

TAKOTSUBO CARDIOMYOPATHY DURING LIVING RELATED LIVER TRANSPLANTATION

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Abstract:

53 years old female patient with end stage liver disease was scheduled to undergo liver transplantation. She developed acute severe left ventricular failure with hemodynamic instability after induction of anesthesia. Echocardiography demonstrated unusual pattern of regional wall motion abnormalities with akinesia involving all segment except the base. Coronary angiography was normal, a picture consistent with Takotsubo cardiomyopathy.

Introduction

The Takotsubo, a traditional Japanese octopus pot or trap with a wide base and tapered top, is similar in appearance to the affected left ventricle, with its hyperdynamic base and akinetic or dyskinetic apex, often extending to the entire mid-heart.

We describe a patient with this rare condition who developed acute severe left ventricular failure after induction of anesthesia during living related liver transplantation.

Case Report:

A female patient aged 53 years old weighing 61 kg was scheduled to undergo living related liver transplantation. She was neither diabetic nor hypertensive. The indication for transplantation was end stage liver disease post hepatitis c cirrhosis. Preoperative work up included resting echocardiography and Dobutamine stress echocardiography. Both of the studies revealed no regional wall motion abnormalities with good systolic function Ejection fraction (EF) 63%. After arrival to operating room the patient was stressed but no pre-medication was given. Patient was monitored for noninvasive arterial blood pressure, 5 leads Electrocardiography ECG, peripheral oxygen saturation SPO₂, end-tidal carbon dioxide tension. General anesthesia was induced with 70 mg propofol, 10 mg cisatracurium, and 200 µg, fentanyl. Anesthesia was maintained with sevoflurane 2% in O₂ FIO 1.0. Arterial catheter 18 G was inserted into the right Femoral artery and blood pressure was 130/70. 10 minutes after induction of anesthesia the end tidal CO₂ fell from 34 to 19 mmHg, blood pressure dropped to 80/60

mmHg and acute massive pulmonary edema had occurred. Transesophageal Echo was done and revealed severe dysfunction of left ventricle. Adrenaline infusion was started at 0.15 µg/kg/min. Patient was transferred to surgical intensive care intubated. In the ICU the patient ventilated with pressure controlled ventilation inspiratory pressure 20 cmH₂O PEEP 10 cmH₂O, FIO₂ 1.0. SPO₂ was 87%. 12 leads ECG showed sinus tachycardia; heart rate 130 beat/min, prolonged QT interval, inverted T wave but no ST segment elevation or depression. Chest X-ray showed severe pulmonary congestion.

A transthoracic echocardiogram was remarkable for global akinesia of the entire left ventricle (LV) except for the base of each wall, which functioned normally. EF was 15%. Figure 1 Moderate mitral regurgitation was evident. The laboratory studies were unremarkable except for troponin T elevated at 0.6 µg/L (normally <0.1 µg/l), creatinine kinase (CK), and CKMB were normal. Emergent cardiac catheterization demonstrated normal coronary arteries. Figure 2. The decision was taken to support the circulation with intraaortic balloon counterpulsation (IABP). Transthoracic echocardiography performed 1 day later showed normal global and regional left ventricular function. 1 day later Adrenaline infusion was stopped, IABP was removed and patient's trachea was successfully extubated. The patient was diagnosed to have Takotsubo cardiomyopathy.

Discussion:

Recent cardiology literature, primarily describing elderly female patients, has introduced an unusual

subset of patients who have ST-segment elevation anterior myocardial infarction MI with no evidence of angiographic disease on cardiac catheterization^{1, 2}. These patients have profound and peculiar reversible cardiac wall motion abnormalities and dysfunction. This cardiomyopathy, named the Takotsubo cardiomyopathy. This relatively newly described heart syndrome consists of characteristic apical ballooning wall motion abnormality with preserved basal contraction in the absence of significant coronary artery disease^{3, 4}. A similar type of apical wall motion abnormality has also been described in patients suffering from subarachnoid haemorrhage⁵, multivessel spasm⁶ and phaeochromocytoma⁷.

Although there are no validated criteria to establish the diagnosis, Takotsubo cardiomyopathy is strongly suggested by the following 4 key clinical criteria: (1) a setting of acute emotional stress; (2) symptoms and findings suggestive of acute coronary syndrome with normal coronary arteries on angiography; (3) echocardiographic demonstration of LV dysfunction with preserved function at the base and akinesia of all other segments; and (4) restoration of normal LV function within several weeks of onset of the symptoms⁸. Our patient fulfilled all of these criteria and developed severe LV dysfunction with normal coronary arteries.

