventricular wall-motion abnormalities and clinical course are typical, other differential diagnosis such as inadequate myocardial protection and coronary air embolism should be considered in this case. Suboptimal myocardial protection is highly unlikely with normal left ventricular function after cardiopulmonary bypass. Coronary air embolism is also improbable in this patient, because such cases affect more frequently the right ventricle and its presence for several hours would determine myocardial infarction in the area of a particular coronary artery. The exact cause of Takotsubo syndrome is unknown. The most accepted hypothesis suggests that it may represent a catecholamine-mediated myocardial stunning that results from a combination of myocardial ischaemia, related to diffuse microvascular dysfunction and in some cases, multivessel epicardial spasm or metabolic injury. The prognosis of patients experiencing this syndrome is generally favorable. Despite the dramatic clinical presentation, almost all patients fully recover and the left ventricular function, heavily compromised at presentation, and rapidly improves in a period of days to weeks. Reports of Takotsubo syndrome after open heart surgery are few. Kogan in Israel described Takotsubo syndrome after elective mitral valve replacement and tricuspid valve repair.³ Ohata et al. in Japan described the syndrome after replacement of a prosthetic aortic valve after prosthetic valve endocarditis.⁵ Belazquez et al. in Spain observed apical ballooning syndrome after elective mitral valve replacement.⁶ Wong in Hong Kong described Takotsubo syndrome after extirpation of a left atrial myxoma.⁷ It is possible that some cases of transient left ventricular sphericalization after mi-

tral valve replacement, which remained unexplained before the recognition of this phenomenon, were cases of Takotsubo syndrome. It is important to consider this disease in the differential diagnosis of patients presenting with acute myocardial infarction and cardiogenic shock after cardiac surgery. Dyskinetic apical and mid-ventricular segments with concomitant hyperdynamic basal segments may result in hemodynamically significant left ventricular intracavitary obstruction. Hemodynamic impairments in this situation require different management from that needed for hypotension due to pure pump failure. In this special hemodynamic situation, the administration of betablockers is recommended to increase the diastolic ventricular filling time and left ventricular end-diastolic volume. Administration of phenylephrine to increase afterload with subsequent reduction of the intraventricular gradient and fluid therapy, if pulmonary congestion is not present.

CONCLUSION

Many questions remain unanswered for this reversible form of cardiomyopathy. Obviously, we urgently need more information about the pathophysiology and the optimal treatment of this syndrome. Research concerning this disorder, especially after open cardiac surgery, is crucial and should be carried out in all the cardiovascular centres to create optimal diagnostic criteria and treatment.

REFERENCES

1. Dote K, Satoh H, Tateishi H, et al Myocardial stunning due to simultaneous multivessel coronary spasm : a review of 5 cases. *J Cardiol* 1991; 21: 203-14.
2. Goustova IA, Bockeria LA, Bockeria OL. Meta-analysis of apical ballooning syndrome data ('Takotsubo' cardiomyopathy). [Dissertation]. Bakoulev scientific center for cardio-vascular surgery RAMN, Moscow. 2010:1-181.
3. Kogan A, Ghosh P, Schwammenthal E, et al. Takotsubo syndrome after cardiac surgery. *Ann Thorac Surg* 2008; 85(4): 1439-41.
4. Tsuchihashi K, Ueshima K, Uchida T, et al Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction; Angina Pectoris-Myocardial Infarction Investigations in Japan. *J Am Coll Cardiol* 2001; 38: 11-8.
5. Ohata T, Ueda H, Kamata S, et al. Fulminant apical ballooning syndrome after replacement of a prosthetic aortic valve. *Gen Thorac Cardiovasc Surg* 2009; 57: 477-80.
6. Blazquez JA, Gonzalez JM, Dalmau MJ, et al. Takotsubo cardiomyopathy after elective mitral valve replacement. *Interact CardioVasc Thorac Surg* 2010; 11: 117-9.
7. Wong CP, Jim MH, Chan OA, et al. Iatrogenic Takotsubo cardiomyopathy. *Journal of Cardiac Surgery* 2010; 25(6): 679-83.

Кардиомиопатия такоцубо после замены аортального и митрального клапана по желанию пациента.

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Абстракт

Кардиомиопатия такоцубо - это синдром, характеризующийся преходящей острой дисфункцией левого желудочка, электрокардиографическими изменениями, которые могут имитировать острый инфаркт миокарда и минимальную продукцию ферментов миокарда в отсутствие обструктивной ишемической болезни сердца (ИБС). Сообщения о синдроме такоцубо после операции на сердце редки. Мы описываем случай кардиомиопатии такоцубо у 57-летнего мужчины после замены аорты и митрального клапана по выбору пациента после недавнего выздоровления от инфекционного эндокардита. Синдром такоцубо следует учитывать при дифференциальной диагностике пациентов с острым инфарктом миокарда, кардиогенным шоком или синдромом низкого сердечного выброса после операции на сердце.

Ключевые слова

кардиомиопатия такоцубо, замена аортального клапана, замена митрального клапана, послеоперационный острый инфаркт миокарда
