Case Series

Takotsubo stress cardiomyopathy, a great mimicker of myocardial infarction in post-operative period: what surgeons should know? a case series

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ABSTRACT

Takotsubo stress cardiomyopathy (TCM) is a rare disorder of the myocardium precipitated mainly by physical, mental stress or sometimes due to non-specific factors. The clinical presentation of takotsubo cardiomyopathy is an eidetic image of CAD and myocardial infarction (MI), but unlike the later takotsubo cardiomyopathy in most cases is reversible. Coronary angiographic evidence showing the absence of narrowing of coronary vessels is the only definitive way to distinguish this from MI. This condition is a less familiar entity to the surgeons and knowing this will help in arriving at the diagnosis early and have an optimistic attitude towards management because the condition is a self-limiting and good recovery is seen generally with supportive management. In this series, 1056 HPB cases and 101 liver transplant cases done from January 2016 to February 2021 and among them we describe four cases who developed TCM in post-operative period and discuss the relevant literature.

Keywords: Takotsubo stress cardiomyopathy, Hepatobiliary, Myocardial infarction, Case series

INTRODUCTION

Takotsubo stress cardiomyopathy (TCM) is a rare disorder of the myocardium precipitated mainly by physical, mental stress or sometimes due to non-specific factors.1 It was first described in 1990 in patients presenting with symptoms of coronary artery disease (CAD).2 In this condition, the shape of the left ventricle resembles ‘takotsubo’ which in Japanese denotes a fishing pot used to catch octopus. The apex of the left ventricle shows hypo-kinesis and ballooning which is characteristic feature.3 The clinical presentation of TCM is an eidetic image of CAD and myocardial infarction (MI), but unlike the later takotsubo cardiomyopathy in most cases is reversible.1,3 Coronary angiographic evidence showing the absence of narrowing of coronary vessels is the only definitive way to distinguish this from MI. Although the condition is usually reversible and not fatal, prompt identification is necessary to prevent complications like left ventricular outflow tract (LVOT) obstruction and also to avoid unwanted interventions like percutaneous coronary intervention (PCI) or thrombolysis in the event of misdiagnosis as MI. This condition is a less familiar entity to the surgeons and knowing this will help in arriving at the diagnosis early and have an optimistic attitude towards management because the condition is a self-limiting and good recovery is seen generally with supportive management.

CASE SERIES

In this series, 1056 HPB cases and 101 liver transplant cases done from January 2016 to February 2021 and among them we describe four cases who developed takotsubo cardiomyopathy in post-operative period and discuss the relevant literature.
Table 1: Details of the cases of TCM.

