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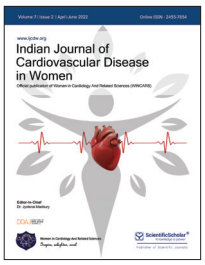

Case Report *Cardiovascular*

Takotsubo Cardiomyopathy in an Elderly Gravida with Twin Gestation

S. Sathiya Priya[1], R. Sujithra Devi[1], Gowri Dorairajan[1], Ajith Ananthakrishna Pillai[2]

Departments of [1]Obstetrics and Gynecology and [2]Cardiology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India.

***Corresponding author:**
R. Sujithra Devi,
Department of Obstetrics and Gynecology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India.

sujiradjan@gmail.com

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ABSTRACT

Takotsubo cardiomyopathy (TCM) is a cardiac emergency characterized by new-onset transient left ventricular systolic dysfunction with variable wall motion abnormalities without significant coronary artery obstructive disease. It presents with acute onset of chest pain, abnormalities in electrocardiogram, echocardiography, and elevated cardiac enzymes that are consistent with acute myocardial infarction. It is more common among postmenopausal women. However, it is reported in pregnant women in their third trimester and peripartum period. We present an elderly primigravida with twin gestation admitted for preterm labor that developed chest pain in the intrapartum period, followed by cardiogenic shock. On evaluation, diagnosed to have TCM and was successfully managed with a multidisciplinary team. TCM is a rare condition in the peripartum period, and it should be differentiated from myocardial infarction and peripartum cardiomyopathy as the lines of management and prognosis are different.

Keywords: Cardiomyopathy, Left ventricular dysfunction, Peripartum cardiomyopathy, Pregnancy, Stress-induced cardiomyopathy

INTRODUCTION

Takotsubo cardiomyopathy (TCM), also known as stress-induced cardiomyopathy, is a cardiac emergency characterized by new-onset transient left ventricular systolic dysfunction with variable wall motion abnormalities without significant coronary artery obstructive disease.[1] It is more common in postmenopausal women; however, it has been increasingly reported during pregnancy and in the peripartum period.[2] Women undergoing cesarean section may develop an earlier onset of TCM when compared to vaginally delivered.[3] Profound emotional or physical stress triggers it, with a higher incidence in people with pre-existing psychiatric illnesses. However, few do not have identifiable stress factors.[3] The widely accepted pathophysiology is a transient ischemic coronary insult and myocardial stunning by exaggerated sympathetic crises and catecholamine storm precipitated by stressful conditions. Other proposed mechanisms are vasopressor usage to counterbalance anesthesia-induced hypotension, decreased estrogen level, coronary artery vasospasm, and coronary microcirculation dysfunction.[4,5]

Differentiating TCM from peripartum cardiomyopathy is of utmost importance because, in TCM, cardiac function usually recovers spontaneously within 4–8 weeks in the majority of patients.[6] The long-term prognosis is also good with very low recurrence in subsequent pregnancies, whereas peripartum cardiomyopathy worsens with subsequent pregnancies and

cardiac function never recovers completely.[7] Still, sudden cardiac death in the acute phase of TCM due to cardiogenic shock and arrhythmias is seen in 1.1% of cases. Differential diagnoses are acute coronary syndrome, pulmonary embolism, amniotic fluid embolism, and viral myocarditis.

CASE REPORT

A 36-year-old, short-statured (135 cm), elderly primigravida with dichorionic diamniotic (DCDA) twins was admitted with threatened preterm labor at 32 weeks of gestation. It was a spontaneous conception after 1 year of non-consanguineous marital life. Her first and second trimesters were uneventful with normal antenatal investigations. She was diagnosed with gestational diabetes in the third trimester and was on medical nutrition therapy. She also had moderate iron-deficiency anemia and was on oral iron therapy. Her sugar profile, renal function test, and liver function tests were normal, and her complete blood count revealed mild anemia at admission. Urine culture and high vaginal swab cultures were sterile. Ultrasonogram revealed DCDA twins both in cephalic presentation, with adequate growth. She was started on steroids for fetal lung maturation under tocolytic coverage (calcium channel blocker). She had spontaneous rupture of membranes at 33+6 weeks of gestation; hence, labor was augmented with oxytocin. In the intrapartum period, she complained of chest pain radiating to the back, not associated with diaphoresis. Electrocardiogram and echocardiography (ECHO) were normal; cardiologist opined as atypical chest pain and was managed conservatively.

