

Sequential Varied Presentation of Takotsubo Cardiomyopathy: Diagnosis and its Management

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Abstract

Takotsubo Cardiomyopathy (TCM) is a reversible cardiac disease with good prognosis and presentation similar to acute coronary syndrome. Most of the times, it occurs secondary to the stress encountered during the perioperative period. It can even present at a later time during postoperative period making the diagnosis difficult. High sympathetic outflow can be prevented by long term beta blockade and acute prevention by alpha adrenergic blockade with dexmedetomidine. Multi-disciplinary approach with preoperative counselling can result in the reduction of stress factors and reduced incidence of stress induced cardiomyopathy.

Keywords: Adrenergic Blockade, Stress Induced Cardiomyopathy, Pregnancy Counselling, Takotsubo Cardiomyopathy

1. Introduction

Takotsubo Cardiomyopathy (TCM) is a neuro-radiological disorder resulting from abnormal pathogenesis of catecholamines and can be the reason for sudden cardiac death. Though the incidence is very rare in pregnancy, and it is more often similar to acute coronary syndrome. Herewith, presenting a case of stress cardiomyopathy (i.e., TCM) in a twin gestation patient complicated with postpartum preeclampsia and pulmonary edema in the postpartum period and its management.

2. Case Report

Elderly primi aged 36 years with 34 weeks of gestation with monochorionic diamniotic twin gestation were planned for emergency caesarean section in view of preterm labour and cephalo-pelvic disproportion. Steroid cover was given due to prematurity. Patient had one episode of

high blood pressure recording associated with chest pain, but Electrocardiogram (ECG) and echocardiogram were within normal limits.

With anti-aspiration prophylaxis, patient was taken up for caesarean section. Vitals were Pulse Rate (PR) 90/min, non-Invasive Blood Pressure (NIBP) 140/90 mmHg, oxygen saturation (SPO₂) on room air 98%. Under spinal anesthesia, surgery was started and twin babies were delivered uneventfully. Despite of oxytocin infusion uterus was atonic, for which methergine 0.2mg intramuscularly (IM) and two doses of inj.Carboprost 250 mcg IM were given at 15 minutes interval and followed by sublingual misoprostol 600 mcg. Intravenous (IV) fluids were given according to the vitals and blood loss of the patient. Following these measures, uterus was well contracted and closure was done, but patient had high NIBP recordings for which IV labetalol was administered during the initial postoperative period, as it would have been thought secondary to the uterotonics.

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8 hrs following surgery, patient continued to have high BP recordings; nitroglycerine infusion was started with invasive monitoring. 2-3 hours later, patient became tachypneic with desaturation episode and bilateral crepitations lead to suspicion of pulmonary edema. 12 lead ECG and 2D-ECHO turned out to be normal except for sinus tachycardia. Patient was managed with oxygen supplementation, diuretics and 30° head up position. 3-4 hrs later, tachypnea worsened and patient became drowsy with decreased responsiveness, and arterial blood gas analysis shown hypoxia with compensated respiratory alkalosis. Intubation was done and inj. noradrenaline infusion started. Differential diagnoses were pulmonary edema secondary to accelerated hypertension and cardiomyopathy or sepsis or pulmonary thromboembolism. Respective measures were taken.

2 hrs post intubation, ST elevation in lead I, aVL, V5-6 in ECG supported by echocardiography which shown anteroapical hypokinesia with ejection fraction 45% and moderate mitral regurgitation. Differential diagnosis was changed to STEMI. Further evaluation shown troponin T of 947 ng/L and loading dose of aspirin, clopidogrel and atorvastatin along with unfractionated heparin were given. But, coronary angiography revealed normal coronaries. Aspirin and atorvastatin were continued and T. Metoprolol and Furosemide were added. Cardiac magnetic resonance imaging shown dilated left atrium and ventricle with pericardial effusion associated with hypokinesia of apical segments of left ventricle with mild mitral regurgitation possibility of stress induced cardiomyopathy. ECHO repeated 2 weeks later shown mild improvement in function of anterior wall and anteroapical wall.

3. Discussion

TCM incidence during perioperative period is 1 in 6,700 cases¹. Multiple surgical procedures have been reported developing perioperative TCM, including gastrointestinal, cardiothoracic, orthopaedic and transplant surgeries as well as caesarean delivery^{2,3}. Stress factors in our case includes elderly gravida with twin gestation³ and preterm labor, intraoperative atonic uterus required medical management (Methergine and Carboprost), coronary vasospasm, along with postoperative high BP recordings. The utility of methergine was an important confounding

factor, as there are reports suggesting methergine induced MI without significant results³. Provocation of stress response can result in cardiac arrest⁴ with the inability to maintain ejection fraction that compromises the cardiac output, leading to pulmonary edema and right heart failure. Mortality is approximately 1%, with recurrence rate of up to 8% and majority (58%) in the postoperative period. Patient with risk factors on exposure to ergot alkaloids predisposes to cardiac ischemia and conduction disturbances⁵.

These patients have transient systolic dysfunction of apical and mid segments of left ventricle in the absence of obstructive coronary arteries and intraventricular thrombus formation is a rare complication of TCM and 5.3% of the cases having left ventricular apical thrombus happening during the acute phase which is more often extensive than acute myocardial infarction⁶. Despite this, prophylactic anticoagulation is not practised widely prior to the detection of thrombus. But in our case, anticoagulation was started after the final diagnosis. And the plasma catecholamine levels were much higher in TCM than ACS. Mayo clinic criteria which are the most commonly used and accepted criteria, which fulfils three out of four⁷. Diagnosis and differentiation of TCM from ACS is clinically challenging but prognosis is entirely different if diagnosed and managed early. Recently, there is a non-invasive tool called troponin-ejection fraction product, derived from peak troponin I level and echocardiography derived LV ejection fraction, in places where the emergent angiogram is contraindicated. A value of > 250 had a high overall accuracy of 91% to differentiate an AMI from TCM.⁷ Recent study suggested two different ECG patterns namely, ST elevation followed by T wave inversions after ST elevations have subsided, and more commonly T wave inversions alone with QTc prolongations^{2,7}. In our case, though it was late presentation, our patient was symptomatic by 8 hrs in the postoperative period, but ECG findings were prominent (i.e., ST elevation) almost 12 hours later. As many tests were available, coronary angiography still remains the confirmatory test to differentiate between ACS and TCM. Though TCM is associated with normal coronaries, yet 10% of the cases can still present as obstructive coronaries. In such scenarios, cardiac MRI need to be performed and the other imaging modalities like PET scan can also be

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