| Parameters                          | Case 1                        | Case 2                                      | Case 3                                      | Case 4                                      |
|------------------------------------|-------------------------------|---------------------------------------------|---------------------------------------------|---------------------------------------------|
| Age (years)                        | 40                           | 65                                         | 54                                         | 69                                         |
| **Primary diagnosis**              | Open splenectomy for splenic abscess (failed PCD drainage) | End stage liver disease (ESLD) due to alcohol underwent successful orthotopic liver transplant (OLT) from a deceased donor | Alcohol related ESLD underwent living donor liver transplant (LDLT) | Cholelithiasis and choledocholithiasis resulting in cholangitis with septicemia. He underwent endoscopic retrograde cholangio pancreateography (ERCP) with sphincterectomy, common bile duct (CBD) stone removal and stenting |
| Comorbidities                      | None                         | BMI 33, diabetes mellitus (DM)              | None                                       | DM, HTN                                    |
| Personal history                   | Chronic alcoholic and smoker  | Chronic alcoholic and smoker                | Chronic alcoholic                           | Chronic alcoholic                           |
| **Initial presentation of cardiac problem** | Hypotension and diaphoresis on POD 2 | POD 2, he developed hypotension, desaturation and was found to have pulmonary edema | POD 4 he developed severe hypotension and desaturation, white out lung on chest X-ray. Initially a TRALI was suspected | Pulmonary edema and hypotension |
| ECG                                | Ventricular tachycardia (VT), poor R wave progression and mildly prolonged QT interval | Poor R wave progression.                   | ST elevation, left bundle branch block with QT prolongation | Sinus tachycardia |
| Cardiac enzymes troponin T ng/ml   | 4.5                          | 6.5                                        | 3.4                                        | 3.6                                        |
| 2D echo                            | Akinesia of mid, distal anterior septum, apex and apico-antero-lateral walls, moderate left ventricular dysfunction with an ejection fraction (EF) of 30% | Apical segment ballooning with basal segment hypo-kinesia and severe LV dysfunction with an EF of 32% | Severe basal hypokinesia with EF 28% | Apical ballooning with basal segment hypokinesis and LV dysfunction with an EF of 35% |
| Coronary angiogram                 | Normal                       | Angiography could not be performed in this case as he had post-transplant acute kidney injury | Normal                                     | Normal                                     |
| Treatment                          | Low molecular weight heparin (LMWH), diuretics, beta blockers, inotropes and supportive care. Patient had another episode of VT which was reverted after pharmacological cardioversion. A cardiac pacemaker was inserted | Iontropic support, diuretics, anti-coagulants and needed mechanical ventilation | Supportive management with inotropes and diuretics. | Diuretics, nitro-glycerine and antihypertensive. Supportive measures like mechanical ventilation |

Continued.
| Parameters | Case 1 | Case 2 | Case 3 | Case 4 |
|------------|--------|--------|--------|--------|
| Outcome    | Recovered | Recovered | There was improvement of the cardiac condition, the lungs worsened and the patient succumbed due to respiratory infection and sepsis | Recovered |

There was improvement of the cardiac condition, the lungs worsened and the patient succumbed due to respiratory infection and sepsis.

DISCUSSION

Takotsubo cardiomyopathy is also known as ‘broken heart syndrome’, ‘apical ballooning syndrome’ or ‘stress cardiomyopathy’ which is synonymous with its etiology.1 The prevalence of takotsubo cardiomyopathy among patients suspected to have acute coronary syndrome (ACS) is around 0.5-2.3%.4,5 Kurowski et al have reported that 35 patients (1.2%) of 2944 patients who underwent left atrial catheterisation for troponin positive ACS had cardiomyopathy with wall motion defects. Similarly, Cangella and group found that 6 (0.5%) out of 1674 patients with ACS admitted over a period of 6 years, had TCM.6 The International Takotsubo Registry, which is a consortium of several centres in America and Europe has identified a total of 1750 patients with takotsubo cardiomyopathy and has given an insight on its epidemiology and complications.7 This condition is more common among old aged women with mean age of presentation 61-76 years. According to the Takotsubo Registry, takotsubo cardiomyopathy is precipitated by physical triggers (36%), emotional triggers (27%) or both (7.8%) and no evident precipitating factor (28.5%). Emotional triggers were more common among females and physical triggers among men.8,9 The pathogenesis of...
takotsubo cardiomyopathy is unknown. Various theories have been proposed. The Catecholamine theory, has shown to cause takotsubo by stimulus trafficking. According to this theory, Catecholamine levels are elevated in Takotsubo patients when compared to normal subjects as well as those with CAD. At high levels of circulatory catecholamine, there is a switch in the beta 2 receptor intracellular cascade of the myocyte from Gs to Gi. Gi, unlike its counterpart inhibits myocardial contractility which might explain the hypo-kinesis.