She underwent cesarean section after 7 hours of labor and delivered twin male babies weighing 1.96 and 2.03 kg, respectively, with APGAR score of 8/9. Intraoperatively, she had a mild atonic postpartum hemorrhage with 600 mL of blood loss, managed with oxytocin, misoprostol, and one packed red cell transfusion. She had a high blood pressure (BP) recording of 200/120 mmHg in the immediate post-operative period, which was managed with i.v. Labetalol 20 mg. Eight hours post-cesarean section, she had tachypnoea and breathlessness with a fall in oxygen saturation to 92% at room air. On auscultation, bilateral fine basal creptitations were present with BP of 140/90 mmHg hence was diagnosed as having pulmonary edema and was treated with injection furosemide 60 mg IV and 6-L oxygen by mask.

She desaturated again after 16 hours post-cesarean section, not maintained with O_2 mask or non-invasive ventilation, requiring intubation. Subsequently, she developed cardiogenic shock and was started on inotropes. The 12-lead electrocardiogram showed ST segment elevation in II, III, aVF, V3-V4 leads suggestive of anterior and inferior wall ST-elevation myocardial infarction (STEMI) [Figure 1]. ECHO showed anterior wall hypokinesia, dilated left atrium and ventricle with left ventricular ejection fraction (LVEF) of 45%, and moderate mitral regurgitation. Troponin levels >900, Creatine- Phospho- kinase -Myocardial Band (CPK-MB) was 28 (Normal: 0–24). She was diagnosed with an acute anterior and inferior wall myocardial infarction. Since thrombolysis could not be done due to the immediate post-operative period, emergency percutaneous angiography was performed, which revealed normal coronaries. Thus, stress-induced cardiomyopathy was diagnosed [Figure 2a-b]. She was managed in the critical care unit, started on anti-failure measures and low molecular weight heparin. Inotropes were weaned off on post-operative day (POD)-1, extubated on POD-4, and repeat echo showed improved hypokinesia and LVEF to 50%. On POD-4, cardiac magnetic resonance imaging (MRI) was done, which showed hypokinesia of apical segments of the left ventricle with a dilated left atrium, left ventricle with mild pericardial effusion, and mild mitral regurgitation, suggestive of stress-induced cardiomyopathy [Figures 3a-c and 4]. On POD-5, she developed irrelevant speech and agitation; her vitals and blood investigations were normal, thus diagnosed with mental and behavioral changes associated with puerperium by a psychiatrist and was treated. On POD-9, her LVEF improved to 55%, suture removal was done, and discharged on POD-15 with both babies in good condition. On follow-up after 1 month, her LVEF was more than 60%.

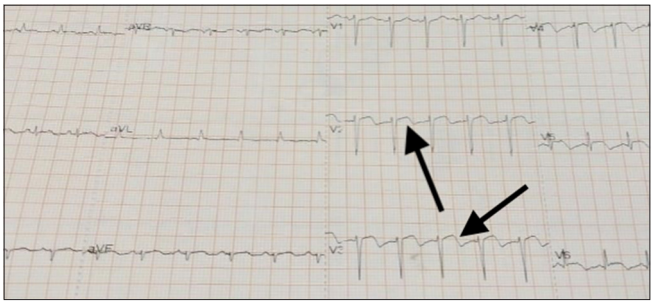

Figure 1: 12 Lead electrocardiogram showing ST elevation in lead II, III, aVF, V-V4 (arrow points to ST segment elevation in lead V2 and V3).

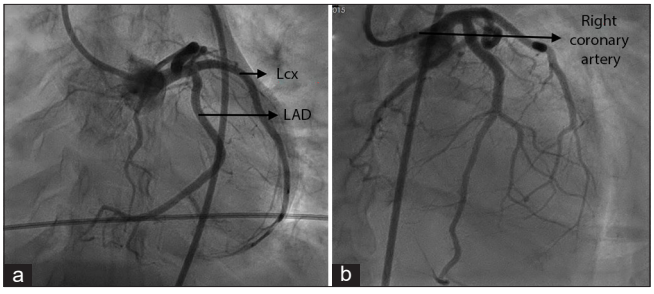

Figure 2: Coronary angiogram: (a) Left dominant system - long arrow points normal left anterior descending artery (LAD) short arrow points and circumflex artery (Lcx). (b) Arrow points to Normal right coronary artery with branches.

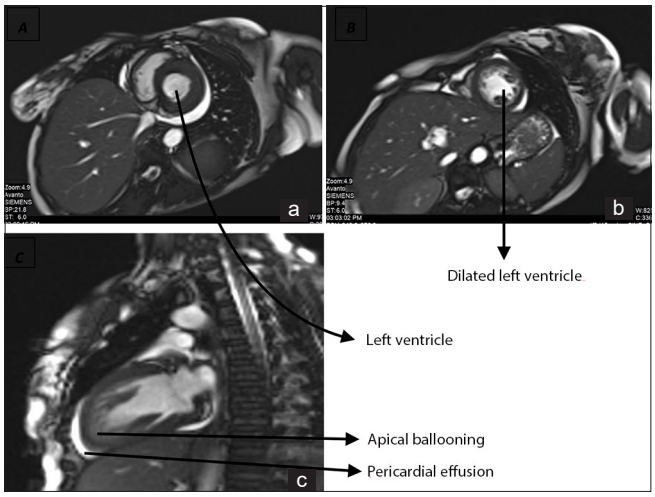

Figure 3: Bright blood images of the heart true fast imaging with steady-state free precession sequence - short axis view at the base of the heart (a) Mid ventricular level-arrow points left ventricle (b) Two chamber view depicting dilated left ventricle (arrow) Sagittal view-Arrow points to (c) Apical ballooning and pericardial effusion.

