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). 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.

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 Teichholz 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 leaflet prolapse and vegetations upon the anterior mitral leaflet (AML). The tricuspid valve had intact valvar 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 segment, 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 markers - 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
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

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, экстрасистолическими 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