As per the second theory, coronary vessel spasm and reduced myocardial perfusion have been suggested as a cause for Takotsubo. Takotsubo is characterised by ST segment elevation and it has been noticed that giving nicorandil to the patient has shown to reverse it. There is documented evidence for decreased myocardial perfusion in Takotsubo. Studies done using Doppler and contrast echocardiography have given conflicting results with regard to coronary vasospasm as causative factor for takotsubo. Other factors that have been described include microvascular dysfunction and outflow tract obstruction that may precipitate apical dilatation. Based on the Takotsubo Registry retrosternal chest pain, dyspnea and syncope are the most common presenting symptoms. Signs and symptoms of heart failure, cardiogenic shock, tachyarrhythmias and systolic murmurs may be occasionally present. ST elevation which mimics ST elevation MI is the most common ECG finding. ST segment depression, QT prolongation, T wave inversion, abnormal Q waves and other non-specific changes can also occur. Troponins are elevated in takotsubo, but their levels are lower than that seen in acute MI. Creatinine kinase and ANP are mildly elevated. Echocardiography shows LV systolic dysfunction which is characterised by bradykinesia, hypokinesia or dyskinesia of the myocardial wall along with myocardial dilatation. Myocardial dilatation can be apical (most common), mid ventricular (second most common), basal (reverse or inverted takotsubo), focal and global. Inverted takotsubo cardiomyopathy, is more prevalent in females and is characterised by basal ballooning of the left ventricle which is triggered by stress factors.

A few differential diagnosis were to considered before making the diagnosis of takotsubo cardiomyopathy. Cocaine induced cardiomyopathy, myocarditis and pheochromocytoma can present as wall motion abnormalities without coronary arterial involvement. Acute brain injury can also present as cause reversible global or focal cardiomyopathy. These conditions must be ruled out. The Mayo clinic has formulated specific diagnostic criteria which are as follows: (a) LV systolic dysfunction with bradykinesia, hypokinesia or dyskinesia. The movement abnormality is regional encompassing multiple coronary arterial territories; (b) absence of obstructive coronary disease or angiographic evidence of acute plaque rupture. Even if a coronary cause is present, a diagnosis of takotsubo can be made if the wall motion abnormalities are not in the distribution of the coronary
disease; (c) new ECG changes; and (d) absence of pheochromocytoma or myocarditis. Management of stress cardiomyopathy is usually supportive and symptomatic. Supportive therapy along with elimination of the physical or emotional stressors reverses the changes seen in the condition. Several drugs have been evaluated for their preventive role in stress cardiomyopathy. Beta blockers with intrinsic sympathomimetic activity like celiprolol, calcium channel blockers like amlodipine, azelnidipine have been shown to improve cardiac functions in TCM. Further studies are needed to better understand the pathogenesis of takotsubo cardiomyopathy. Some patients may develop complications like heart failure, cardiogenic shock, pulmonary edema, ventricular rupture and arrhythmias due to QT prolongation which must be managed appropriately. The prognosis is generally favourable and since most of the patients with Takotsubo will present as a case of CAD, a high index of suspicion is needed to make the diagnosis. In our series of 1056 HPB cases and 101 liver transplant cases done from January 2016 to February 2021 we managed four such cases as described above.

The incidence of takotsubo cardiomyopathy in post liver transplant setting though rare has been well proven. It requires careful suspicion of the condition and is usually a diagnosis of exclusion. Treatment of the condition in post liver transplant setting is not much different. Standard approaches with diuretics, and inotropic and vasopressor support are the mainstays of initial management. However, if these fail, percutaneous devices for circulatory support need to be considered.

Intra-aortic balloon pumps are used acutely in the setting of hypotensive crises secondary to acute coronary syndromes. However, they are rarely considered as a bridge to myocardial recovery. Patients undergoing OLT for alcohol related ESLD are more susceptible for TCM due to pre-existing variable degree of alcoholic cirrhosis induced cardiomyopathy and an associated imbalance and/or fluctuations in the catecholamine levels during the immediate perioperative period. These patients will benefit by a routine screening ECHO on post-operative day 2 and day 4 to identify any degree of cardiac dysfunction and address them early.

CONCLUSION

This condition is a less familiar entity to the surgeons and knowing this will help in arriving at the diagnosis early and have an optimistic attitude towards management because the condition is a self-limiting and good recovery is seen generally with supportive management. We suggest a modification of postoperative management with routine ECHO on day 2 and day 4 for early identification and management.

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