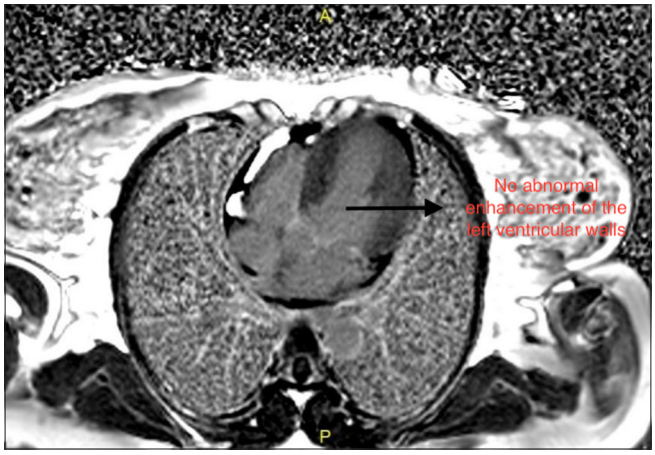

Figure 4: Post-contrast late gadolinium enhanced magnetic resonance imaging phase - sensitive inversion recovery sequence arrow mark shows the left ventricular walls with no abnormal enhancement. (A: Anterior, P: Posterior).

DISCUSSION

TCM is a rare, life-threatening cardiac emergency diagnosed in 2% of patients hospitalized for acute myocardial infarction.[8] It typically presents with chest pain in 44%, shortness of breath in 28%, palpitations, and orthopnea.[9] TCM has been categorized into primary and secondary. Secondary TCM is those patients developing cardiac symptoms while being hospitalized for other pathologies.[5] Cesarean delivery, labor, and abortion are some of the possible triggers.[5] Approximately 80% develop after administering vasoconstrictive drugs such as adrenaline, phenylephrine, atropine, ephedrine, and sulprostone.[1] It has been seen that 81% of cases had a cesarean delivery, and symptoms appeared during surgery in 38% of TCM.[9]

Being an elderly primigravida with short stature and having twin gestation in labor for 7 h followed by emergency cesarean section could have led to catecholamine surge and triggered TCM. Initially, she had a non-STEMI due to increase afterload during labor, followed by STEMI in the post-operative period. Although she did not having a prior history of any psychiatric illness, it might not have been brought into light earlier, which exhibited itself during the postpartum period.

ECG, cardiac biomarkers, ECHO, and angiography played a vital role in the diagnosis. The common ECG abnormalities include ST-segment elevation, ST-segment depression, T-wave inversion, abnormal Q waves, QTc prolongation, and nonspecific abnormalities.

Typical ECHO findings are regional wall motion akinesia involving a large area of LV extending beyond a single coronary artery territory with reduced LVEF and apical ballooning. Other findings in ECHO include Mitral regurgitation with or without a systolic anterior motion of the anterior leaflet. Systolic ballooning can involve the apical region, mid-ventricular region, basal region, or focal area in the decreasing order. Apical ballooning is the most common finding, which was present in the index case.

Cardiac MRI differentiates TCM from acute myocardial infarction and myocarditis.[3] Our case fulfilled mayo's diagnostic criteria of stress-induced cardiomyopathy, i.e., transient hypokinesis of the left ventricular apical myocardial segments extending beyond a single epicardial vascular distribution, with cardiac enzyme elevation and new electrocardiographic changes, normal angiogram, and absence of myocarditis.

Management includes symptomatic treatment for heart failure, pulmonary edema, rhythm disorder, and cardiac arrest. Cardiac optimization is done with assisted ventilation. Beta-blockers, diuretics, angiotensin-converting-enzyme inhibitors (only postpartum) for heart failure, inotropes and vasopressors to be avoided except in cardiogenic shock and anticoagulants.[10] Our multidisciplinary management helped the patient for complete resolution of triggers and rapid recovery by 3rd week. Occurrence of TCM subsequently is below 10% with rapid recovery; hence, subsequent pregnancy is not contraindicated, though a long-term follow-up should be necessary to look for complications.

CONCLUSION

TCM is a rare condition in the peripartum period, and it should be differentiated from peripartum cardiomyopathy.

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