The classic wall motion abnormalities of Takotsubo cardiomyopathy described by Seth et, al.,⁹ involving the distal septum exclusively, with associated hyperkinetic base. In our case the regional wall motion involves the entire mid heart and distal apex while the basal segment was not hyperkinetic. Several published literatures described atypical presentation of takotsubo cardiomyopathy which could open the door to the possibility that Takotsubo may have shades of expression¹⁰.

In most patients with Takotsubo cardiomyopathy, ECG findings show ST-T changes that include both ST elevation and depression. Clinical symptoms of Takotsubo cardiomyopathy mimic those of MI, and ECG variations in the acute phase of Takotsubo cardiomyopathy are indistinguishable from those of MI. In this case, neither ST elevation nor depression was found on any ECG leads; however, abnormal ECG results consisting of inverted T waves in V1 and V2 with prolonged QT interval.

To the best of our knowledge; this is the first report which describes severe transient left ventricular dysfunction after induction of anesthesia. All other case reports had occurred in the postoperative period. Our patient had a severe cardiogenic shock with severe left ventricular failure that mandate the placement of IABP. IABP is very useful in these patients as it helps in load reduction¹¹.

Despite the increasing awareness of acute stress-induced myocardial dysfunction, the mechanism remains unknown. Studies have suggested that reversible myocardial contraction abnormalities may occur via mechanisms other than a reduction in epicardial coronary blood flow¹². There is increasing evidence that an exaggerated sympathetic stimulation is probably central to the cause of this syndrome, although the reason for the distinctive contractile pattern with basal sparing remains unclear¹³.

The question now is as follow: Is this patient still can undergo liver transplantation? Up till now the recurrence seems to be rare in these patients when followed for 1 to 4 years¹⁴. However all available data were drawn from non surgical patient and we don't know what will happen when the patient exposed to the same surgical stress. The current recommendations are that β blockers along with heavy pre-medication are administered preoperatively¹⁵. But are these enough for patient undergoing major non-cardiac surgery like liver transplantation? Moreover the recommendation of β -blockers is mainly useful in cases with hyperkinetic base that may be associated with outflow obstruction which is not the case in our patient.

In summary we describe a case of intraoperative acute severe reversible left ventricular failure Takotsubo cardiomyopathy that mimicked acute myocardial infarction. This emerging new entity may have impact on the perioperative anesthetic consideration.

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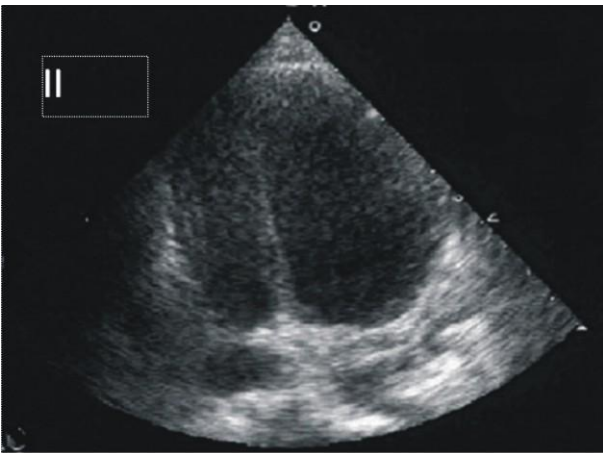
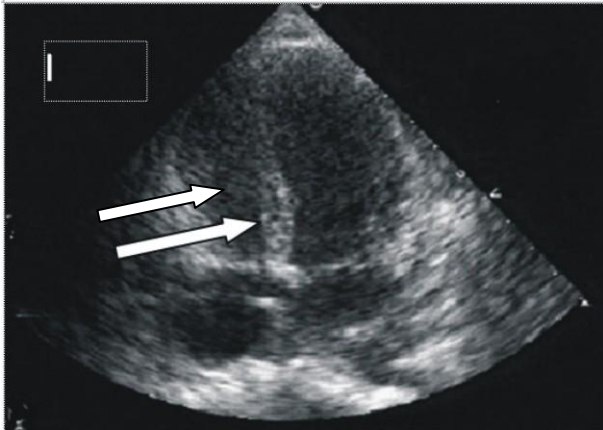


Figure 1 During systole (I) normal contraction of the base (white arrow) while the mid and distal walls are akinetic (II) Diastolic phase



Figure 2: normal coronary angiography